Thieme

Effect of Bariatric Surgery on the Circulating Level of Adiponectin, Chemerin, Plasminogen Activator Inhibitor-1, Leptin, Resistin, and Visfatin: A Systematic Review and Meta-Analysis

Authors
Moein Askarpour[1], Shahab Alizadeh[2], Amir Hadi[3, 4], Michael E. Symonds[5], Maryam Miraghajani[6], Ali Sheikhi[1], Ehsan Ghaedi[2, 7]

Affiliations
1 Department of Community Nutrition, Tehran University of Medical Sciences, Tehran, Iran
2 Department of Cellular and Molecular Nutrition, Tehran University of Medical Sciences, Tehran, Iran
3 Halal Research Center of IRI, FDA, Tehran, Iran
4 Department of Clinical Nutrition, Isfahan University of Medical Sciences, Isfahan, Iran
5 The Early Life Research Unit, Division of Child Health, Obstetrics and Gynaecology, University of Nottingham, Nottingham, UK
6 Cancer Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
7 Students' Scientific Research Center (SSRC), Tehran University of Medical Sciences (TUMS), Tehran, Iran

Key words
bariatric surgery, meta-analysis, leptin, adiponectin, resistin

received 29.04.2019
accepted 18.02.2020

Bibliography
DOI https://doi.org/10.1055/a-1129-6785
Horm Metab Res 2020; 52: 207–215

ISSN 0018-5043

Correspondence
Ehsan Ghaedi
Department of Cellular and Molecular Nutrition, School of Nutrition Sciences and Dietetics, Tehran University of Medical Sciences
Poorsina Street
Enghelab Avenue
PO Box 14155-6446
Tehran
Iran
Tel : +982188954911, Fax : +982188974462
ehsanghaedi073@gmail.com

Supplementary Material for this article is available online at http://www.thieme-connect.de/products.

ABSTRACT

Different adipokines secreted from adipose tissue, exert a range of physiological effects. The aim of present systematic review and meta-analysis was to critically investigate the consequence of bariatric surgery on circulating adipokines, that is, adiponectin, leptin, visfatin, resistin, plasminogen activator inhibitor, and chemerin. After systematically checking the following electronic databases: ISI web of Science, Scopus and PubMed without limitation in time and language up to February 2019, a pool based on a random effect model was established. Eighty-five eligible studies were entered for quantitative analysis. Our meta-analysis revealed that circulating adiponectin increased significantly after bariatric surgery [Standardized mean difference (SMD) = 1.401, 95 % CI: 1.101, 1.701, p < 0.001]; whilst leptin (SMD = –2.178, 95 % CI: –2.433, –1.923, p < 0.001), PAI-1 (–14.928 ng/ml 95 % CI: –21.794, –8.063, p < 0.001), and chemerin (–50.238 ng/ml 95 % CI: –85.708, –14.768, p < 0.001) decreased. However, serum visfatin (2.05 ng/ml, 95 % CI: –5.07, 9.17, p = 0.573) and resistin (–2.080 ng/ml, 95 % CI: –5.352, 1.192, p = 0.21) were unchanged. In conclusion, bariatric surgery is associated with a reduction in specific adipokines including leptin, chemerin, and PAI-1, whereas adiponectin is raised, adaptations that could be indicative of improved fat mass and function.

Introduction

Obesity, which is defined as excessive expansion of white adipose tissue, is a global health problem with reported prevalence of more than 1.9 billion around the world [1]. It is associated with a range of health-related problems and major chronic diseases including cancer, cardiovascular diseases (CVD), and diabetes mellitus (DM) [2–4]. There is thus an urgent need for patients with obesity to get rid of their excess fat. Many conservative therapies such as restricted diet take time and do not result in sustained weight loss [5], whereas bariatric surgery appears to have a better outcome [6, 7]. Food intake is reduced following bariatric surgery as a consequence of either reducing the size of stomach through insertion of a gastric band, or the removal of a part of the stomach or reducing the length of the small intestine. Long-term studies indicate this type of surgery, is not effective for weight loss [5], but also ameliorate a variety of obesity-related diseases such as DM and CVD [8]. However, some adverse effects may be expected due to the magnitude of the procedure [9]. Moreover, whether it also has further benefits due to an improved profile of circulating adipokines remains to be fully clarified.

Adipose tissue is a loose connective tissue, which is mainly composed of adipocytes. It acts as an energy storing for lipids and is recognized as the biggest endocrine organ of the human body, secreting several soluble factors, known as adipokines [10]. These are critical regulators of systemic lipid and glucose homeostasis, although most are associated with obesity-related health problems such as insulin resistance, beta-cell dysfunction, endothelial dysfunction, and atherosclerosis [11–14]. Although leptin can regulate energy balance by inhibiting hunger, obesity results in a loss of sensitivity [15]. Adiponectin is a protein hormone with an anti-inflammatory role that can modulate glucose and lipid homeostasis [16]. Chemerin, is a chemoattractant protein, that is necessary for adipogenesis, which when elevated in adipose tissue may be a marker for the onset of DM [17]. Visfatin is an enzyme that activates insulin and has insulin-mimetic effects [18], whereas plasminogen activator inhibitor-1 (PAI-1), is the primary inhibitor of tissue- and urokinase-type plasminogen activators, and considered to be a critical regulator of the fibrinolytic system [19]. Finally, resistin promotes hepatic production of low density lipoproteins (LDL), which degrades LDL receptors [20].

Weight loss after bariatric surgery is mainly due to the loss of visceral fat [21], but whether the secretion of adipokines is then modulated remains to be fully established as the response varies [22–24] and is not always significant [25–30]. We, therefore, conducted a meta-analysis and systematic review to investigate the effect of different types of bariatric surgeries on these adipokines.

Materials and Methods

This meta-analysis and systematic review was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [31].

Search strategy

PubMed/Medline and Scopus were searched from inception to January 2019 using the following terms in titles and abstracts: ((*leptin* [Title/Abstract]) OR (*adiponectin* [Title/Abstract]) OR (*PAI-1* [Title/Abstract]) OR ("*Plasminogen activator inhibitor-1*" [Title/Abstract]) OR (*visfatin* [Title/Abstract]) OR ("*pre-B cell colony-enhancing factor-1*" [Title/Abstract]) OR (*PBEF* [Title/Abstract]) OR ("*nicotinamide phosphoribosyltransferase*" [Title/Abstract]) OR (*Nampt* [Title/Abstract]) OR (*Chemerin* [Title/Abstract]) OR (*resistin* [Title/Abstract]) OR ("*adipocyte secreted factor*" [Title/Abstract]) OR (*ADSF* [Title/Abstract]) OR (*FIZZ-3* [Title/Abstract])) AND (("*Bariatric Surgery*" [Mesh]) OR ("*Gastric Bypass*" [Title/Abstract]) OR ("*Gastrectomy*" [Title/Abstract]) OR ("*Bariatric Surgery*" [Title/Abstract]) OR ("*Biliopancreatic Diversion*" [Title/Abstract]) OR (*Gastroplasty* [Title/Abstract]) OR (*bariatric* [Title/Abstract]) OR (*Roux-en-Y* [Title/Abstract]) OR (*RYGB* [Title/Abstract]) OR ("*Sleeve gastrectomy*" [Title/Abstract]) OR ("*Gastric sleeve*" [Title/Abstract]) OR ("*gastric band*" [Title/Abstract]) OR (*Lap-Band* [Title/Abstract]) OR ("*duodenal switch*" [Title/Abstract])). As we did not want to miss any relevant articles, all the reference list of eligible articles, related reviews, and meta-analyses were hand-searched.

Eligibility criteria

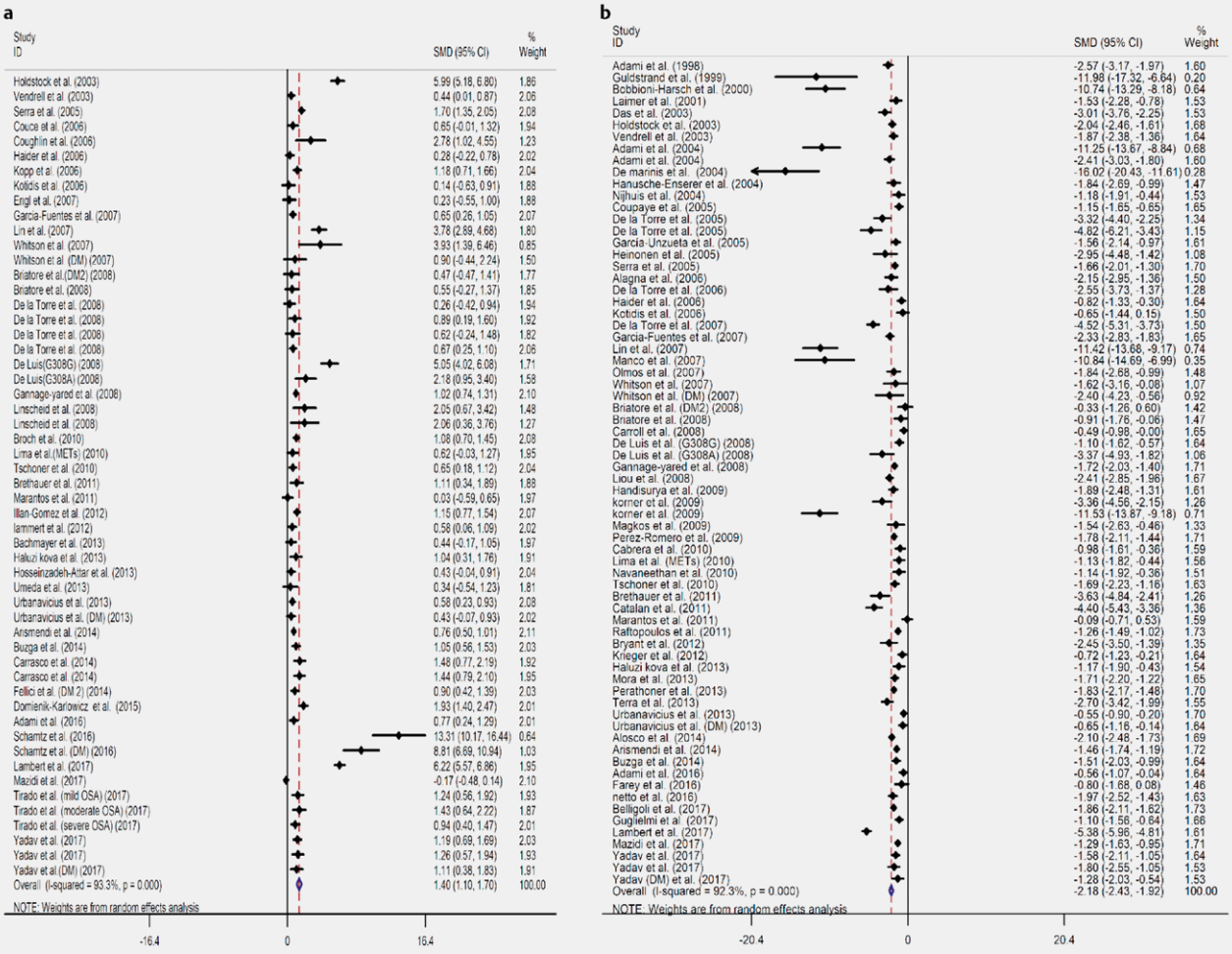
Title and abstract of all retrieved articles in the initial search were screened separately by two different reviewers. Studies were included if they met the following criteria (1) reported baseline BMI and at least one of the following measures; leptin, adiponectin, PAI-1, visfatin, chemerin, and resistin before and after surgical intervention, (2) performed one of the common bariatric surgeries [Roux-en-Y gastric bypass (RYGB); SG, gastric bypass (GBP); vertical banded gastroplasty (VBG); GB, Biliopancreatic diversion (BPD)], and (3) bariatric surgery were performed only for treatment of obesity and not any other reason. Articles were excluded if they had the following exclusion criteria: (1) the reason of surgery was not obesity, (2) had insufficient data for the outcomes of interest in individuals, and (4) experimental and animal studies. All editorials, reviews, letters to editors, conference papers, animal studies, and molecular studies were excluded.

Data extraction

Two independent researchers (MAE and GH) did the study selection, whereas to resolve any controversies, a chief investigator (SA) was present. The following data were acquired from each study; first author's name; year of publication; study location; study duration; number of participant's, participant's age and gender; study design; weight and BMI before and after intervention; participant's health status; mean and SD of serum or plasma levels of leptin, adiponectin, PAI-1, visfatin, chemerin, and resistin. Also if all individuals just had the same disease, it was reported (▶ **Fig. 1,2**).

Assessment of study quality

Two authors independently assessed the quality of included studies by the Newcastle-Ottawa Quality Assessment Scale (NOS). This scale comprising of three quality factors: selection (maximum 4 stars), comparability (maximum 2 stars), and outcome (maximum 3 stars). A maximum of 9 stars represents the highest quality. A total score of 7 or more was considered to indicate high-quality studies [32, 33].



▶ **Fig. 1** Forest plot presenting mean difference (MD) and 95 % confidence intervals for the impact of bariatric surgery on **a** adiponectin and **b** leptin. Results from Hedges' g analysis.

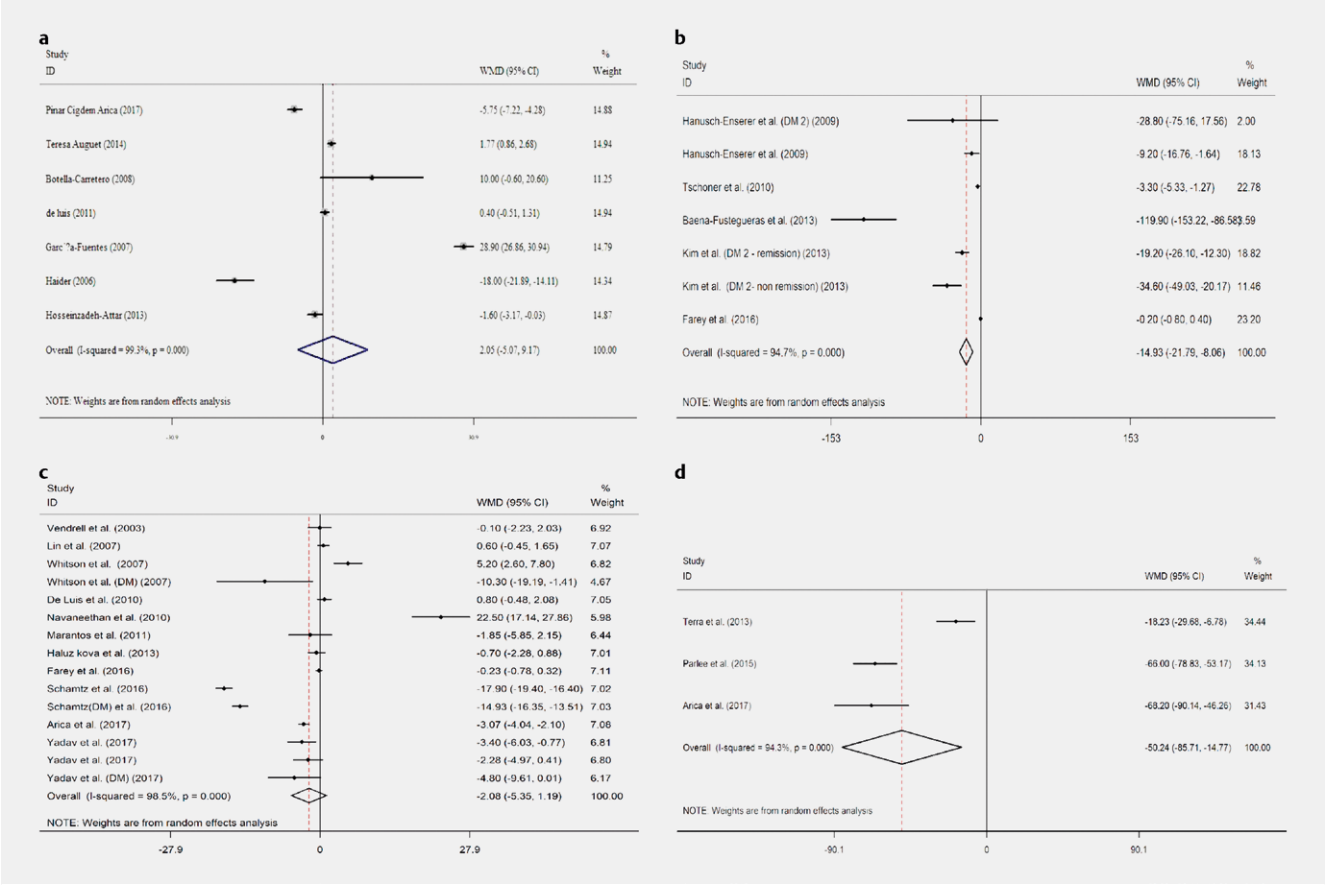
Statistical analysis

Mean difference was used as the effect size and a random effects model was utilized for the meta-analysis [34]. Using random-effects model, effect sizes were acquired as the weighted mean difference (WMD) and 95 % CI by DerSimonian–Laird method. When reported variable could not be pooled, Hodges's adjusted g was used to calculate overall estimate of effect size. A test for heterogeneity was performed (using Q statistic and I^2) [35]. The subgroup analysis (▶ **Table 2S**) was conducted to find out possible sources of heterogeneity among included studies. Meta-regression analysis was performed to investigate whether participant baseline BMI and also duration of follow up could explain the heterogeneity across studies and also affect measured effect size. Publication bias was investigated by visual inspection of the funnel plot as well as by using the Egger's regression method. In case of publication, bias trim and fill analysis were performed. This specific analysis was conducted for evaluating the possible effect of publication bias on overall estimate of effect size based on possible relevant unpublished studies [36]. We used the sensitivity analysis to assess the effect of each study on the overall effect size by removing each individual study in turn. All statistical analyses were carried out using Stata MP V.14.0 (StataCorp, College Station, Texas, USA).

Results

Study selection

PRISMA flow diagram of the search process is depicted in (▶ **Fig. 1S**) (Supporting Information). A total of 2071 papers were found. After removing duplicates, 1706 abstracts were selected for a more detailed review. Following screening based on the title and abstract, 1599 articles were excluded: 1) 1493 unrelated topics, 2) 28 animal studies, 3) 78 letters, reviews, and book sections. One hundred and seven citations remained after screening for title and abstracts. After full-text evaluation a further 22 studies were also excluded: 1) combination with other treatments (n = 3), and 2) with not enough information (n = 19). Finally, 83 prospective and 2 retrospective studies remained for the systematic review (▶ **Table 1S**) (Supporting Information).



▶ **Fig. 2** Forest plot presenting mean difference (MD) and 95 % confidence intervals for the impact of bariatric surgery on **a** visfatin (ng/ml), **b** plasminogen activator inhibitor-1 (ng/ml), **c** resistin (ng/ml), and **d** chemerin (ng/ml).

Findings from systematic review

Characteristics of eligible studies are summarized in (▶ **Table 1S**), with a sample size of between 5 and 180 participants who underwent bariatric surgery (total sample size of studies; n = 3512).

Sixty-one studies reported leptin [22, 23, 25, 27, 28, 30, 37–91], 40 adiponectin [22, 23, 26, 28–30, 40, 43, 46, 47, 49, 54, 56, 57, 61, 62, 67, 72–74, 78, 79, 89–106], 11 resistin [23, 24, 27, 30, 62, 74, 78, 82, 91, 105, 107], 7 visfatin [24, 25, 57, 61, 100, 108, 109], PAL-1 [27, 65, 89, 110, 111], and 3 chemerin [24, 88, 112]. Across the included studies 8 were performed on patients with DM [30, 47, 65, 90, 91, 99, 105, 111], one study was on the metabolic syndrome [73], one on obstructive sleep apnea [106], another study stratified subjects based on different polymorphisms of tumor necrosis factor-α-G308A genotype [107] and other studies were carried out just on participants with obesity [22–29, 37–46, 48–64, 66–72, 74–89, 92–98, 100–104, 108–110, 112].

Across the included studies 26 were on patients with a Roux-en-Y gastric bypass (RYGB) [29, 30, 42, 45–48, 51, 63, 67–69, 73, 74, 76, 79, 80, 86, 87, 91, 95, 99, 104, 105, 110, 111], 18 after various forms of surgery [22, 43, 54, 56, 57, 72, 78, 81–83, 88, 89, 92, 100, 103, 106, 108, 109], 14 bilio-pancreatic diversion (BPD) [25, 28, 37–41, 54, 55, 58, 77, 97, 107, 112], 8 gastric bypass (GBP) [23, 53, 54, 93, 94, 96, 98, 101], 6 adjustable gastric banding (AGB) [24, 52, 64, 69, 70, 90], 6 gastric banding (GB) [26, 61, 65, 66, 71, 104], 5 laparoscopic sleeve gastrectomy (LSG) [27, 44, 49, 62, 85], 4 after vertical banded gastroplasty (VBG) [54, 60, 84, 102], 2 sleeve gastrectomy (SG) [59, 94], 1 laparoscopic gastric banding (LGB) [50], and 1 laparoscopic minigastric bypass surgery (LMGBP) [75].

In addition, studies were performed in subjects with different baseline BMIs, 7 were in subjects between 30–39.9 kg/m^2 [29, 40, 72, 75, 94, 99, 111], 21 in subjects between 40–44.9 kg/m^2 [26, 27, 38, 45, 49, 50, 55, 56, 62, 67, 69, 71, 77, 78, 80, 85, 89, 94, 104–106], 44 in subjects between 45–49.9 kg/m^2 [23, 24, 37, 39, 41–44, 46–48, 51, 52, 54, 58–61, 64, 66, 68–70, 73, 74, 81–84, 87, 88, 90, 92, 93, 98, 100–103, 106–108, 110, 112], and 17 studies in subjects over than 50 kg/m^2 [22, 28, 53, 57, 76, 86, 91, 95–97, 99, 109], whilst 4 studies did not report BMI [30, 54, 65, 79].

Twenty studies were on females only [25, 26, 45, 51, 52, 54, 55, 60, 62, 71, 73, 77, 78, 94, 96, 98, 101, 102, 108, 109] and 2 on males only [41, 81] and the rest included studies conducted on both genders [22–24, 27–30, 37–40, 42–44, 46–50, 53, 56–59, 61, 63–70, 72, 74–76, 79, 80, 82–93, 95, 97, 99, 100, 103–107, 110–112].

Meta-analysis results

Effect of bariatric surgery on circulating adiponectin

Fifty-four prospective studies including a total of 1862 participants reported circulating adiponectin after bariatric surgery. Overall, combined results showed that circulating adiponectin increased

following bariatric surgery (Hedges' g = 1.401, 95 % CI: 1.101, 1.701, p < 0.001) (▶ **Fig. 1a**). Due to a significant heterogeneity between studies (I^2 = 99.3 %, p < 0.001), subgroup analyses were performed based on gender (female/both), type of surgery (RYGB/GB/GBP/BPD/miscellaneous/various), baseline BMI (< 45 kg/m^2/ ≥ 45 kg/m^2), follow-up period (≤ 3 months/6–11 months/12 months/> 12 months). This revealed that the effect of bariatric surgery on adiponectin remained significant in all subgroups except for GB type of surgery, although a lack of eligible studies in this type of surgery must be considered before making any conclusion.

Effect of bariatric surgery on circulating leptin

Seventy prospective and retrospective studies including a total of 2751 participants reported leptin as an outcome measure. Pooled effect size showed that circulating leptin decreased significantly following bariatric surgery (Hedges' g = –2.178, 95 % CI: –2.433, –1.923, p < 0.001) (▶ **Fig. 1b**). There was significant heterogeneity among included studies (I^2 = 92.3 %, p < 0.001). Subgroup analysis also was performed (▶ **Table 2S**) and showed a significant association in all subgroups for gender, baseline BMI, type of surgery, and follow-up duration.

Effect of bariatric surgery on circulating resistin, visfatin, PAI-1, and chemerin

Seven prospective articles with 330 participants reported resistin as an outcome measure. Pooled effect size indicated no effect of bariatric surgery on circulating resistin (–2.080 ng/ml, 95 % CI: –5.352, 1.192, p = 0.21) (▶ **Fig. 2c**). There was no significant heterogeneity between studies (I^2 = 98.5 %, p < 0.001), and subgroup analysis revealed no association for baseline BMI, type of surgery, and follow-up period (▶ **Table 2S**).

Overall, 8 studies with 171 participants assessed PAI-1 as an outcome measure. Pooled effect size showed that serum PAI-1 decreased significantly following bariatric surgery (–14.928 ng/ml 95 % CI: –21.794, –8.063, p < 0.001) with significant heterogeneity among included studies (I^2 = 94.7 %, p < 0.001) (▶ **Fig. 2b**).

Finally, three studies reported chemerin as an outcome measure following bariatric surgery, that resulted in reduced circulating concentrations (–50.238 ng/ml 95 % CI: –85.708, –14.768, p < 0.001) with significant heterogeneity between included studies (I^2 = 0.0 %, p < 0.001) (▶ **Fig. 2d**).

Seven studies assessed circulating visfatin following bariatric surgery (2.05 ng/ml, 95 % CI: –5.07, 9.17, p = 0.573). Between-study heterogeneity was significant (I^2 = 99.3 %, p < 0.001) (▶ **Fig. 2a**).

Sensitivity analysis

Sensitivity analysis revealed that overall estimates of effect size were not excessively influenced by any of included studies significantly for adiponectin, leptin, chemerin, PAI-1, and visfatin. Pooled effect size for resistin showed significant effect following elimination study of Navaneethan et al. [82] (–3.63 ng/ml, 95 % CI: –6.87, –0.39, I^2 = 98.3 %).

Publication bias and trim and fill analysis

Publication bias analysis was found for studies reporting adiponectin and leptin. Therefore, following trim and fill analysis were performed for these two factors. The modified estimate of pooled effect size for adiponectin, from 64 hypothesized studies changed but remained significantly increased following bariatric surgery (0.819, 95 % CI: 0.45, 1.188, p < 0.001). Trim and fill sensitivity analysis for leptin showed that overall estimate of effect size from 70 hypothesized negative unpublished studies did not change (–2.178, 95 % CI: –4.33, –1.923), and remained significant (p < 0.001). Publication bias did not appear to affect these results. There was no evidence of publication bias for studies examining the effect of bariatric surgery on PAI-1 (p = 0.453, Begg's test), resistin (p = 0.4, Begg's test), and visfatin (p = 0.54, Begg's test).

Meta-Regression analysis

Meta-regression indicated that there was not a linear association between the change in serum adipokine with baseline BMI of participants [i. e., adiponectin (p = 0.41), leptin (p = 0.49), PAI-1 (p = 0.31), and resistin (0.33)]. Moreover, no significant associations with follow up duration and changes in adiponectin (p = 0.85), leptin (p = 0.39), PAI-1 (p = 0.82), visfatin (p = 0.61), and resistin (p = 0.4) were found. There was a significant association between the change in visfatin and baseline BMI of participants (β coefficient = 3.6, p = 0.026).

Discussion

Our comprehensive meta-analysis has shown that circulating leptin, chemerin, and PAI-1 decreased after bariatric surgery, whereas adiponectin decreased, and both visfatin and resistin were unchanged.

Adiponectin is an adipokine, which is not secreted exclusively by the adipose tissue [113], but a range of human tissues [114]. Obesity is associated with decreased adiponectin, that in turn is linked to reduced insulin sensitivity [115]. Increased adiponectin status, mediated by enhanced action, or function of adiponectin receptors (AdipoR) [116], or pharmacological elevation of adiponectin can relieve obesity-related health problems [114]. For instance, thiazolidinediones (TZDs) or other medicinal herbs like Zataria or astragaloside II can increase circulating adiponectin, and improve insulin sensitivity [117–120]. In addition to pharmacological interventions, exercise and training can also raise plasma adiponectin [121–123], and would be predicted to ameliorate obesity-related health problems like DM and CVD, given the cardio-protective effects of adiponectin [124]. The positive effect of all types of bariatric surgery on circulating adiponectin, irrespective of baseline BMI and gender, would therefore be expected to represent a beneficial outcome, as would the decrease in leptin, that has a range of physiological functions including appetite regulation, and energy homeostasis [125]. Serum leptin level is correlated with resting metabolic rate (RMR) in some but not all studies [126], and obesity is accompanied with leptin resistance [127]. Raised leptin has also been found with insulin resistance, DM [128], and thickening of the intima-media thickness with the onset of atherosclerosis [128]. Reduced leptin with bariatric surgery, occurs concurrently with loss of body fat, and has also been seen with exercise and dietary interventions [128, 129].

Increased circulating PAI-1 with obesity is well documented [130], and is an established risk factor for coronary artery disease, atherosclerosis, and stroke [131, 132]. Conversely lowering PAI-I

reduces the risk of CVD [133], so the decrease with bariatric surgery would be expected to be beneficial. Resistin is a pro-inflammatory cytokine that can modulate substrate metabolism through blocking the action of insulin [134], as well as the onset of CVD, due to inflammation, endothelial and smooth muscle cell dysfunction, thrombosis, and angiogenesis [134, 135]. It was, however, unaffected by bariatric surgery as was visfatin. This adipokine can modulate inflammation, and is raised with obesity, DM, metabolic syndrome, and CVD [18, 136, 137]. Chemerin also contributed to adipogenesis, glucose homeostasis, food intake, and body weight; and elevated chemerin with obesity has been implicated in the onset of DM [138–140]. Bariatric surgery decreased chemerin, but the lack of eligible studies for chemerin and significant sensitivity analysis for resistin suggests these findings must be interpreted with caution, and larger scale studies are needed.

Our meta-analysis has both strengths and limitations, and is the first such study of its kind. Although there were sufficient studies investigating the effects of bariatric surgery on leptin and adiponectin the lack of eligible studies for the other adipokines limits the interpretation of this data. In addition, the effects of confounding factors including, genetic background and other lifestyle modification remained unclear, together with the potential effects of different types of bariatric surgery.

Conclusion

In conclusion, our meta-analysis of prospective and retrospective studies show that bariatric surgery has a beneficial effect on several adipokines including reduced leptin, chemerin and PAI-1, and increased adiponectin, but has no effect on resistin and visfatin. However, these results must be interpreted with caution especially for adipokines with fewer eligible studies for quantitative analysis.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

[1] WHO Obesity and overweight. 2018; www.who.int

[2] Steele CB, Thomas CC, Henley SJ et al. Vital Signs: Trends in Incidence of Cancers Associated with Overweight and Obesity – United States, 2005–2014. MMWR Morbidity Mortality Weekly Report 2017; 66: 1052–1058

[3] Barnes AS. The epidemic of obesity and diabetes: Trends and treatments. Texas Heart Inst J 2011; 38: 142–144

[4] Al-Goblan AS, Al-Alfi MA, Khan MZ. Mechanism linking diabetes mellitus and obesity. Diabetes Metab Syndr Obes Targets Therap 2014; 7: 587–591

[5] MacLean PS, Wing RR, Davidson T et al. NIH working group report: Innovative research to improve maintenance of weight loss. Obesity (Silver Spring. Md) 2015; 23: 7–15

[6] Wolfe BM, Kvach E, Eckel RH. Treatment of obesity: Weight loss and bariatric surgery. Circul Res 2016; 118: 1844–1855

[7] Schigt A, Gerdes V, Cense H et al. Bariatric surgery is an effective treatment for morbid obesity. Neth J Med 2013; 71: 4–9

[8] Tham JC, Howes N, le Roux CW. The role of bariatric surgery in the treatment of diabetes. Ther Adv Chronic Dis 2014; 5: 149–157

[9] Xanthakos SA. Nutritional deficiencies in obesity and after bariatric surgery. Pediatr Clin North Am 2009; 56: 1105–1121

[10] Pessin JE, Kwon H. Adipokines mediate inflammation and insulin resistance. Front Endocrinol 2013; 4: 71

[11] Bulcao C, Ferreira SR, Giuffrida FM et al. The new adipose tissue and adipocytokines. Curr Diabetes Rev 2006; 2: 19–28

[12] Birbrair A, Zhang T, Wang Z-M et al. Role of pericytes in skeletal muscle regeneration and fat accumulation. Stem Cells Develop 2013; 22: 2298–2314

[13] Smekal A, Vaclavik J. Adipokines and cardiovascular disease: A comprehensive review. Biomed Papers Med Faculty Univ Palacky, Olomouc, Czech 2017; 161: 31–40

[14] Kwon H, Pessin JE. Adipokines mediate inflammation and insulin resistance. Front Endocrinol 2013; 4: 71

[15] Pan H, Guo J, Su Z. Advances in understanding the interrelations between leptin resistance and obesity. Physiol Behav 2014; 130: 157–169

[16] Diez JJ, Iglesias P. The role of the novel adipocyte-derived hormone adiponectin in human disease. Eur J Endocrinol 2003; 148: 293–300

[17] Roh SG, Song SH, Choi KC et al. Chemerin – a new adipokine that modulates adipogenesis via its own receptor. Biochem Biophys Res Commun 2007; 362: 1013–1018

[18] Sonoli SS, Shivprasad S, Prasad CV et al. Visfatin – a review. Eur Rev Med Pharmacol Sci 2011; 15: 9–14

[19] Jung RG, Motazedian P, Ramirez FD et al. Association between plasminogen activator inhibitor-1 and cardiovascular events: A systematic review and meta-analysis. Thromb J 2018; 16: 12

[20] Lazar MA. Resistin- and Obesity-associated metabolic diseases. Horm Metab Res 2007; 39: 710–716

[21] Busetto L, Perini P, Giantin V et al. Relationship between energy expenditure and visceral fat accumulation in obese women submitted to adjustable silicone gastric banding (ASGB). Int J Obes Relat Metab Disord 1995; 19: 227–233

[22] Serra A, Granada ML, Romero R et al. The effect of bariatric surgery on adipocytokines, renal parameters and other cardiovascular risk factors in severe and very severe obesity: 1-year follow-up. Clin Nutr (Edinburgh, Scotland) 2006; 25: 400–408

[23] Vendrell J, Broch M, Vilarrasa N et al. Resistin, adiponectin, ghrelin, leptin, and proinflammatory cytokines: Relationships in obesity. Obes Res 2004; 12: 962–971

[24] Arica PC, Aydin S, Zengin U et al. The Effects on obesity related peptides of laparoscopic gastric band applications in morbidly obese patients. J Invest Surg 2018; 31: 89–95

[25] De Luis DA, Izaola O, Conde R et al. Visfatin levels in female, morbid, nondiabetic obese patients after biliopancreatic diversion surgery. Surg Obes Relat Dis 2011; 7: 195–198

[26] Engl J, Bobbert T, Ciardi C et al. Effects of pronounced weight loss on adiponectin oligomer composition and metabolic parameters. Obesity 2007; 15: 1172–1178

[27] Farey JE, Preda TC, Fisher OM et al. Effect of laparoscopic sleeve gastrectomy on fasting gastrointestinal, pancreatic, and adipose-derived hormones and on non-esterified fatty acids. Obes Surg 2017; 27: 399–407

[28] Kotidis EV, Koliakos G, Papavramidis TS et al. The effect of biliopancreatic diversion with pylorus-preserving sleeve gastrectomy and duodenal switch on fasting serum ghrelin, leptin and adiponectin levels: Is there a hormonal contribution to the weight-reducing effect of this procedure? Obes Surg 2006; 16: 554–559

[29] Umeda LM, Pereira AZ, Carneiro G et al. Postprandial adiponectin levels are associated with improvements in postprandial triglycerides after Roux-en-Y gastric bypass in type 2 diabetic patients. Metab Syndr Relat Disord 2013; 11: 343–348

[30] Whitson BA, Leslie DB, Kellogg TA et al. Adipokine response in diabetics and nondiabetics following the Roux-en-Y gastric bypass: A preliminary study. J Surg Res 2007; 142: 295–300

[31] Moher D, Liberati A, Tetzlaff J et al. Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. PLoS Med 2009; 6: e1000097

[32] Wells G, Shea B, O'connell D et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Dept of Epidemiology and Community Medicine, University of Ottawa: Ottawa, Canada. 2011; www.ohri.ca

[33] Wells G, Shea B, O'Connell D et al. The Newcastle Ottawa Scale (NOS) for assessing the quality of nonrandomized studies in meta-analyses. 2011; www.ohri.ca

[34] Borenstein M, Hedges LV, Higgins JP et al. Introduction to Meta-analysis. New York: Wiley; 2011

[35] Higgins JP, Thompson SG, Deeks JJ et al. Measuring inconsistency in meta-analyses. Br Med J 2003; 327: 557

[36] Palmer TM, Sutton AJ, Peters JL et al. Contour-enhanced funnel plots for meta-analysis. STATA J 2008; 8: 242–254

[37] Adami GF, Cordera R, Andraghetti G et al. Changes in serum ghrelin concentration following biliopancreatic diversion for obesity. Obes Res 2004; 12: 684–687

[38] Adami GF, Cordera R, Camerini G et al. Long-term normalization of insulin sensitivity following biliopancreatic diversion for obesity. Int J Obes 2004; 28: 671–673

[39] Adami GF, Cordera R, Campostano A et al. Serum leptin and weight loss in severely obese patients undergoing biliopancreatic diversion. Int J Obes 1998; 22: 822–824

[40] Adami GF, Gradaschi R, Andraghetti G et al. Serum leptin and adiponectin concentration in type 2 diabetes patients in the short and long term following biliopancreatic diversion. Obes Surg 2016; 26: 2442–2448

[41] Alagna S, Cossu ML, Gallo P et al. Biliopancreatic diversion: Long-term effects on gonadal function in severely obese men. Surg Obes Relat Dis 2006; 2: 82–86

[42] Alosco ML, Spitznagel MB, Strain G et al. Improved serum leptin and ghrelin following bariatric surgery predict better postoperative cognitive function. J Clin Neurol (Korea) 2015; 11: 48–56

[43] Arismendi E, Rivas E, Agustí A et al. The systemic inflammome of severe obesity before and after bariatric surgery. PLoS One 2014; 9: e107859

[44] Belligoli A, Sanna M, Serra R et al. Incidence and predictors of hypoglycemia 1 year after laparoscopic sleeve gastrectomy. Obes Surg 2017; 27: 3179–3186

[45] Bobbioni-Harsch E, Morel P, Huber O et al. Energy economy hampers body weight loss after gastric bypass. J Clin Endocrinol Metab 2000; 85: 4695–4700

[46] Brethauer SA, Heneghan HM, Eldar S et al. Early effects of gastric bypass on endothelial function, inflammation, and cardiovascular risk in obese patients. Surg Endoscop Intervent Tech 2011; 25: 2650–2659

[47] Briatore L, Salani B, Andraghetti G et al. Restoration of acute insulin response in T2DM subjects 1 month after biliopancreatic diversion. Obesity 2008; 16: 77–81

[48] Bryant EJ, King NA, Falken Y et al. Relationships among tonic and episodic aspects of motivation to eat, gut peptides, and weight before and after bariatric surgery. Surg Obes Relat Dis 2013; 9: 802–808

[49] Buzga M, Zavadilova V, Holeczy P et al. Dietary intake and ghrelin and leptin changes after sleeve gastrectomy. Videosurg Miniinvas Tech 2014; 9: 554–561

[50] Carroll JF, Franks SF, Smith AB et al. Visceral adipose tissue loss and insulin resistance 6 months after laparoscopic gastric banding surgery: A preliminary study. Obes Surg 2009; 19: 47–55

[51] Catalan V, Gomez-Ambrosi J, Rodriguez A et al. Increased levels of chemerin and its receptor, chemokine-like receptor-1, in obesity are related to inflammation: tumor necrosis factor-alpha stimulates mRNA levels of chemerin in visceral adipocytes from obese patients. Surg Obes Relat Dis 2013; 9: 306–314

[52] Coupaye M, Bouillot JL, Coussieu C et al. One-year changes in energy expenditure and serum leptin following adjustable gastric banding in obese women. Obes Surg 2005; 15: 827–833

[53] Das SK, Roberts SB, McCrory MA et al. Long-term changes in energy expenditure and body composition after massive weight loss induced by gastric bypass surgery 1–4. Am J Clin Nutr 2003; 78: 22–30

[54] De La Torre NG, Rubio MA, Bordiú E et al. Effects of weight loss after bariatric surgery for morbid obesity on vascular endothelial growth factor-A, adipocytokines, and insulin. J Clin Endocrinol Metab 2008; 93: 4276–4281

[55] De Marinis L, Bianchi A, Mancini A et al. Growth hormone secretion and leptin in morbid obesity before and after biliopancreatic diversion: Relationships with insulin and body composition. J Clin Endocrinol Metab 2004; 89: 174–180

[56] Gannage-Yared MH, Yaghi C, Habre B et al. Osteoprotegerin in relation to body weight, lipid parameters insulin sensitivity, adipocytokines, and C-reactive protein in obese and non-obese young individuals: Results from both cross-sectional and interventional study. Eur J Endocrinol 2008; 158: 353–359

[57] García-Fuentes E, García-Almeida JM, García-Arnés J et al. Plasma visfatin concentrations in severely obese subjects are increased after intestinal bypass. Obesity 2007; 15: 2391–2395

[58] Garcia-Unzueta MT, Fernandez-Santiago R, Dominguez-Diez A et al. Fasting plasma ghrelin levels increase progressively after biliopancreatic diversion: One-year follow-up. Obes Surg 2005; 15: 187–190

[59] Guglielmi V, Bellia A, Gentileschi P et al. Parathyroid hormone in surgery-induced weight loss: No glucometabolic effects but potential adaptive response to skeletal loading. Endocrine 2018; 59: 288–295

[60] Guldstrand M, Backman L, Adamson U et al. Lowering of circulating insulin and leptin is closely associated following weight reduction after vertical banded gastroplasty in obese women. Diabetes Obes Metab 1999; 1: 53–55

[61] Haider DG, Schindler K, Schaller G et al. Increased plasma visfatin concentrations in morbidly obese subjects are reduced after gastric banding. J Clin Endocrinol Metab 2006; 91: 1578–1581

[62] Haluzikova D, Lacinova Z, Kavalkova P et al. Laparoscopic sleeve gastrectomy differentially affects serum concentrations of FGF-19 and FGF-21 in morbidly obese subjects. Obesity (Silver Spring. Md) 2013; 21: 1335–1342

[63] Handisurya A, Riedl M, Vila G. Serum vaspin concentrations in relation to insulin sensitivity following RYGB-induced weight loss. Obes Surg 2010; 20: 198–203

[64] Hanusch-Enserer U, Cauza E, Brabant G et al. Plasma ghrelin in obesity before and after weight loss after laparoscopical adjustable gastric banding. J Clin Endocrinol Metab 2004; 89: 3352–3358

[65] Hanusch-Enserer U, Zorn G, Wojta J et al. Non-conventional markers of atherosclerosis before and after gastric banding surgery. Eur Heart J 2009; 30: 1516–1524

[66] Heinonen MV, Purhonen AK, Miettinen P et al. Apelin, orexin-A and leptin plasma levels in morbid obesity and effect of gastric banding. Regulat Pept 2005; 130: 7–13

[67] Holdstock C, Engstrom BE, Ohrvall M et al. Ghrelin and adipose tissue regulatory peptides: Effect of gastric bypass surgery in obese humans. J Clin Endocrinol Metab 2003; 88: 3177–3183

[68] Joao Cabrera E, Valezi AC, Delfino VD et al. Reduction in plasma levels of inflammatory and oxidative stress indicators after Roux-en-Y gastric bypass. Obes Surg 2010; 20: 42–49

[69] Korner J, Inabnet W, Febres G et al. Prospective study of gut hormone and metabolic changes after adjustable gastric banding and Roux-en-Y gastric bypass. Int J Obes 2009; 33: 786–795

[70] Krieger AC, Youn H, Modersitzki F et al. Effects of laparoscopic adjustable gastric banding on sleep and metabolism: A 12-month follow-up study. Int J Gen Med 2012; 5: 975–981

[71] Laimer M, Ebenbichler CF, Kaser S et al. Weight loss increases soluble leptin receptor levels and the soluble receptor bound fraction of leptin. Obes Res 2002; 10: 597–601

[72] Lambert G, Lima MMO, Felici AC et al. Early regression of carotid intima-media thickness after bariatric surgery and its relation to serum leptin reduction. Obes Surg 2018; 28: 226–233

[73] Lima MM, Pareja JC, Alegre SM et al. Acute effect of roux-en-y gastric bypass on whole-body insulin sensitivity: A study with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab 2010; 95: 3871–3875

[74] Lin E, Phillips LS, Ziegler TR et al. Increases in adiponectin predict improved liver, but not peripheral, insulin sensitivity in severely obese women during weight lossIncreases in adiponectin predict improved liver, but not peripheral, insulin sensitivity in severely obese women during weight loss. Diabetes 2007; 56: 735–742

[75] Liou JM, Lin JT, Lee WJ et al. The serial changes of ghrelin and leptin levels and their relations to weight loss after laparoscopic minigastric bypass surgery. Obes Surg 2008; 18: 84–89

[76] Magkos F, Fabbrini E, McCrea J et al. Decrease in hepatic very-low-density lipoprotein-triglyceride secretion after weight loss is inversely associated with changes in circulating leptin. Diabetes Obes Metab 2010; 12: 584–590

[77] Manco M, Fernandez-Real JM, Valera-Mora ME et al. Massive weight loss decreases corticosteroid-binding globulin levels and increases free cortisol in healthy obese patients: An adaptive phenomenon? Diabetes Care 2007; 30: 1494–1500

[78] Marantos G, Daskalakis M, Karkavitsas N et al. Changes in metabolic profile and adipoinsular axis in morbidly obese premenopausal females treated with restrictive bariatric surgery. World J Surg 2011; 35: 2022–2023

[79] Mazidi M, Gao HK, Li L et al. Changes in inflammatory and cardiometabolic profile after Roux-en-Y gastric bypass: A prospective study in an overweight chinese cohort. Bariatr Surg Pract Patient Care 2017; 12: 45–48

[80] Molin Netto BD, Earthman CP, Cravo Bettini S et al. Early effects of Roux-en-Y gastric bypass on peptides and hormones involved in the control of energy balance. Eur J Gastroenterol Hepatol 2016; 28: 1050–1055

[81] Mora M, Aranda GB, De Hollanda A et al. Weight loss is a major contributor to improved sexual function after bariatric surgery. Surg Endosc Intervent Tech 2013; 27: 3197–3204

[82] Navaneethan SD, Kelly KR, Sabbagh F et al. Urinary albumin excretion, HMW adiponectin, and insulin sensitivity in type 2 diabetic patients undergoing bariatric surgery. Obes Surg 2010; 20: 308–315

[83] Nijhuis J, van Dielen FM, Buurman WA et al. Ghrelin, leptin and insulin levels after restrictive surgery: A 2-year follow-up study. Obes Surg 2004; 14: 783–787

[84] Olmos JM, Vazquez LA, Amado JA et al. Mineral metabolism in obese patients following vertical banded gastroplasty. Obes Surg 2008; 18: 197–203

[85] Perathoner A, Weißenbacher A, Sucher R et al. Significant weight loss and rapid resolution of diabetes and dyslipidemia during short-term follow-up after laparoscopic sleeve gastrectomy. Obes Surg 2013; 23: 1966–1972

[86] Pérez-Romero N, Serra A, Granada ML et al. Effects of two variants of Roux-en-Y gastric bypass on metabolism behaviour: Focus on plasma ghrelin concentrations over a 2-year follow-up. Obes Surg 2010; 20: 600–609

[87] Raftopoulos I, Bernstein B, O'Hara K et al. Protein intake compliance of morbidly obese patients undergoing bariatric surgery and its effect on weight loss and biochemical parameters. Surg Obes Relat Dis 2011; 7: 733–742

[88] Terra X, Auguet T, Guiu-Jurado E et al. Long-term changes in leptin, chemerin and ghrelin levels following different bariatric surgery procedures: Roux-en-Y gastric bypass and sleeve gastrectomy. Obes Surg 2013; 23: 1790–1798

[89] Tschoner A, Sturm W, Engl J et al. Plasminogen activator inhibitor 1 and visceral obesity during pronounced weight loss after bariatric surgery. Nutr Metab Cardiovasc Dis 2012; 22: 340–346

[90] Urbanavičius V, Abalikšta T, Brimas G et al. Comparison of changes in blood glucose, insulin resistance indices, and adipokine levels in diabetic and nondiabetic subjects with morbid obesity after laparoscopic adjustable gastric banding. Medicina (Lithuania) 2013; 49: 9–14

[91] Yadav R, Hama S, Liu Y et al. Effect of Roux-en-Y bariatric surgery on lipoproteins, insulin resistance, and systemic and vascular inflammation in obesity and diabetes. Front Immunol 2017; 8: 1512

[92] Bachmayer C, Lammert A, Hasenberg T et al. Healthy obese and post bariatric patients – Metabolic and vascular patterns. Exp Clin Endocrinol Diabetes 2013; 121: 483–487

[93] Broch M, Gomez JM, Auguet MT et al. Association of retinol-binding protein-4 (RBP4) with lipid parameters in obese women. Obes Surg 2010; 20: 1258–1264

[94] Carrasco F, Basfi-Fer K, Rojas P et al. Changes in bone mineral density after sleeve Gastrectomy or gastric bypass: Relationships with variations in vitamin D, ghrelin, and adiponectin levels. Obes Surg 2014; 24: 877–884

[95] Couce ME, Cottam D, Esplen J et al. Is ghrelin the culprit for weight loss after gastric bypass surgery? A negative answer. Obes Surg 2006; 16: 870–878

[96] Coughlin CC, Finck BN, Eagon JC et al. Effect of marked weight loss on adiponectin gene expression and plasma concentrations. Obesity (Silver Spring. Md) 2007; 15: 640–645

[97] de Luis DA, Pacheco D, Aller R et al. Influence of G308A polymorphism of tumor necrosis factor alpha gene on surgical results of biliopancreatic diversion. Obes Surg 2010; 20: 221–225

[98] Domienik-Karlowicz J, Rymarczyk Z, Dzikowska-Diduch O et al. Emerging markers of atherosclerosis before and after bariatric surgery. Obes Surg 2015; 25: 486–493

[99] Fellici AC, Lambert G, Lima MM et al. Surgical treatment of type 2 diabetes in subjects with mild obesity: Mechanisms underlying metabolic improvements. Obes Surg 2015; 25: 36–44

[100] Hosseinzadeh-Attar MJ, Golpaie A, Janani L et al. Effect of weight reduction following bariatric surgery on serum visfatin and adiponectin levels in morbidly obese subjects. Obes Facts 2013; 6: 193–202

[101] Illan-Gomez F, Gonzalvez-Ortega M, Orea-Soler I et al. Obesity and inflammation: Change in adiponectin, C-reactive protein, tumour necrosis factor-alpha and interleukin-6 after bariatric surgery. Obes Surg 2012; 22: 950–955

[102] Kopp HP, Krzyzanowska K, Mohlig M et al. Effects of marked weight loss on plasma levels of adiponectin, markers of chronic subclinical inflammation and insulin resistance in morbidly obese women. Int J Obes 2005; 29: 766–771

[103] Lammert A, Hasenberg T, Kraupner C et al. Improved arteriole-to-venule ratio of retinal vessels resulting from bariatric surgery. Obesity (Silver Spring. Md) 2012; 20: 2262–2267

[104] Linscheid P, Christ-Crain M, Stoeckli R et al. Increase in high molecular weight adiponectin by bariatric surgery-induced weight loss. Diabetes Obes Metab 2008; 10: 1266–1270

[105] Schmatz R, Zanini D, Gutierres JM et al. Evaluation of the biochemical, inflammatory and oxidative profile of obese patients given clinical treatment and bariatric surgery. Clin Chim Acta 2017; 465: 72–79

[106] Tirado R, Masdeu MJ, Vigil L et al. Impact of bariatric surgery on heme oxygenase-1, inflammation, and insulin resistance in morbid obesity with obstructive sleep apnea. Obes Surg 2017; 27: 2338–2346

[107] De Luis DA, Terroba MC, Cuellar L et al. Resistin levels in morbid obese patients following the biliopancreatic diversion surgery. Horm Metab Res 2011; 43: 205–208

[108] Auguet T, Terra X, Hernández M et al. Clinical and adipocytokine changes after bariatric surgery in morbidly obese women. Obesity 2014; 22: 188–194

[109] Botella-Carretero JI, Luque-Ramirez M, Alvarez-Blasco F et al. The increase in serum visfatin after bariatric surgery in morbidly obese women is modulated by weight loss, waist circumference, and presence or absence of diabetes before surgery. Obes Surg 2008; 18: 1000–1006

[110] Baena-Fustegueras JA, Pardina E, Balada E et al. Soluble CD40 ligand in morbidly obese patients: effect of body mass index on recovery to normal levels after gastric bypass surgery. JAMA Surg 2013; 148: 151–156

[111] Kim MK, Jang EH, Hong OK et al. Changes in serum levels of bone morphogenic protein 4 and inflammatory cytokines after bariatric surgery in severely obese Korean patients with type 2 diabetes. Int J Endocrinol 2013; 681205

[112] Parlee SD, Wang Y, Poirier P et al. Biliopancreatic diversion with duodenal switch modifies plasma chemerin in early and late post-operative periods. Obesity 2015; 23: 1201–1208

[113] Hu E, Liang P, Spiegelman BM. AdipoQ is a novel adipose-specific gene dysregulated in obesity. J Biol Chem 1996; 271: 10697–10703

[114] Achari A, Jain S. Adiponectin, a therapeutic target for obesity, diabetes, and endothelial dysfunction. Int J Mol Sci 2017; 18: 1321

[115] Hotta K, Funahashi T, Arita Y et al. Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. Arteriosc Thromb Vascul Biol 2000; 20: 1595–1599

[116] Yamauchi T, Kadowaki T. Physiological and pathophysiological roles of adiponectin and adiponectin receptors in the integrated regulation of metabolic and cardiovascular diseases. Int J Obes 2009; 32: S13

[117] Combs TP, Wagner JA, Berger J et al. Induction of adipocyte complement-related protein of 30 kilodaltons by PPARγ agonists: A potential mechanism of insulin sensitization. Endocrinology 2002; 143: 998–1007

[118] Phillips SA, Ciaraldi TP, Kong AP et al. Modulation of circulating and adipose tissue adiponectin levels by antidiabetic therapy. Diabetes 2003; 52: 667674

[119] Xu A, Wang H, Hoo RL et al. Selective elevation of adiponectin production by the natural compounds derived from a medicinal herb alleviates insulin resistance and glucose intolerance in obese mice. Endocrinology 2009; 150: 625–633

[120] Mohammadi A, Gholamhoseinian A, Fallah H. Zataria multiflora increases insulin sensitivity and PPARγ gene expression in high fructose fed insulin resistant rats. Iran J Basic Med Sci 2014; 17: 263

[121] Jürimäe J, Hofmann P, Jürimäe T et al. Plasma adiponectin response to sculling exercise at individual anaerobic threshold in college level male rowers. Int J Sports Med 2006; 27: 272–277

[122] Jürimäe J, Purge P, Jürimäe T. Adiponectin is altered after maximal exercise in highly trained male rowers. Eur J Appl Physiol 2005; 93: 502–505

[123] Kriketos AD, Gan SK, Poynten AM et al. Exercise increases adiponectin levels and insulin sensitivity in humans. Diabetes Care 2004; 27: 629–630

[124] Geagea AG, Mallat S, Matar CF et al. Adiponectin and inflammation in health and disease: An update. Open Med J 2018; 5: Publisher Id: MEDJ-5-20. DOI: 10.2174/1874220301805010020

[125] Kelesidis T, Kelesidis I, Chou S et al. Narrative review: The role of leptin in human physiology: Emerging clinical applications. Ann Inter Med 2010; 152: 93–100

[126] Paz-Filho G, Mastronardi C, Franco CB et al. Leptin: Molecular mechanisms, systemic pro-inflammatory effects, and clinical implications. Arquiv Brasil. Endocrinol Metab 2012; 56: 597–607

[127] Pan WW, Myers MG Jr. Leptin and the maintenance of elevated body weight. Nat. Rev Neurosci 2018; 19: 95

[128] Facey A, Dilworth L, Irving R. A review of the leptin hormone and the association with obesity and diabetes mellitus. J Diabetes Metab 2017; 8. DOI: 10.4172/2155-6156.1000727

[129] Fontes-Villalba M, Lindeberg S, Granfeldt Y et al. Palaeolithic diet decreases fasting plasma leptin concentrations more than a diabetes diet in patients with type 2 diabetes: A randomised cross-over trial. Cardiovasc Diabetol 2016; 15: 80

[130] Somodi S, Seres I, Lőrincz H et al. Plasminogen activator inhibitor-1 level correlates with lipoprotein subfractions in obese nondiabetic subjects. Int J Endocrinol. 2018; 9596054. doi: 10.1155/2018/9596054 eCollection 2018

[131] Kruithof EK, Gudinchet A, Bachmann F. Plasminogen activator inhibitor 1 and plasminogen activator inhibitor 2 in various disease states. Thromb Haemost 1988; 59: 007–012

[132] Chen R, Yan J, Liu P et al. Plasminogen activator inhibitor links obesity and thrombotic cerebrovascular diseases: The roles of PAI-1 and obesity on stroke. Metab Brain Dis 2017; 32: 667–673

[133] Lerman RH, Desai A, Lamb JJ et al. A phytochemical-rich multivitamin-multimineral supplement is bioavailable and reduces serum oxidized low-density lipoprotein, myeloperoxidase, and plasminogen activator inhibitor-1 in a four-week pilot trial of healthy individuals. Glob Adv Health Med 2014; 3: 34–39

[134] Jamaluddin MS, Weakley SM, Yao Q et al. Resistin: functional roles and therapeutic considerations for cardiovascular disease. Br J Pharmacol 2012; 165: 622–632

[135] McTernan PG, Kusminski CM, Kumar S. Resistin. Curr Opin Lipidol 2006; 17: 170–175

[136] Chang YH, Chang DM, Lin KC et al. Visfatin in overweight/obesity, type 2 diabetes mellitus, insulin resistance, metabolic syndrome and cardiovascular diseases: A meta-analysis and systemic review. Diabetes Metab Res Rev 2011; 27: 515–527

[137] Stastny J, Bienertova-Vasku J, Vasku A. Visfatin and its role in obesity development. Diabetes Metab Syndr 2012; 6: 120–124

[138] Ferland DJ, Watts SW. Chemerin: A comprehensive review elucidating the need for cardiovascular research. Pharmacol Res 2015; 99: 351–361

[139] Roman AA, Parlee SD, Sinal CJ. Chemerin: A potential endocrine link between obesity and type 2 diabetes. Endocrine 2012; 42: 243–251

[140] Helfer G, Wu QF. Chemerin: A multifaceted adipokine involved in metabolic disorders. J Endocrinol 2018; 238: R79–R94