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Article

Follow-Up Study of Hormones, Interlukines, Total Antioxidant Capacity, and Oxidative Stress in Sleeve Gastrectomy

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Abstract: This prospective follow-up study was designed to evaluate the metabolic changes in patients undergoing gastric sleeve surgery. The results reported a substantial improvement at ($p \leq 0.05$) in the level of T-AOC in the G3 (after 8 months) when compared with G1 (after 3 months) and G2 (after 6 months). A considerable diminution at ($p \leq 0.05$) in the level of TOS was also detected in the third group versus G1and G2 groups. Moreover, the levels of appetite regulating hormones, including ghrelin and leptin showed a significant decrease at ($p \leq 0.05$) in the levels of these hormones in G3 group when compared with the G1and G2 groups. These data imply a progressive, time- dependent improvement in redox balance and hormonal variation in patients over time following sleeve gastrectomy.

Keywords: Sleeve Gastrectomy, ghrelin, leptin, T-AOc , TOS, IL-17, and IL34

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1. Introduction

Obesity is a changeable variable pathophysiological state marked by not only by adipose tissue expansion but also by significant dysregulation in hormonal regulation, inflammatory signaling, and antioxidant-oxidant equilibrium. persistent subclinical inflammation and increased oxidative stress are cardinal characteristics of obesity, underpinning impaired insulin sensitivity metabolic impairment, and the advancement of obesity-related concurrent disorders [1]. Adipose tissue serves as a dynamic secretory organ and immunological organ, elaborating a wide range of adipose-derived mediators that impair whole-body metabolic equilibrium [2]. Among the key mediators involved in obesity-associated metabolic dysregulation are orexigenic/anorexigenic mediators and inflammatory interleukins[3]. Leptin, produced by adipocytes, has been correlated with adiposity and influences satiety-related pathways [4], [5]. Ghrelin, the peptide responsible for hunger sensation and predominantly produced in the fundic region of the stomach, plays the central role in the stimulation of hunger and energy balance. The effects of excessive calorie ingestion and the development of adiposity are the result of the disruption in the production of ghrelin and the development of resistance to the action of leptin[6]. In this context, immune-activating factors such as interleukin-17 (IL-17) and interleukin-34 (IL-34) are now recognized to be the key factors in the development of obesity-related inflammatory conditions, which facilitate the activation of leukocytes and the deterioration of adipose tissue [7], [8]. The second important factor in the development of obesity-related pathological conditions is oxidative stress. Adipose tissue expansion and Nutrient overload amplify reactive oxygen species generation, while weakening antioxidant defenses, which causes cytotoxic injury and further potentiation of inflammatory pathways[9]. An imbalance between oxidant and antioxidants worsens metabolic disorders and accelerates cardiovascular problems linked to obesity [10]. one of the most efficacious treatment modalities for severe obesity is gastric sleeve procedure, through which significant and long-term weight reduction [11]. This procedure not only reduces food intake but also triggers important hormonal and inflammatory changes[12]. Ghrelin secretion is decreased by the removal of gastric fundus, while fat loss improves leptin levels reduces systematic inflammation and enhances oxidative balance[13]. However, the chronological progression of these functional modifications remains insufficiently characterized. While few studies have reported metabolic improvements following bariatric surgery, majority of these studies depend on brief evaluation periods or focus on individual biochemical markers. Insufficient data is available from prospective sequential studies that concurrently assess hormonal, inflammatory, and oxidative stress markers at sequential assessment intervals [14], [15]. Such an in-depth time-dependent assessment is of vital importance in order to comprehend the progression, sustainability, and interrelation of metabolic recovery mechanisms in patients undergoing sleeve gastrectomy surgery. Accordingly, the novelty of the present study is based on the unified prospective assessment of appetite-regulating hormones (ghrelin and leptin), pro-inflammatory interleukins (IL-17 and IL-34), and markers of oxidative stress during the navigation time points (3, 6, and 8 months) after sleeve gastrectomy surgery. This study aims to clearly describe how metabolic recovery change over time and to better understanding the hormonal, inflammatory, and oxidative changes caused by bariatric surgery. A detailed time-course course of metabolic recovery is provided, and the coordinated endocrine adaptations, and inflammatory are examined to explain how they are triggered by the surgery.

2. Materials and Methods

Study Population

A total of (90) participants were divided into groups based on the length of their follow-up; the same patients were followed at:

G1(3 months group no 30), G2(6 months group no 30) and G1(8 months group no 30)

Inclusion criteria included adult's men aged (18–40) years.

Exclusion criteria included chronic inflammatory diseases, malignancy, renal or hepatic failure, and use of anti-inflammatory or hormonal medications.

Biochemical and Hormonal Analysis

Hormonal parameters: Leptin and Ghrelin were measured using ELISA kits (Melsin) according to the manufacturer's instructions. Sensitivity, the minimum detectable dose of Interleukin 34 (IL-34) is typically less than 10 pg/mL.

Inflammatory markers: IL-17 and IL-34 were quantified using sandwich ELISA technique (Melsin).

Sensitivity, the minimum detectable dose of Interleukin 17 (IL-17) is typically less than 1.0 pg/mL.

TOS determination using Erel method and T-AOC determination using solarbio kit ELISA.

3. Results and Discussion

Table 1: Comparison of Follow-up T-AOC, TOS in Bariatric Surgery group at 3, 6, and 8 months, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameters	(Mean±SD)		
	3 months(G1)	6 months(G2)	8 months(G3)
T-AOC(μmol/ml)	0.093 ± 0.011 [a]	0.126 ± 0.0163 [a]	1.004 ± 0.680 [b]
TOS(μmol/l)	40.874 ± 6.298 [a]	23.433 ± 7.152 [b]	6.543 ± 3.355 [c]

The data demonstrated of Table (1) showed a meaningful statistical variation increase in T-AOC levels in period of 8 months against (3,6 months), while marked reduction in TOS levels in period of 8 months when compared with (6,8 months), as presented in Fig (1), these findings stem from a surgical recovery associated decline in redox imbalance across postoperative intervals, which might be explained by body weight reduction, enhanced metabolic regulation, and reduced adipose mediated reactive oxygen species generation. Initial post-surgical intervals often show a state of augmented pro-oxidant activity, as seen in surgical trauma and acute metabolic responses, consistent with the observed increase in Total Oxidant Status (TOS) levels at 3 months. Conversely, Total Antioxidant Capacity (T-AOC) levels are seen to decrease within the first 3 months and increase at 6 and 8 months, peaking at 8 months. The increase in T-AOC levels may be attributed to the enhancement of antioxidant defense mechanisms, as seen in the alleviation of generalized inflammatory responses, nutritional improvements, and metabolic regulation, all of which are consequences of significant weight loss. The temporal profile of T-AOC in the postoperative period after bariatric surgery reveals time-dependent variations within the different periods of follow-up. In the early postoperative period, there were decreased T-AOC levels, as seen in rapid weight loss, metabolic adaptation, and surgical stress. However, at around 8 months, T-AOC levels increase because of improved nutritional uptake, redox balance, and enhancement of antioxidant defense mechanisms. The time-dependent variations of T-AOC are seen, as levels initially decrease because of metabolic strain and then increase because of nutritional improvements and enhancement of antioxidant defense mechanisms.

This progression of events may be attributed to prospective studies that show progressive improvements in redox balance within mid-range periods of follow-up after bariatric surgery, as seen in this study, and the need for continuous monitoring of redox status to assess metabolic improvements. This study supports previous evaluations, as seen in previous studies [14], [16], [14], [17], [2], [3], [18], [19], [20], [21], [22], [18].

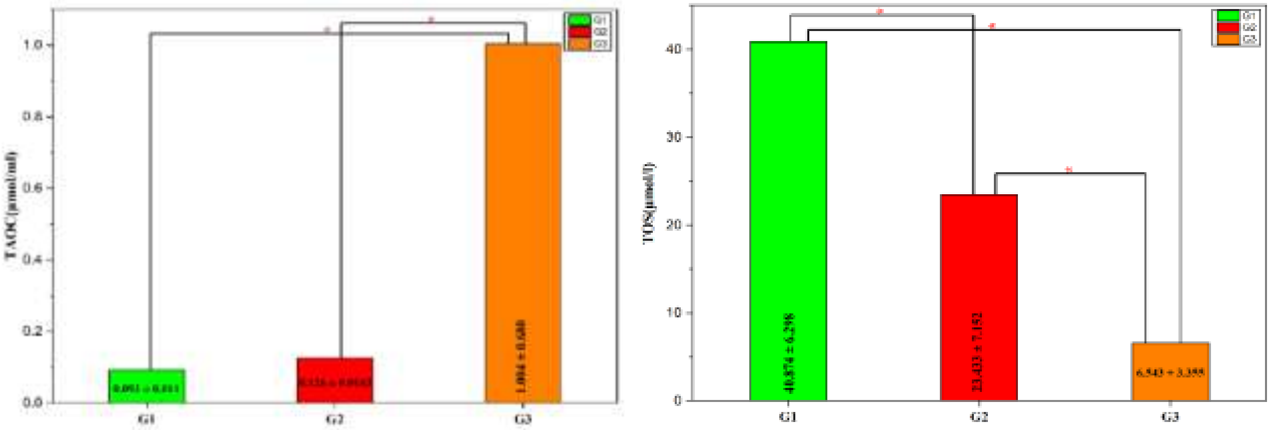

Figure (1): Longitudinal changes in T-AOC, TOS at 3, 6, and 8 months following bariatric surgery.

Table 2: Comparison of Follow-up Ghrelin,Leptin levels in Bariatric Surgery group at 3, 6, and 8 months, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameters	(Mean±SD)			P-Value
	3 months(G1)	6 months(G2)	8 months(G3)	
Ghrelin (pg/ml)	201.750±40.638 [a]	144.250±16.459 [b]	101.250±18.228 [c]	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.002
Leptin (ng/ml)	37.822±7.732 [a]	22.770±3.787 [b]	12.655±1.794 [c]	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.000

As shown in the results presented in Table 2, there was a significant reduction in ghrelin and leptin levels sequentially in all intervals of time, i.e., 3, 6, and 8 months, as shown in Figure 2. The Ghrelin level decreased from the third month to the sixth month, and this reduction continued up to the eighth month. The ghrelin hormone level continued to decrease, and this reduction in ghrelin levels is attributed to long-term structural and functional changes in the stomach. The ghrelin hormone level continues to decrease as a long-term adaptation to maintain appetite and stabilize weight, as shown in references [23], [24], and [25].

As shown in the results, the leptin hormone level decreased from the third month to the sixth month and then to the eighth month. In the third month, the leptin hormone level was high, and this high level of leptin persisted up to the sixth month. The leptin hormone level decreased significantly from the sixth month to the eighth month, and this reduction in the leptin hormone level is attributed to long-term adaptation and significant loss of body fat and improvement in insulin levels, as shown in references [26], [27], [28], and [29].

As shown in the results, bariatric surgery affects hormonal levels, and this effect of bariatric surgery on hormonal levels is shown by the gradual reduction in ghrelin and leptin hormone levels. The gradual reduction in ghrelin and leptin hormone levels helps in the adaptation of the body and maintains a stable weight. The reduction in ghrelin, i.e., the hunger hormone, and the normalization of leptin, i.e., the satiety hormone, help in maintaining a stable hormonal environment and result in long-term weight loss and a reduced tendency for weight regain. A coordinated endocrine remodeling is observed, where orexigenic signaling is reduced and satiety signaling is improved, resulting in better long term metabolic outcomes.

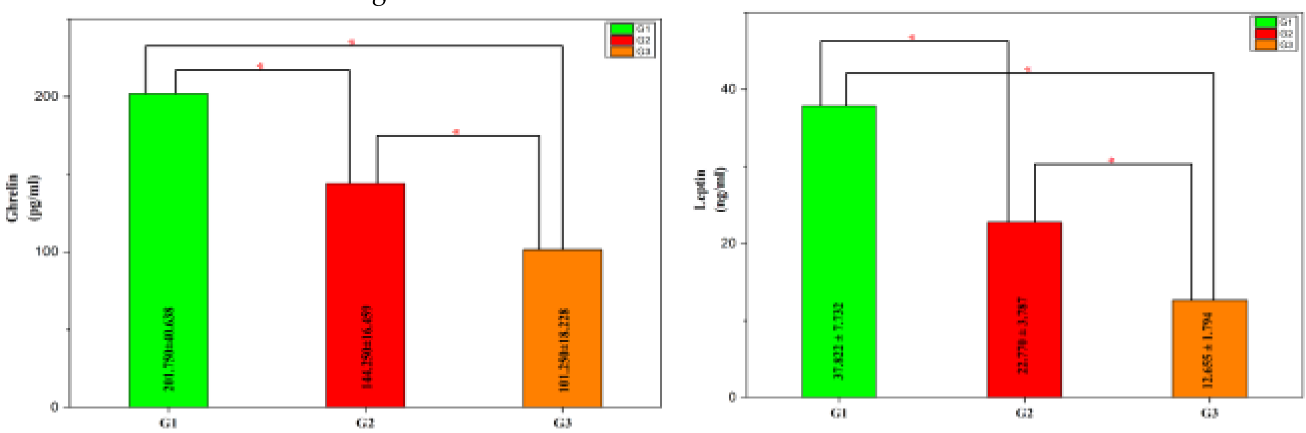

Figure (2): changes in Ghrelin and Leptin at 3, 6, and 8 months following bariatric surgery.

Table 3: Comparison of Follow-up IL-17,IL-34 levels in Bariatric Surgery group at 3, 6, and 8 months, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameters	(Mean±SD)			P-Value
	3 months(G1)	6 months(G2)	8 months(G3)	
IL-17 (pg/ml)	212.499±72.943 [a]	62.286±25.964 [b]	14.539±7.127 [c]	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.025
IL-34 (pg/ml)	297.572±140.677 [a]	113±39.414 [b]	23.857±9.736 [c]	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.026

The present study established statistically meaningful variations and reductions in IL-**17** and IL-34 levels at each postoperative period interval (3, 6, and 8 months), with between-group analyses showing significant differences (p ≤ 0.05), as indicated in Fig (3). Obesity-associated inflammation and acute post-surgical immune and metabolic stress, reflecting residual at 3 months post-surgery, IL-17 and IL-34 levels remained relatively elevated. Fat tissue restructuring and immune readjustment are not yet fully achieved at this initial postoperative stage, while substantial weight loss has been initiated.

Both cytokines showed a marked reduction by 6 months, indicating significant attenuation of generalized inflammatory activity. This diminishment aligns with continuous fat mass reduction, recovered insulin pathway activity, and decreased macrophage activation and T-helper 17 cell-mediated response throughout adipose tissue.

The midterm attenuation demonstrates that bariatric surgery induces effective immuno-physiological reorganization independent of simple caloric restriction. IL-17 and IL-34 achieved minimum recorded measurements and observed values at 8 months, approximating levels typically observed in non-obese individuals or physically active individuals. This substantial reduction reflects that immuno-physiological homeostasis has been reinstated and sustained immunological stimulation has undergone marked suppression; this result assessment with [30] [31].

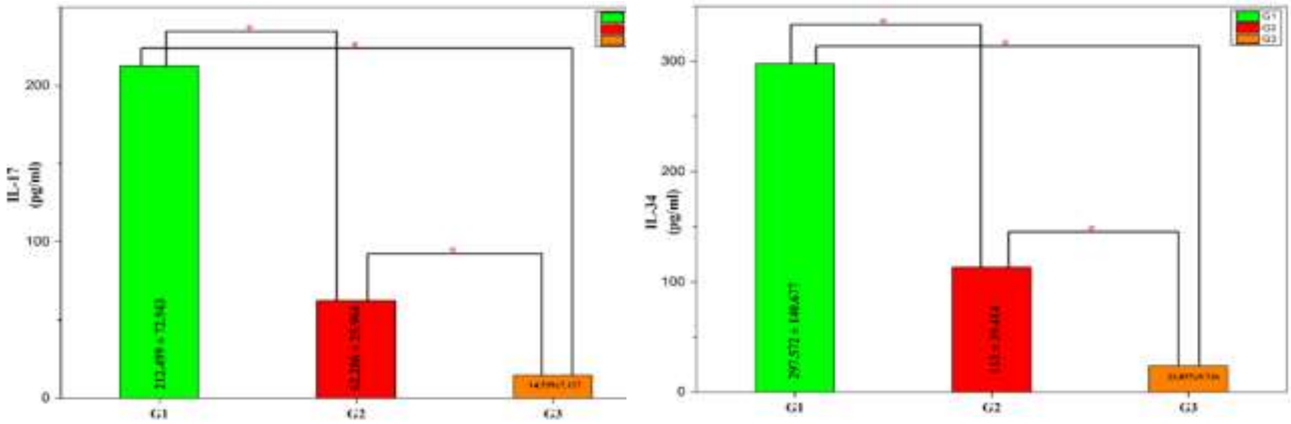

Figure (3): changes in IL-17, IL-34 at 3, 6, and 8 months following bariatric surgery.

4. Conclusion

This longitudinal follow-up study offers substantial evidence that bariatric surgery, or specifically sleeve gastrectomy, results in progressive and coordinated improvements in oxidative stress status, hormonal regulation, and inflammation. An increase in total antioxidant capacity (TAOC) with a significant decrease in total oxidant status (TOS), with significant improvements evident by the eighth month post-operatively, indicates progressive improvements in oxidative stress status. These findings also confirm that resolution of oxidative stress following bariatric surgery is time dependent.

Significant decreases in ghrelin and leptin levels were evident at the 3-month, 6-month, and 8-month follow-ups. The progressive decrease in leptin levels is consistent with adipose tissue loss and increased sensitivity to leptin. The suppression of ghrelin levels is consistent with the resection of the gastric fundus. This suppression is likely responsible for long-term regulation of appetite. The suppression of ghrelin levels is also likely responsible for long-term regulation of appetite.

Bariatric surgery also had a significant anti-inflammatory effect, with significant decreases in pro-inflammatory cytokines IL-17 and IL-34. By the 8th month post-operatively, cytokine levels returned almost to normal levels, indicating effective immunometabolism reprogramming with almost complete resolution of inflammation.

These findings demonstrate the advantages of bariatric surgery for multi-system, including oxidative, inflammatory, and endocrine pathways, in addition to weight loss. The longitudinal approach used in this study captures the temporal dynamics of metabolic recovery and highlights the importance of extended postoperative observation. The combined assessment of biomarker provides important insight into how metabolic improvement continuous over time, showing that Bariatric surgery is a powerful treatment for long-term obesity management. Important mechanistic insights are also provided through biomarker evaluation, and long-term metabolic improvement is supported by bariatric surgery as an effective intervention.

REFERENCES

[1] R. Barazzoni, G. Gortan Cappellari, M. Ragni, and E. Nisoli, “INSULIN RESISTANCE IN OBESITY-An overview of fundamental alterations.”

[2] E. Martínez-Martínez and V. Cachofeiro, “Oxidative Stress in Obesity,” Apr. 01, 2022, *MDPI*. doi: 10.3390/antiox11040639.

[3] S. Furukawa *et al.*, “Increased oxidative stress in obesity and its impact on metabolic syndrome,” *J. Clin. Invest.*, vol. 114, no. 12, pp. 1752–1761, 2017.

[4] T. D. Müller *et al.*, “Ghrelin,” *Mol. Metab.*, vol. 4, no. 6, pp. 437–460, Jun. 2015, doi: 10.1016/J.MOLMET.2015.03.005.

[5] J. Nijhuis, F. M. H. van Dielen, W. A. Buurman, and J. W. M. Greve, “Ghrelin, Leptin and Insulin Levels after Restrictive Surgery: a 2-Year Follow-up Study,” *Obes. Surg.*, vol. 14, no. 6, pp. 783–787, Jun. 2004, doi: 10.1381/0960892041590980.

[6] P. Kalinowski *et al.*, “Ghrelin, leptin, and glycemic control after sleeve gastrectomy versus Roux-en-Y gastric bypass—results of a randomized clinical trial,” *Surgery for Obesity and Related Diseases*, vol. 13, no. 2, pp. 181–188, Feb. 2017, doi: 10.1016/J.SOARD.2016.08.025.

[7] L. A. Zúñiga *et al.*, “IL-17 regulates adipogenesis, glucose homeostasis, and obesity,” *The Journal of Immunology*, vol. 185, no. 11, pp. 6947–6959, 2010.

[8] J. Shang, Y. Xu, S. Pu, X. Sun, and X. Gao, “Role of IL-34 and its receptors in inflammatory diseases,” *Cytokine*, vol. 171, p. 156348, Nov. 2023, doi: 10.1016/J.CYTO.2023.156348.

[9] E. Colak and D. Pap, “The role of oxidative stress in the development of obesity and obesity-related metabolic disorders,” 2021, *Society of Medical Biochemists of Serbia*. doi: 10.5937/jomb0-24652.

[10] A. Azlan, S. Sultana, C. S. Huei, and M. R. Razman, “Antioxidant, Anti-Obesity, Nutritional and Other Beneficial Effects of Different Chili Pepper: A Review,” Feb. 01, 2022, *MDPI*. doi: 10.3390/molecules27030898.

[11] K. Fay and A. Patel, “Bariatric Surgery,” in *Anatomic, Physiologic, and Therapeutic Principles of Surgical Diseases*, Cham: Springer International Publishing, 2023, pp. 173–189. doi: 10.1007/978-3-031-25596-0_12.

[12] O. Bulur *et al.*, “Nutritional and hematological changes after sleeve gastrectomy,” *Rom. J. Diabetes Nutr. Metab. Dis.*, vol. 28, no. 4, pp. 343–351, 2021.

[13] X. Terra *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.*, vol. 23, no. 11, pp. 1790–1798, Nov. 2013, doi: 10.1007/s11695-013-1033-9.

[14] S. Şimşek, B. E. R. URGANCI, S. Yilmaz, M. R. Aykota, T. Sari, and İ. AÇIKBAŞ, “One-year follow-up of the oxidative stress profile of patients after laparoscopic sleeve gastrectomy.,” *Eur. Rev. Med. Pharmacol. Sci.*, vol. 27, no. 21, 2023.

[15] T. Min, S. L. Prior, G. Dunseath, R. Churm, J. D. Barry, and J. W. Stephens, "Temporal Effects of Bariatric Surgery on Adipokines, Inflammation and Oxidative Stress in Subjects with Impaired Glucose Homeostasis at 4 Years of Follow-up," *Obes. Surg.*, vol. 30, no. 5, pp. 1712–1718, May 2020, doi: 10.1007/s11695-019-04377-3.

[16] M. M. A. Abulmeaty *et al.*, "Inflammatory Cytokines, Redox Status, and Cardiovascular Diseases Risk after Weight Loss via Bariatric Surgery and Lifestyle Intervention," *Medicina (B. Aires).*, vol. 59, no. 4, p. 751, Apr. 2023, doi: 10.3390/medicina59040751.

[17] G. de Souza Bett *et al.*, "Bariatric Surgery Reduces Lipid Profile and Oxidative Stress in Patients With Obesity: A Prospective Cohort Study," *Journal of Metabolic and Bariatric Surgery*, vol. 14, no. 1, p. 32, 2025, doi: 10.17476/jmbs.2025.14.1.32.

[18] aniar Ghazizadeh *et al.*, "Definition of an oxidative stress status by combined assessment of Malondialdehyde and Oxidized-LDL: A study in patients with type2 diabetes and control," *Meta Gene*, vol. 19, 2019, doi: 10.1016/j.mgene.2018.10.005.

[19] J. Gutiérrez-Hellín *et al.*, "Research Trends in the Effect of Caffeine Intake on Fat Oxidation: A Bibliometric and Visual Analysis," *Nutrients*, vol. 15, no. 20, p. 4320, Oct. 2023, doi: 10.3390/nu15204320.

[20] A. Metere, C. E. Graves, D. Pietraforte, and G. Casella, "The Effect of Sleeve Gastrectomy on Oxidative Stress in Obesity," *Biomedicines*, vol. 8, no. 6, p. 168, Jun. 2020, doi: 10.3390/biomedicines8060168.

[21] M. M. Bayliak, O. B. Abrat, J. M. Storey, K. B. Storey, and V. I. Lushchak, "Interplay between diet-induced obesity and oxidative stress: Comparison between Drosophila and mammals," *Comp. Biochem. Physiol. A Mol. Integr. Physiol.*, vol. 228, pp. 18–28, Feb. 2019, doi: 10.1016/J.CBPA.2018.09.027.

[22] P. Chaudhary *et al.*, "Oxidative stress, free radicals and antioxidants: potential crosstalk in the pathophysiology of human diseases," *Front. Chem.*, vol. 11, May 2023, doi: 10.3389/fchem.2023.1158198.

[23] F. B. Langer *et al.*, "Sleeve Gastrectomy and Gastric Banding: Effects on Plasma Ghrelin Levels," *Obes. Surg.*, vol. 15, no. 7, pp. 1024–1029, Aug. 2005, doi: 10.1381/0960892054621125.

[24] G. Sharma, P. S. Nain, P. Sethi, A. Ahuja, and S. Sharma, "Plasma ghrelin levels after laparoscopic sleeve gastrectomy in obese individuals," *Indian Journal of Medical Research*, vol. 149, no. 4, pp. 544–547, Apr. 2019, doi: 10.4103/ijmr.IJMR_984_18.

[25] P. Kalinowski *et al.*, "Ghrelin, leptin, and glycemic control after sleeve gastrectomy versus Roux-en-Y gastric bypass—results of a randomized clinical trial," *Surgery for Obesity and Related Diseases*, vol. 13, no. 2, pp. 181–188, Feb. 2017, doi: 10.1016/j.soard.2016.08.025.

[26] X. Terra *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.*, vol. 23, no. 11, pp. 1790–1798, Nov. 2013, doi: 10.1007/s11695-013-1033-9.

[27] M. Salman, K. Noureldin, M. Issa, H. Agwa, M. G. Fadlallah Elhaj, and A. Shalaby, "ThTP2.4 Long-Term Changes in Leptin, Chemerin, and Ghrelin Levels Following Roux-en-Y Gastric Bypass and Laparoscopic Sleeve Gastrectomy," *British Journal of Surgery*, vol. 110, no. Supplement_6, Aug. 2023, doi: 10.1093/bjs/znad241.255.

[28] M. L. Alosco *et al.*, "Improved Serum Leptin and Ghrelin Following Bariatric Surgery Predict Better Postoperative Cognitive Function," *Journal of Clinical Neurology*, vol. 11, no. 1, p. 48, 2015, doi: 10.3988/jcn.2015.11.1.48.

[29] M. Bužga *et al.*, "Dietary intake and ghrelin and leptin changes after sleeve gastrectomy.," *Wideochir. Inne Tech. Maloinwazyjne.*, vol. 9, no. 4, pp. 554–61, Dec. 2014, doi: 10.5114/wiitm.2014.45437.

[30] F. Turkoglu *et al.*, "Effect of bariatric surgery procedures on serum cytokine and Nesfatin-1 levels," *The Surgeon*, vol. 21, no. 5, pp. e287–e291, Oct. 2023, doi: 10.1016/j.surge.2023.03.002.

[31] K. Zorena, O. Jachimowicz-Duda, D. Ślęzak, M. Robakowska, and M. Mrugacz, "Adipokines and obesity. Potential link to metabolic disorders and chronic complications," *Int. J. Mol. Sci.*, vol. 21, no. 10, p. 3570, 2020.