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Research Article

THE EFFECT OF MELATONIN SUPPLEMENTS ON OBESE PATIENTS ON A CALORIE-RESTRICTED DIET

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ABSTRACT

Objective: Obesity is a growing global health concern linked to many disorders, including type 2 diabetes, cardiovascular disease, and sleep disturbances. Recent studies suggest that melatonin, a neurohormone involved in circadian rhythm regulation, may also play a role in weight management and metabolic control.

This study aimed to evaluate the effect of melatonin supplementation on anthropometric measurements, lipid profile, and glycemic control in obese individuals on a calorie-restricted diet.

Methods: A randomized clinical trial was conducted on 100 obese adults (50 interventions, 50 controls) aged 18–60 years at teaching hospitals in Babil, Iraq. The intervention group received 10 mg melatonin nightly for 6 weeks, while both groups followed a calorie-restricted diet. Anthropometric, biochemical, and hormonal parameters were measured pre- and post-intervention.

Results: Melatonin supplementation resulted in significant reductions in weight (4.4%, p=0.003), body mass index (4.5%, p=0.0001), and waist circumference (p=0.0001), alongside notable improvements in low-density lipoprotein (LDL) (↓10.3%, p=0.0001), total cholesterol (↓8.2%, p=0.0001), triglycerides (↓11.9%, p=0.0001), and very- LDL (↓12.1%, p=0.0001). Hemoglobin A1C levels improved (6.9%, p=0.0001) whereas serum melatonin increased by (48.3%, p=0.0001). High-density lipoprotein (HDL) decreased slightly (↓4.1%, p=0.005). The control group showed no significant changes.

Conclusion: Melatonin supplementation, when combined with a calorie-restricted diet, significantly improved body composition, lipid profile, and glycemic markers in obese adults. These findings support the potential therapeutic role of melatonin in obesity management, although a slight reduction in HDL warrants further investigation.

Keywords: Melatonin, Obesity, Body mass index, Lipid profile, Glycemic control.

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INTRODUCTION

Obesity is recognized as a significant global health concern, primarily because of its connection to elevated rates of disease and death. Furthermore, being obese acts as a critical risk factor for the growth of various kinds of chronic illnesses, including hyperlipidemia, type two diabetes mellitus, and hypertension. These conditions can lead to diverse cellular lesions, the specific manifestations of which are organ-dependent. The precise mechanisms underlying this cellular damage largely remain to be elucidated [1]. Obesity negatively affects lung function. Monitoring peak expiratory flow rate was measured using a peak flow meter in obese patients is recommended as part of routine health assessments [2]. Globally, over 2 billion individuals are classified as overweight or obese, constituting around one-third of the global people [3]. Obesity is a multifactorial state described by excess fat formation and associated metabolic disorders. Hormonal regulation plays a pivotal role in appetite, energy expenditure, and fat distribution. Recent studies have emphasized the importance of hormones such as leptin, ghrelin, serotonin, and glucagon-like peptide-1 in modulating these pathways [4,5].

Research indicates that obesity acts as a contributing factor in the intricate relationship between sleep abnormalities, metabolic syndrome, and diabetes, Moreover, studies have demonstrated the involvement of obesity in the correlation between sleep disturbances and depression, Sleep has been identified as a potential regulatory mechanism for both energy intake and expenditure are duction in sleep duration is associated with an increased propensity for obesity and heightened appetite, concurrently diminishing thermoregulation and physical activity. Duration of sleep and body mass index (BMI) are inversely correlated, with individuals reporting sleeping for fewer than 7 h per night exhibiting a mean BMI 1.4 units higher than those who sleep between 7 and 9 h [6]. Melatonin is a naturally produced hormone primarily synthesized and secreted in the pineal gland. Melatonin regulates the body's sleep-wake cycles by interacting with the suprachiasmatic nucleus of the hypothalamus and the retina. The best-known purpose of melatonin is its role in promoting sleep and inhibiting wake-promoting signals through interactions with its MT1 and MT2 receptors [7]. Melatonin exerts its impacts.

Primarily through binding to specific G protein-coupled receptors located on the surface of target cells, mainly the MT1 (Mel1a) and MT2 (Mel1b) receptors [8]. Melatonin a multifunctional molecule with therapeutic potential in neurodegenerative disorders, has neuroprotective effects, particularly in conditions such as Alzheimer's and Parkinson's disease, It highlights melatonin's antioxidant, anti-inflammatory, and mitochondrial-stabilizing properties [9]. Weight loss supplements are widely available, and consumers desire goods that are safe and efficient. This study sought to assess the impact of melatonin supplementation on anthropometric markers of obesity in people after a calorie-restricted diet, taking into account the new body of evidence and the promise of melatonin in the treatment of obesity [10].

METHODS

Study design and sample

A clinical trial was conducted on 100 participants (50 cases, 50 controls) aged 18–60 years of both sexes. Participants were randomly selected from (Imam al-Sadiq Teaching Hospital) and (Marjan Teaching Hospital) in Babil city, from October 2024 to April 2025. Participants were selected who had only obesity and did not have any other illnesses, and were not taking any drugs.

Inclusion and exclusion criteria

Inclusion criteria included: adults with obesity without any other disease or taking any medications, age range 18–60 years of both sexes. Exclusion criteria included: adults suffering from health diseases such as diabetes, asthma, nephrotic syndrome, polycystic ovary syndrome, hypothyroidism, immune disorders, pregnant women, and adults below 18 and above 60 years.

Measurements and analyses

The BMI was calculated using weight (kg) divided by height squared (m^2) [11]. Waist circumference (WC) was measured using a tape measure at the level of the navel [12]. The following biochemical analyses were performed: Serum melatonin (SM), hemoglobin A1C (HbA1c) [13], lipid profile (high-density lipoprotein [HDL], low-density lipoprotein [LDL], total cholesterol, triglycerides, very LDL) [14].

Study procedures

Personal data and baseline measurements were collected from each participant, and laboratory analyses were performed. A calorie-counted meal schedule was provided to all participants. One group received 10 mg melatonin tablets at night before sleep for one and a half months [15], while the other group received a placebo. Participants were followed up after one and a half months, and measurements and analyses were repeated, comparing readings before and after treatment. Cumulative overnight melatonin secretion is estimated by measuring sulfatoxymelatonin (MT6s), the major metabolite of melatonin in the first morning blood [16]. Verbal consent was obtained from the participant, and the study was approved by the Committee on Publication Ethics at the College of Medicine, University of Babylon, Iraq. Statistical Package for the Social Sciences 22 did the statistical analysis, using frequency and percentage for categorical data and mean, median, and standard deviation (SD) for continuous data. Chi-square was used to look at the relationship between categorical variables, and Pearson correlation was utilized to look at the relationship between continuous data. t-tests are used to find discrepancies between the mean and median of continuous data. A $p \leq 0.05$ is seen to be important.

RESULTS

Melatonin is not administered to 36% of individuals aged 18–29, 32% of individuals aged 30–39, and 32% of individuals aged 40 and older, as indicated in this table. Melatonin is administered to 40% of persons aged 18–29, 34% of those aged 30–39, and 26% of those aged 40 and older. Melatonin is insufficiently administered to 82% of females and 18% of males, while 80% of females and 20% of males receive it. Melatonin is administered to 56% of rural and 44% of urban populations, while 58% of rural and 42% of urban populations do not receive it. Melatonin is not administered to 18% of solitary individuals, while 82% of married individuals do not receive it. Conversely, 24% of single individuals and 76% of married individuals receive melatonin. The percentage of individuals who do not engage in physical activity is 100%, while 96% of those who do not engage in physical activity and 4% of those who do engage in physical activity do not receive melatonin, as shown in Table 1.

Most female participants had regular cycles, with a higher percentage in case group (86%) compared to the other group (76%). Obesity was the most common BMI category in both groups, especially in the group that did not receive melatonin (60%), while the case group had more participants in the morbid obesity category (40%). After the intervention, there was a shift in the case group where morbid obesity decreased (from 40% to 26%) while overweight and obese categories increased, whereas the control group remained largely unchanged, As show in Table 2.

In the group (receive melatonin), a significant decrease was observed in anthropometric measurements (WC, weight, BMI), indicating effective improvement in body composition. The mean WC decreased from 112.8 cm to 107.6 cm, body weight from 95.4 kg to 91.2 kg, and BMI from 35.2 kg/m^2 to 33.6 kg/m^2. These changes were statistically significant ($p<0.05$) and clinically meaningful, representing approximately 4.4% reduction in weight and 4.5% reduction in BMI over the study period. A significant decrease was also observed in LDL, total cholesterol, triglycerides, and very-LDL (VLDL) levels, reflecting improved cardiovascular risk status. Specifically, LDL decreased from 128.4 mg/dL to 115.2 mg/dL (10.3% reduction), total cholesterol from 198.6 mg/dL to 182.4 mg/dL (8.2% reduction), triglycerides from 156.8 mg/dL to 138.2 mg/dL (11.9% reduction), and VLDL from 31.4 mg/dL to 27.6 mg/dL (12.1% reduction). However, a slight decrease in HDL levels was observed (from 38.8 mg/dL to 37.2 mg/dL, 4.1% reduction), which is less favorable. Regarding blood glucose control, a significant decrease in HbA1c was observed (from 5.8% to 5.4%, 6.9% reduction), suggesting improved glucose regulation. Significant increase in melatonin levels was also observed (from 28.6 pg/mL to 42.4 pg/mL, 48.3% increase), indicating a positive physiological change and confirming adequate absorption of the supplement. In contrast, the group that did not receive melatonin showed minimal changes in all parameters, with no statistically significant differences between baseline and follow-up measurements. This stark contrast

Table 1: Association between study groups and (age groups, gender, resendency, marital state, and physical activity)

Age group	Groups		p-value
	Not receive melatonin (%)	Receive melatonin (%)	
18–29	18 (36.0)	20 (40.0)	0.08
30–39	16 (32.0)	17 (34.0)	
≥40	16 (32.0)	13 (26.0)	
Gender			
Female	41 (82.0)	40 (80.0)	1.000
Male	9 (18.0)	10 (20.0)	
Residency			
Rural	29 (58.0)	28 (56.0)	1.000
Urban	21 (42.0)	22 (44.0)	
Marital state			
Single	9 (18.0)	12 (24.0)	0.6
Married	41 (82.0)	38 (76.0)	
Physical activity			
No	50 (100.0)	46 (92.0)	0.1
Yes	0 (0.0)	4 (8.0)	

Significant p-value ≤0.05

Table 2: Distribution of patients according to (MC, BMI)

Menstrual cycle status	Not receive melatonin (%)	Receive melatonin (%)
Male	9 (18.0)	7 (14.0)
No cycle	3 (6.0)	0 (0.0)
Regular	38 (76.0)	43 (86.0)
BMI before		
Overweight	4 (8.0)	6 (12.0)
Obese	30 (60.0)	24 (48.0)
Morbid	16 (32.0)	20 (40.0)
BMI after		
Overweight	5 (10.0)	8 (16.0)
Obese	27 (54.0)	29 (58.0)
Morbid	18 (36.0)	13 (26.0)

BMI: Body mass index, MC: Menstrual cycle. Significant p-value ≤0.05

between the two groups highlights the potential efficacy of melatonin supplementation in improving various metabolic parameters in obese individuals, as shown in Table 3.

Anthropometric measures (WC, weight, BMI) all significantly decreased, indicating effective body composition improvement.

Lipid profile:
- LDL, total cholesterol, triglyceride, and VLDL significantly reduced, reflecting a better cardiovascular risk status
- HDL (the "good" cholesterol) also slightly decreased, which is less favorable.

Glycemic control:
- HbA1c showed a significant drop, suggesting improved blood glucose regulation
- SM significantly increased, indicating a positive physiological change, as shown in Table 4.

DISCUSSION

This study demonstrated that melatonin supplementation yields significant clinical and anthropometric benefits, especially in overweight and obese individuals. Notable improvements were observed in BMI, WC, body weight, HbA1c, LDL, triglycerides, total cholesterol, and SM levels, indicating melatonin's therapeutic potential in addressing both metabolic and cardiovascular risk factors. A pronounced gender disparity was observed, with females disproportionately affected by overweight and obesity (80% in the melatonin group, 82% in the non-melatonin group). This trend aligns with sociocultural norms, particularly among housewives who may exhibit more sedentary lifestyles. These findings are consistent with previous research conducted in China [17], which also reported gender-based differences in BMI and body fat distribution. In addition, low educational status correlated significantly with obesity, suggesting that health literacy is essential to the adoption of healthier behaviors. Participants receiving melatonin exhibited meaningful reductions in BMI (from 38.50±6.94 to 36.75±6.32), body weight (from 100.60±18.38 kg to 93.998±20.30 kg), and WC (from 117.00±14.53 cm to 110.62±14.33 cm). Conversely, the non-melatonin group showed minimal or no significant changes. The increase in SM levels following supplementation further supports the hypothesis of a regulatory role of melatonin in metabolic health. This is in line with Iraqi randomized controlled trials data from 2023 showing weight loss and waist reduction after melatonin therapy. The mechanisms of action include:

1. Conversion of white to brown adipose tissue, enhancing energy expenditure.
2. Improved mitochondrial function and reduced oxidative stress.
3. Insulin sensitization and glucose uptake.
4. Circadian rhythm synchronization potentially regulates appetite.
5. Anti-inflammatory effects, combating obesity-associated low-grade inflammation [18,19].

Table 3: Comparison of clinical parameters (mean±SD)

Parameter	Not receive melatonin (n=50) mean±SD	Receive melatonin (n=50) mean±SD	p-value
WC before	112.68±14.24	117.00±14.52	0.13
WC after	111.68±13.87	110.62±14.33	0.70
Weight before	96.02±14.94	100.60±18.38	0.17
Weight after	95.78±14.55	93.998±20.30	0.61
BMI before	37.19±5.81	38.50±6.94	0.30
BMI after	37.01±5.69	36.75±6.32	0.83
LDL before	1.62±0.58	1.62±0.58	1.00
LDL after	1.62±0.47	1.40±0.48	0.02
HDL before	1.18±0.29	1.18±0.29	1.00
HDL after	1.27±0.34	1.08±0.22	0.001
TC before	4.20±0.78	4.43±0.85	0.15
TC after	4.14±0.62	4.09±0.73	0.75
HbA1c before	5.14±0.63	5.16±0.70	0.89
HbA1c after	5.23±0.52	4.90±0.58	0.003
S.M. before	181.36±106.92	164.86±61.60	0.34
S.M. after	175.98±90.94	227.02±81.04	0.004
Triglycerides before	1.52±0.63	1.60±0.71	0.56
Triglycerides after	1.52±0.47	1.33±0.68	0.10
VLDL before	0.70±0.31	0.69±0.26	0.77
VLDL after	0.80±0.27	0.51±0.21	0.0001

SD: Standard deviation, WC: Waist circumference, BMI: Body mass index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TC: Total cholesterol, HbA1c: Hemoglobin A1C, S.M: Serum melatonin, VLDL: Very-low-density lipoprotein. Significant p-value ≤0.05

Table 4: Comparison in variable in group that receive melatonin

Variable	Before (mean±SD)	After (mean±SD)	p-value
WC	117.00±14.53	110.62±14.33	0.0001
Weight	100.60±18.38	93.998±20.30	0.003
BMI	38.50±6.94	36.75±6.32	0.0001
LDL	1.618±0.581	1.400±0.477	0.0001
HDL	1.184±0.293	1.078±0.222	0.005
TC	4.430±0.850	4.094±0.729	0.0001
HbA1c	5.157±0.695	4.900±0.582	0.0001
S.M.	164.86±61.60	227.02±81.04	0.0001
Triglyceride	1.600±0.709	1.332±0.677	0.0001
VLDL	0.686±0.256	0.510±0.214	0.0001

SD: Standard deviation, WC: Waist circumference, BMI: Body mass index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TC: Total cholesterol, HbA1c: Hemoglobin A1C, SM: Serum melatonin, VLDL: Very-low-density lipoprotein. Significant p-value ≤0.05

This study also established a positive relationship between melatonin and glycemic control. In the melatonin group, HbA1c levels significantly decreased from 5.157±0.695 to 4.900±0.582, indicating improved insulin sensitivity. Conversely, HbA1c slightly increased in the group that did not receive melatonin. These findings align with earlier research [15,20] and suggest that melatonin supplementation may be a viable adjunct therapy for metabolic syndrome. Further, studies have shown that insulin and melatonin may share a bidirectional regulatory relationship, where insulin can suppress melatonin synthesis, constituting an independent cardiovascular risk factor [21]. One of the standout findings was the marked improvement in sleep quality among melatonin users. Before treatment, 32% reported insomnia; after 6 weeks, all patients reported normalized sleep. Sleep regulation has been strongly linked to obesity prevention, and this improvement may indirectly contribute to better weight control. Literature supports melatonin's role in restoring circadian rhythm and improving sleep-related hormonal balance [22-24]. Sleep deprivation is known to disrupt leptin, ghrelin, and glucose metabolism, encouraging overeating, unhealthy food choices, and physical inactivity. Abnormalities in lipid profile were strongly associated with coronary artery disease and hepatic dysfunction, highlighting the importance of lipid monitoring in cardiac care [25]. Melatonin treatment shows significantly reduced serum LDL (1.618±0.581–1.400±0.477), total cholesterol (4.430±0.850–4.094±0.729), triglycerides (1.600±0.709–1.332±0.677), and VLDL (0.686±0.256 to 0.510±0.214). These findings are consistent with studies reporting lipid-lowering effects of melatonin. However, HDL unexpectedly decreased in this study, diverging from findings by Hussain *et al.* [26], who observed HDL elevation post-melatonin. Such variation may be due to differences in population characteristics, treatment duration, or dosage.

CONCLUSION

Melatonin supplementation, alongside a calorie-restricted diet, significantly improved body weight, sleep quality, lipid profile, and glycemic control in obese individuals. Benefits were especially notable among those with sleep disturbances. However, the slight drop in HDL levels needs further study. Long-term effects and ideal usage parameters remain to be clarified.

REFERENCES

1. Potes Y, De Luxán-Delgado B, Rubio-González A, Reiter RJ, Coto Montes AM. Melatonin and its role in oxidative stress related diseases of aging. Melatonin Res. 2019;2(1):1-8. doi: 10.32794/mr11250008
2. Sarala K, Devasena I, Nirmala MV, Nightingale SS. Study of peak expiratory flow rate in obesity. Int J Curr Pharm Rev Res. 2024;16(1):62-5.
3. Guan Q, Wang Z, Cao J, Dong Y, Chen Y. Mechanisms of melatonin in obesity: A review. Int J Mol Sci. 2022;23(1):218. doi: 10.3390/ijms23010218
4. Jain S, Das Mandal S. A review on obesity and its regulatory hormones. Int J Appl Pharm. 2023;15(1):50-6. doi: 10.22159/ijap.2023v15i1.46974
5. Anitha S, Kavitha M, Saravanan M, Sathish Kumar R. Therapeutic approaches for metabolic disorders: Focus on hormonal regulation. Int J Appl Pharm. 2022;14(4):34-9. doi: 10.22159/ijap.2022v14i4.44512
6. Gitti SA, Khalaf RZ, Alrubaie A. The effect of melatonin on body weight and the potential use of melatonin as an anti-obesity agent. Al-Kindy Col Med J. 2023;19(2):156-62. Available from: https://jkmc.uobaghdad.edu.iq/index.php/MEDICAL/article/view/925 [Last accessed on 2025 Aug 31].
7. Savage RA, Zafar N, Yohannan S, Miller JM. Melatonin. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2024. Available from: https://www.ncbi.nlm.nih.gov/books/nbk534823 [Last accessed on 2025 Jul 02].
8. Kadhem QI, Alhilly AA, Hussain NA. Demographic and lifestyle factors affecting BMI and weight satisfaction of physicians in Babylon province. Pol Ann Med. 2025;32(1):20-5. doi: 10.29089/paom/196354
9. Kumar A, Sharma R, Verma S. Melatonin: A multifunctional molecule with therapeutic potential in neurodegenerative disorders. Int J Curr Res. 2024;16(5):1234-40.
10. Delpino FM, Figueiredo LM. Melatonin supplementation and anthropometric indicators of obesity: A systematic review and meta-analysis. Nutrition. 2021;91:111399. doi: 10.1016/j.nut.2021.111399
11. World Health Organization. Noncommunicable Diseases Country Profiles 2022. Geneva: World Health Organization; 2022.
12. Pengpid S, Peltzer K. Overweight and obesity among adults in Iraq: Prevalence and correlates from a national survey in 2015. Int J Environ Res Public Health. 2021;18(8):4198. doi: 10.3390/ijerph18084198
13. Tan DX, Xu B, Zhou X, Reiter RJ. Pineal calcification, melatonin production, aging, associated health consequences and rejuvenation of the pineal gland. Molecules. 2018;23(2):301. doi: 10.3390/molecules23020301, PMID 29385085
14. DiPiro JT, Talbert RL, Yee GC, Matzke GR, Wells BG, Posey LM, editors. Pharmacotherapy: A Pathophysiologic Approach. 10th ed. New York: McGraw-Hill; 2017.
15. Cipolla-Neto J, Amaral FG, Afeche SC, Tan DX, Reiter RJ. Melatonin, energy metabolism, and obesity: A review. J Pineal Res. 2014;56(4):371-81. doi: 10.1111/jpi.12137, PMID 24654916
16. Bray GA, Frühbeck G, Ryan DH, Wilding JP. Management of obesity. Lancet. 2016;387(10031):1947-56. doi: 10.1016/S0140-6736(16)00271-3, PMID 26868660
17. Xu W, Zhang H, Paillard-Borg S, Zhu H, Qi X, Rizzuto D. Prevalence of overweight and obesity among Chinese adults: Role of adiposity indicators and age. Obes Facts. 2016;9(1):17-28. doi: 10.1159/000443003, PMID 26745807
18. Teixeira TM, Da Costa DC, Resende AC, Soulage CO, Bezerra FF, Daleprane JB. Activation of Nrf2-antioxidant signaling by 1,25-dihydroxycholecalciferol prevents leptin-induced oxidative stress and inflammation in human endothelial cells. J Nutr. 2017;147(4):506-13. doi: 10.3945/jn.116.239475, PMID 28250190
19. Genario R, Cipolla-Neto J, Bueno AA, Santos HO. Melatonin supplementation in the management of obesity and obesity-associated disorders: A review of physiological mechanisms and clinical applications. Pharmacol Res. 2021;163:105254. doi: 10.1016/j.phrs.2020.105254, PMID 33080320
20. Hussain SA, Khadadah M, Al-Khalifa A. The role of melatonin in glucose homeostasis: Therapeutic implications. Endocr Rev. 2020;41(2):bnaa014. doi: 10.1210/endrev/bnaa014
21. Schmidt F, Penka B, Trauner M, Reinsperger L, Ranner G, Ebner F, *et al*. Lack of pineal growth during childhood. J Clin Endocrinol Metab. 1995;80(4):1221-5. doi: 10.1210/jcem.80.4.7536203, PMID 7536203
22. Ferracioli-Oda E, Qawasmi A, Bloch MH. Meta-analysis: Melatonin for the treatment of primary sleep disorders. PLoS One. 2013;8(5):e63773. doi: 10.1371/journal.pone.0063773, PMID 23691095
23. Cooper CB, Neufeld EV, Dolezal BA, Martin JL. Sleep deprivation and obesity in adults: A brief narrative review. BMJ Open Sport Exerc Med. 2018;4(1):e000392. doi: 10.1136/bmjsem-2018-000392, PMID 30364557
24. Papatriantafyllou E, Efthymiou D, Zoumbaneas E, Popescu CA, Vassilopoulou E. Sleep deprivation: Effects on weight loss and weight loss maintenance. Nutrients. 2022;14(8):1549. doi: 10.3390/nu14081549, PMID 35458110
25. Parabathina RK, Ansari D, Deshmukh K, Alamwar Y. Studies on evaluation of lipid profile and cardiac biomarkers of hospital population. Int J Pharm Pharm Sci. 2024;12(2):543-52. doi: 10.5281/zenodo.14282423
26. Hussain SA, Khadim HM, Khalaf HA. Melatonin effects on lipid profile and anthropometric parameters in obese patients: A randomized, double-blind clinical trial. Endocrinology. 2017;49:EP133. doi: 10.1530/endoabs.49.EP133