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The Effect of Overweight and Obesity on Contralateral Suppression in Patients with Multiple Sclerosis*

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ABSTRACT

Background: This study aimed to evaluate the effects of overweight and obesity on the auditory efferent system in patients with multiple sclerosis (MS).

Methods: A total of 49 patients with MS, aged between 18 and 50 years, were included in the study. Participants were divided into 2 groups according to their body mass index (BMI). Distortion product otoacoustic emissions (DPOAE) and contralateral suppression (CS) measurements were obtained to evaluate efferent auditory function. Statistical analyses were performed using IBM SPSS Statistics for Windows, version 21.0 (IBM SPSS Corp.; Armonk, NY, USA).

Results: There was no statistically significant difference in signal-to-noise ratio (SNR) values of DPOAE or in CS values between overweight/obese and non-obese MS patients.

Conclusion: Long-term studies with larger participant groups are required to better understand the effects of MS and obesity on the auditory system. Future studies should include patient groups with a BMI of 30 or higher.

Keywords: Contralateral suppression, DPOAE, efferent system, multiple sclerosis , obesity

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Introduction

Multiple sclerosis (MS) is a chronic autoimmune disease that affects the central nervous system. Although the disease typically progresses with relapses and remissions, progression can also be observed in its course. Nutrition is thought to play a role in the pathogenesis and clinical progression of the disease. Evaluating patients in terms of cachexia and obesity, developing nutritional treatments targeting symptoms, and lifelong monitoring are crucial for understanding the natural course of the disease.[1]

Obesity is defined as abnormal fat accumulation that can hinder optimal health.[2] Increased immune cells in adipose tissue contribute to insulin resistance in obese individuals.[3] Compared to subcutaneous fat, visceral adipose tissue is a smaller lipid storage compartment but is closely associated with many obesity-related metabolic disorders.[4,5] Obesity accelerates processes such as hypertension, insulin resistance, and atherosclerosis through immune system dysregulation and chronic inflammation.[2]

Metabolic disorders, including dyslipidemia, atherosclerosis, hypertension, and diabetes, can damage hair cells, leading to hearing loss, tinnitus, and dizziness.[6] The increasing prevalence of obesity may potentially contribute to a higher incidence of MS in both children and adults, as well as an adverse clinical course in diagnosed patients. The relationship between MS, obesity, and other environmental factors continues to be investigated.[1,7]

Different types of malnutrition, such as cachexia and obesity, are frequently observed in MS patients.[8] The prevalence of overweight and obesity among MS patients has been reported as 64.5%,[9] while this rate was found to be 40.5% among Turkish MS patients.[10]

Obesity has a significant impact on vascular function, which can lead to negative consequences in organs with high vascular structures, such as the auditory system. Additionally, obesity has been associated with the onset of sensorineural hearing loss.[11]

Recent studies have emphasized the role of the medial olivocochlear (MOC) system in modulating cochlear gain and enhancing speech-in-noise perception. Disruption in this system has been linked to increased listening effort and reduced speech intelligibility, particularly in populations with neurological or metabolic disorders.[12,13]

The aim of this study is to evaluate the integrity of efferent auditory pathways in overweight/obese and non-obese MS patients using the contralateral suppression (CS) test and to investigate the effects of obesity on the auditory system in MS patients.

Material and Methods

Ethical Approval

This study was approved by the Ondokuz Mayıs University Clinical Research Ethics Committee (Approval No.: 2022/300, Date: May 31, 2022) The ClinicalTrials.gov identifier (NCT number) was NCT07136311. This research protocol has been registered in the PROSPERO database (CRD420251116719).

Participants

A total of 49 voluntary patients aged between 18 and 50 years, who were diagnosed with definite MS according to the revised McDonald criteria, were included in the study. All participants were recruited from the Neurology Clinic of Ondokuz Mayıs University. Each individual was informed about the study protocol, and written informed consent was obtained from all participants prior to testing.

Exclusion criteria included patients who had experienced an MS exacerbation and/or received steroid treatment within the last 3 months, as well as those with a history of smoking, alcohol or sedative drug use, unstable vital signs, pregnancy, or serious systemic disorders such as heart or kidney failure, major depression, severe anemia, immunodeficiency, or narcolepsy.

Inclusion criteria for the control group were as follows: absence of MS diagnosis, age between 18 and 50 years, ability to cooperate with the scales and audiological tests administered, normal otolaryngological (outer and middle ear) examination findings, a type A tympanogram with a peak compliance between ±50 daPa on acoustic immittance testing, and a pure tone average within normal limits (250-8000 Hz). Participants were also required to have normal speech discrimination scores and no history of other neurological, psychiatric, or metabolic disorders. Additionally, control subjects had no history of noise exposure (e.g., occupational noise or blast trauma) and no record of ototoxic drug use.

All MS patients included in the study had the relapsing–remitting subtype and Expanded Disability Status Scale scores between 0 and 2, indicating minimal or no functional disability. According to their body mass index (BMI) values, patients were divided into 2 groups: non-obese (BMI < 25 kg/m^2) and overweight/obese (BMI ≥ 25 kg/m^2).[14]

Assessment Tools

Body Mass Index: Body mass index was calculated by dividing body weight (kg) by the square of height (m^2).[14]

Distortion Product Otoacoustic Emissions Contralateral Suppression Test: Distortion product otoacoustic emissions (DPOAE) and CS measurements were performed in a soundproof room using the Madsen Capella2 device (GN Otometrics, ICS Medical, Chicago, USA). The contralateral noise stimulus was delivered through insert earphones using the Piano Clinical Audiometer (Inventis, Padua, Italy) at 65 dB SL white noise. Before testing, probe calibration was performed using a 1 cc acoustic calibration cavity. Care was taken to ensure that the probe tip used for all measurements was appropriately sized to completely seal each participant's ear canal. All otoacoustic emission (OAE) measurements were conducted bilaterally.

After achieving a proper probe fit, DPOAEs were elicited using pure-tone primary stimuli at frequencies f_1 and f_2 with an f_2/f_1 ratio of 1.22. The intensities of f_2 and f_2 were set to 65 dB SPL and 55 dB SPL, respectively. The presence of DPOAEs was confirmed when the signal-to-noise ratio (SNR) exceeded 6 dB SPL in at least 2 frequency bands.

DPOAE amplitudes and SNR values were analyzed at frequencies of 1000, 2000, 3000, 4000, 5000, 6000 Hz, and 8000 Hz under 2 conditions: in the absence and in the presence of 65 dB SPL contralateral white noise. Amplitude values were used for analysis, and a reduction of ≥1 dB in amplitude in the presence of contralateral noise was considered indicative of CS.[15]

Tanita BC 418 Measurement: Body composition analysis was conducted using the Tanita BC 418 device, which operates based on the bioelectrical impedance analysis method. This device evaluates body fat percentage, muscle mass, and other parameters, displaying the results on a digital screen.

Procedure

Each participant first underwent DPOAE measurement without contralateral noise. Subsequently, DPOAE suppression measurements were conducted by delivering 65 dB SL white noise to the contralateral ear. The groups were then compared based on their CS values.

Statistical Analysis

Data were analyzed using SPSS version 21 (IBM SPSS Corp.; Armonk, NY, USA). Normality of the data was assessed using the Shapiro–Wilk test. Data were tested for conformity to a normal distribution.

MAIN POINTS

- *This study is one of the few to investigate the impact of obesity on the efferent auditory pathways in patients with multiple sclerosis (MS) using the contralateral suppression (CS) test. While MS-related auditory dysfunction has been studied, the specific contribution of obesity remains underexplored.*
- *No statistically significant differences were found in distortion product otoacoustic emissions or CS values between overweight or obese and non-obese MS patients. However, both groups demonstrated greater suppression at lower frequencies and reduced suppression at higher frequencies, suggesting frequency-dependent auditory effects.*
- *Further large-scale and long-term studies are required to assess the combined effects of obesity and MS on the auditory system. Including patients with body mass index ≥30 and evaluating inflammatory biomarkers could provide deeper insight into underlying mechanisms.*

For normally distributed data, mean ± standard deviation ($\bar{x}$ ± SD) was used, while for non-normally distributed data, median (min-max) was reported. Student's *t*-test was applied for comparisons between groups when data followed a normal distribution, whereas the Mann-Whitney *U*-test was used for non-normally distributed data. Chi-square analysis was performed to evaluate relationships between categorical variables. Results were interpreted with a 95% CI, and statistical significance was set at $P < .05$. The authors declare that no artificial intelligence (AI) tools were used in the design of the study, data collection, data analysis, or interpretation of the results.

Sample Size Calculation

The sample size was calculated using G*Power (version 3.1.7). Based on a previous study that reported a large effect size (Cohen's $d = 0.8$),[16] an a priori power analysis was conducted. The analysis revealed that a total of 52 participants (26 per group) would be required to achieve a power of 0.80 with a significance level of 0.05 (2-tailed).

In the current study, 49 participants (23 in the overweight/obese group and 26 in the non-obese group) were included. A post-hoc analysis indicated that this sample size still yields a power of approximately 0.77, which is considered acceptable for detecting large effect sizes.

Results

The participants were divided into 2 groups based on their BMI: BMI > 25 and BMI < 25. A total of 23 patients were included in the BMI > 25 group, while 26 patients were included in the BMI < 25 group. The distributions of age and BMI showed significant differences between the groups (independent Samples *t*-test, $P < .001$). In contrast, the gender distribution did not differ significantly between the groups (Pearson's chi-square test, $P > .005$) (Table 1).

The study sample consisted of 23 patients with BMI > 25 (male/female: 4/19; mean age: 37.00) and 26 patients with BMI < 25 (male/female: 7/19; mean age: 29.42). No significant difference was found between the groups in terms of age (independent samples *t*-test, $P = .000$) or gender distribution (Pearson chi-square test, $P = .376$) (Table 1).

Table 1. The Distribution of Participants' Body Mass Index, Age, and Gender Characteristics Across Groups

	BMI >25 Group (n = 23)	BMI <25 (n = 26)	*P*
Age (years) ($\bar{x}$ ± s)	37.00 ± 8.61	29.42 ± 7.05	.000
Sex (male/female)	4/19	7/19	.376
BMI (kg/m^2) ($\bar{x}$ ± s)	31.47 ± 3.75	21.17 ± 2.48	.000

Independent Samples *t*-test, chi-square test (Continuity correction); $P < .05$ was considered to be statistically significant.

Table 2 presents the comparison of DPOAE SNR values between the MS and control groups.

When the SNR values of the right and left ears were compared between the groups, no statistically significant difference was observed at any frequency ($P > .05$, Table 2).

When the CS values of the right and left ears were compared between the groups, no statistically significant difference was observed at any frequency ($P > .05$, Table 3).

In the BMI > 25 group, the highest suppression rate (56.5%) was observed at 1400 Hertz (Hz) and 2800 Hz, while the lowest suppression rate (17.3%) was found at 8000 Hz (Table 4).

In the BMI < 25 group, the highest suppression rate (59.6%) was observed at 2000 Hz, whereas the lowest suppression rate (17.3%) was also found at 8000 Hz. In both groups, greater suppression effects were observed at lower frequencies, while fewer suppression effects were observed at higher frequencies (Table 4).

Discussion

Obesity can significantly impact microvascular and macrovascular circulation, potentially reducing blood flow within the cochlea. This may be a contributing factor in the development of hearing loss. Meta-analyses have confirmed that increases in BMI and waist circumference are associated with an elevated risk of hearing loss.[17]

It has been reported that for every 5 kg/m^2 increase in BMI, the risk of hearing loss increases by 14%, providing strong evidence for the relationship between obesity and hearing impairment. Additionally,

Table 2. Comparison of DPOAE SNR Values by Groups

	F(Hz)	BMI <25 Group ($\bar{x}$ ± s)	BMI >25 Group ($\bar{x}$ ± s)	BMI<25 95% CI (Lower-Upper)	BMI> 25 95% CI (Lower-Upper)	*P*
Right ear	1000	11.78 ± 5.25	10.42 ± 6.37	9.51-14.06	7.85-13.00	.841
	1400	16.57 ± 6.49	17.46 ± 7.61	13.76-19.37	14.38-20.54	.666
	2000	16.91 ± 7.00	15.77 ± 6.68	13.88-19.94	13.07-18.47	.562
	2800	18.22 ± 6.67	15.04 ± 7.11	15.33-21.10	12.17-17.91	.115
	4000	12.91 ± 7.89	12.77 ± 5.82	9.50-16.33	10.42-15.12	.942
	5600	8.78 ± 7.44	8.88 ± 7.67	5.57-12.00	5.78-11.98	.963
	8000	2.00 ± 10.61	4.46 ± 5.45	-2.59-6.59	2.26-6.66	.41
	1000	13.22 ± 5.62	11.88 ± 6.69	10.79-15.65	9.18-14.59	.458
	1400	16.91 ± 7.26	15.00 ± 7.65	13.77-20.05	11.91-18.09	.376
Left Ear	2000	15.70 ± 6.07	16.50 ± 7.66	13.07-18.32	13.40-19.60	.689
	2800	16.30 ± 6.78	16.38 ± 7.60	13.37-19.24	13.31-19.46	.969
	4000	10.74 ± 8.33	10.23 ± 9.38	7.13-14.34	6.44-14.02	.843
	5600	8.43 ± 9.51	8.58 ± 7.92	4.32-12.55	5.38-11,78	.955
	8000	5.70 ± 6.29	3.58 ± 6.31	2.97-8.42	1.03-6.13	.246

F, frequency; independent Samples *t*-test; t, test statistic.

Table 3. Comparison of CS Values Between MS and Control Groups

	F	BMI < 25 Group ($\bar{x} \pm s$)	BMI > 25 Group ($\bar{x} \pm s$)	BMI < 25 95% CI (Lower-Upper)	BMI > 25 95%CI (Lower-Upper)	*P*
Right Ear	1000	2.00 ± 2.81	1.54 ± 2.19	0.78-3.22	0.65-2.43	.703
	1400	2.17 ± 3.09	1.62 ± 2.08	0.83-3.51	0.78-2.46	.966
	2000	1.61 ± 2.46	2.00 ± 1.64	0.54-2.67	1.33-2.67	.147
	2800	1.52 ± 2.35	0.92 ± 1.49	0.50-2.54	0.32-1.53	.234
	4000	0.91 ± 1.56	1.65 ± 3.16	0.24-1.59	0.38-2.93	.819
	5600	1.04 ± 1.94	1.12 ± 2.25	0.20-1.88	0.21-2.02	.949
	8000	0.52 ± 2.10	0.69 ± 1.59	−0.39-1.43	0.05-1.34	.207
Left Ear	1000	2.22 ± 2.41	1.85 ± 2.60	1.17-3.26	0.79-2.90	.427
	1400	1.70 ± 2.63	1.04 ± 1.73	0.56-2.84	0.34-1.74	.209
	2000	1.30 ± 2.16	1.04 ± 1.58	0.37-2.24	0.40-1.68	.758
	2800	1.09 ± 1.20	1.38 ± 2.31	0.57-1.61	0.45-2.32	.676
	4000	0.48 ± 1.88	0.81 ± 1.60	−0.33-1.29	0.16-1.45	.221
	5600	0.74 ± 1.42	1.12 ± 3.16	0.12-1.35	−0.16-2.39	.507
	8000	0.65 ± 1.26	0.35 ± 1.09	0.11-1.20	−0.10-0.79	.207

CS, contralateral suppression; F, frequency; Mann-Whitney *U*-test.
P < .05 was considered to be statistically significant.

obesity may negatively affect the function of the cochlea and auditory pathways, potentially leading to permanent damage in the sensory system.

Adipose tissue has been reported to influence the immune system by secreting pro-inflammatory cytokines, such as tumor necrosis factor-α (TNF-α) and interleukin-6. Studies emphasize that inflammation, particularly by damaging vascular structures in the cochlea, induces oxidative stress in cochlear cells and reduces auditory sensitivity.[18] This understanding helps clarify the interaction between hearing loss and inflammation. The inflammatory changes induced by obesity may weaken the cochlear microvascular structures, disrupt cochlear functions, and potentially lead to hearing loss.

In a study conducted by Saka et al,[10] dietary records and anthropometric measurements were collected from 15 male and 22 female patients. The study classified 5.4% of patients as underweight, 54.1% as normal weight, 24.3% as overweight, and 16.2% as obese. Obesity is directly linked to increased fat tissue and the resulting elevation in systemic inflammation, which is considered a potential factor affecting the pathogenesis of hearing loss.

In MS patients, both peripheral and central regions of the auditory system may be affected. Changes in OAEs reported in the literature indicate the involvement of the auditory efferent pathways.[19] It has been suggested that, in addition to hearing function impairment in MS patients, obesity may further exacerbate this dysfunction. Studies have observed more pronounced auditory impairments in MS patients, suggesting that the combination of MS and obesity may lead to worse hearing performance.[18]

Obesity may have distinct effects on the auditory efferent system. Compared to non-obese MS patients, overweight/obese MS patients may exhibit more pronounced hearing loss. CS measurements serve as a critical test for evaluating the impact of the cochlea on the auditory efferent system. In obese individuals, suppression in the auditory efferent pathways could indicate negative effects on cochlear mechanisms. However, in this study, when comparing overweight/obese and non-obese groups, no significant differences were observed in OAEs or CS measurements between the groups.

In MS patients, the efferent auditory system, particularly CS mechanisms, has been reported to be affected. This is attributed to myelin loss and the disruption of communication networks within the central nervous system.[20-22]

Konomi et al[23] also investigated the dynamic characteristics of DPOAE CS and reported that while suppression magnitude tended to decrease with age, suppression time constants (reflecting the onset and offset response speed of the MOC system) remained unaffected by age. This finding suggests that although the strength of the efferent response may decline in older adults, the temporal efficiency of the MOC pathway can remain stable. In the present study, which included participants aged 18-50 years, no significant age-related

Table 4. Distribution of the Presence (≥1 dB) and Absence (<1 dB) of the MOC Reflex in the Right and Left Ears in BMI <25 and BMI >25 Groups

	BMI >25 Group		BMI <25 Group		Total	
Frequency (Hz)	Present (%)	Absent (%)	Present (%)	Absent (%)	Present (%)	Absent (%)
1000	25 (54.3)	21 (45.7)	24 (46.1)	28 (53.9)	49 (50.0)	21 (45.7)
1400	26 (56.5)	20 (43.5)	26 (50.0)	26 (50.0)	52 (53.0)	46 (74.0)
2000	24 (52.1)	22 (47.9)	31 (59.6)	21 (40.4)	55 (56.1)	43 (43.9)
2800	26 (56.5)	20 (43.5)	22 (42.3)	30 (57.7)	48 (48.9)	50 (51.1)
4000	12 (26.0)	34 (74.0)	17 (32.6)	35 (67.4)	29 (29.5)	69 (70.5)
5600	13 (28.2)	33 (73.8)	12 (23.0)	40 (77.0)	25 (25.5)	73 (74.5)
8000	8 (17.3)	38 (82.7)	9 (17.3)	43 (82.7)	17 (17.3)	81 (82.7)

differences in suppression outcomes were observed. Therefore, in agreement with previous findings, age was not considered a confounding factor influencing DPOAE CS.

Obesity has been identified as a significant risk factor for MS, with high BMI during childhood and adolescence increasing the risk of developing MS. Studies have shown that individuals with a BMI ≥ 30 kg/m^2 during adolescence have twice the risk of developing MS compared to those with normal weight.

In MS patients, leptin levels are elevated, which is associated with increased production of pro-inflammatory cytokines such as TNF-α and interferon gamma. Leptin may act as a hormone that supports the autoimmune characteristics of MS. The coexistence of obesity and MS may therefore have additive or synergistic effects on the efferent auditory system. It has been suggested that obesity-induced inflammation could exacerbate the neuroinflammatory processes already present in MS patients, potentially leading to further reductions in CS levels.

Although leptin functions as a neurotrophic factor, it also promotes pro-inflammatory responses. The increase in leptin levels due to obesity may contribute to MS pathogenesis, further complicating its effects on the auditory efferent system. In MS patients, leptin expression has been shown to increase during active inflammatory lesions and relapse periods. Leptin can worsen disease progression by enhancing immune responses, while also playing a critical role in neuronal cell survival as a neurotrophic factor.[24] The increase in leptin levels associated with obesity may contribute to the pathogenesis of MS and potentially exacerbate auditory system impairments. Although no biomarkers were measured in the present study, these associations were discussed in the context of existing literature and are considered potential mechanisms to be investigated in future research rather than definitive conclusions.

In this study, no statistically significant difference was found in CS values between overweight/obese and non-obese MS patients. To better understand the effects of obesity and MS coexistence on the auditory system, long-term studies with larger participant groups are needed. Research focusing on individuals with a BMI of 30 and above could provide a clearer understanding of how CS mechanisms are affected.

This study has several limitations. Although the total sample size approached the level suggested by power analysis, the imbalance between overweight/obese and non-obese subgroups may have reduced the overall statistical power. The study also focused solely on CS of OAEs without including additional auditory or neurophysiological assessments that could provide a more comprehensive evaluation of efferent auditory function.

In addition, participants with a BMI of 25 kg/m^2 or higher were classified as a single "overweight/obese" group, which may have introduced heterogeneity. The control group consisted of normal-weight MS patients; therefore, the findings cannot be directly compared with those from healthy individuals. This limits the ability to fully isolate the independent effects of obesity on auditory function.

Future studies with larger and more diverse samples, including both healthy participants and MS patients with varying BMI levels, and incorporating broader auditory or neurophysiological measures across multiple centers, are recommended to obtain more robust and generalizable results. Moreover, biomarker-based research is crucial to better understand the role of adipose-derived cytokines in hearing loss and to elucidate the mechanisms underlying auditory dysfunction in MS patients.

No statistically significant differences were found in the SNR ratios of the right and left ears when comparing overweight/obese and non-obese MS patients. Additionally, the coexistence of obesity with MS did not result in any measurable differences in the auditory efferent system.

From a clinical perspective, the assessment of efferent auditory function in obese MS patients may have important implications. The MOC system contributes to the ability to suppress background noise and maintain speech perception in challenging listening environments. Therefore, monitoring MOC activity through CS measures could help detect subtle auditory dysfunctions that are not evident in standard audiometric tests.

Moreover, both MS and obesity can influence central and peripheral neural processing through inflammatory and metabolic mechanisms, which may increase listening effort and affect cognitive auditory abilities. Future research should integrate speech-in-noise perception and cognitive listening evaluations to better understand these interactions.

In conclusion, while no significant differences were observed in the present study, clinical monitoring of efferent auditory function in obese MS patients may provide early insights into auditory–cognitive interaction and contribute to the development of targeted rehabilitation approaches.

Data Availability Statement: *The data that support the findings of this study are available on request from the corresponding author.*

Ethics Committee Approval: *This study was approved by the Ondokuz Mayıs University Clinical Research Ethics Committee (Approval No.: 2022/300, Date: May 31, 2022).*

Informed Consent: *Written informed consent was obtained from the patients who agreed to take part in the study.*

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