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# Relationships Between Obesity, Dyslipidemia and Manual Wheelchair Mobilization in Individuals with Chronic Spinal Cord Injury

## Kronik Spinal Kord Yaralanmalı Bireylerde Obezite, Dislipidemi ve Manuel Tekerlekli Sandalye Mobilizasyonu Arasındaki İlişki

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**ABSTRACT Objective:** This study investigated the impact of obesity and dyslipidemia on manual wheelchair mobility in individuals with spinal cord injury (SCI). **Material and Methods:** A total of 102 individuals with chronic motor-complete SCI, aged 18-60 years, with an injury duration of ≥1 year, were included. The body mass index (BMI) was used to assess obesity. The lipid profiles were measured, including high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and total cholesterol (TC). Manual wheelchair propulsion tests (MWPTs) (20-meter propulsion test, slalom test, and 6-minute propulsion test) were conducted to evaluate mobility. SCI-specific BMI⩾22 kg/m² was classified as obese, while <22 kg/m² was classified as non-obese. HDL-C<40 mg/dL was considered low, whereas LDL-C ≥130 mg/dL, TG ≥150 mg/dL, and TC ≥200 mg/dL were considered high. Participants were stratified into binary groups, and their MWPT results were compared. **Results:** Among participants, 71.6% (n=73) were classified as obese, 69.6% had low HDL-C, 27.4% had high LDL-C, 45.1% had high TG, and 22.5% had high TC. Participants with TG<150 mg/dL demonstrated significantly shorter 20-meter propulsion times than those with TG≥150 mg/dL (p=0.014), while HDL-C, LDL-C, and TC levels did not significantly affect test outcomes (p>0.05). Participants with BMI <22 kg/m² exhibited shorter 20-m and slalom test times and better 6-min propulsion performance than those with BMI ≥22 kg/m² (p=0.014, p=0.008, p=0.035, respectively). **Conclusion:** Obesity and hypertriglyceridemia negatively impact manual wheelchair mobility in individuals with chronic SCI.

**Keywords:** Spinal cord injury; obesity; dyslipidemia; mobilization; wheelchair

**ÖZET** Amaç: Çalışmanın amacı spinal kord yaralanmalı (SKY) bireylerde obezite ve dislipideminin manuel tekerlekli sandalye mobilizasyonuna etkisini araştırmaktır. Gereç ve Yöntemler: Yaralanma süresi ≥1 yıl olan, 18-60 yaş arası, 102 kronik motor komplet SKY'li birey dâhil edildi. Obezite değerlendirmesinde beden kitle indeksi (BKİ) kullanıldı. Lipit profili olarak yüksek yoğunluklu lipoprotein [High Density Lipoprotein (HDL)]-kolesterol (HDL-K), düşük yoğunluklu lipoprotein [Low Density Lipoprotein (LDL)]-kolesterol (LDL-K), trigliserid (TG) ve total kolesterol (TK) değerleri ölçüldü. Mobilizasyonu değerlendirmek için manuel tekerlekli sandalye itme testleri (MTSİT) (20 m itme testi, slalom testi, 6 dk itme testi) uygulandı. SKY'ye özgü BKİ⩾22 kg/m² obez olarak sınıflandırılırken, <22 kg/m² obez olmayan olarak sınıflandırıldı. HDL-K<40 mg/dL düşük kabul edilirken, LDL-K≥130 mg/dL, TG≥150 mg/dL ve TK≥200 mg/dL yüksek olarak değerlendirildi. Katılımcılar ikili gruplara ayrıldı ve manuel tekerlekli sandalye itme testi sonuçları karşılaştırıldı. Bulgular: Katılımcıların %71,6'sının (n=73) obez olduğu, %69,6'sında HDL-K düşüklüğü, %27,4'ünde LDL-K yüksekliği, %45,1'inde TG yüksekliği ve %22,5'inde TK yüksekliği bulundu. TG<150 mg/dl olan grupta TG≥150 mg/dl olan gruba göre 20 m itme testi süresi anlamlı olarak daha düşük bulundu (p=0,014) ancak HDL-K, LDL-K ve TK düzeylerinin manuel tekerlekli sandalye itme testlerine etkisi anlamlı bulunmadı (p>0,05). BKİ<22 kg/m2 olan grupta BKİ≥22 kg/m2 olan gruba göre 20 metre itme testi süresi ve slalom testi süresi kısalığı ile 6 dk itme testi performansının yüksekliği anlamlı olarak bulundu (sırasıyla p=0,014, p=0,008, p=0,035). Sonuç: Kronik SKY'li bireylerde obezite ve hipertrigliserideminin manuel tekerlekli sandalye mobilizasyonunu olumsuz etkilediği gösterilmiştir.

**Anahtar Kelimeler:** Spinal kord yaralanması; obezite; dislipidemi; mobilizasyon; tekerlekli sandalye

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Metabolic changes observed after spinal cord injury (SCI) lead to obesity due to decreased sympathetic nervous system activity and a restriction in physical activity.[1-4] Within 5 years following the injury, the prevalence of obesity in individuals with SCI ranges from 40% to 66%.[1,2,5] Obesity limits the mobility of individuals with SCI and accelerates the risk of cardiovascular disease (CVD), which is one of the primary causes of mortality.[4,6]

Physical inactivity and immobilization resulting from SCI lead to progressive changes in body composition over time. Another consequence of SCI-related immobilization is dyslipidemia, which represents an additional risk factor.[7] Skeletal muscle atrophy develops below the injury level after SCI. Compared to body mass index (BMI)-matched healthy individuals, those with SCI have been found to exhibit lower muscle mass and higher fat mass.[8] An increase in both whole-body and central adiposity is observed, which is positively correlated with triglyceride (TG) levels and negatively correlated with high-density lipoprotein cholesterol (HDL-C) levels.[9,10] Increased adiposity further elevates the risks of dyslipidemia and CVD.[11,12] Lipid profiles in individuals with SCI differ significantly from those of able-bodied controls. In a systematic review of 4,512 individuals with chronic SCI, the mean low-density lipoprotein cholesterol (LDL-C) level was found to be 115.5±14.6 mg/dL.[13] Koyuncu et al. reported that, in patients with SCI, the mean TC level exceeded the upper limit of normal by 21%, while the LDL-C and TG levels were 24% and 31% higher, respectively. Additionally, the HDL-C level was below 40 mg/dL in 80% of the patients.[14] In a study of 9,081 individuals with SCI in the United States, hypercholesterolemia was reported to be 5% more prevalent and associated with a 1.53-fold higher cardiac risk.[15] In a study of the Finnish SCI population, Tallqvist et al. found that hypercholesterolemia was present in 22% of individuals.[16] Understanding the extent of dyslipidemia within the SCI population is crucial, as it is a well-established risk factor for CVD, which ranks among the leading causes of death in individuals with SCI.

A wheelchair is the primary mobility tool for individuals with SCI.[17] Manual Wheelchair Propulsion Tests (MWPTs) are commonly used to assess mobility. These tests provide more comprehensive information than loggers that measure daily distance traveled or tests such as the "Wheelchair Skills Test", which merely indicate success or failure in performing specific skills.[18]

Following SCI, alterations in body composition, changes in lipid metabolism, and restricted mobility are commonly observed. Studies evaluating mobility in individuals with SCI are relatively limited, and research examining the relationship between mobility, obesity, and dyslipidemia is also scarce. The aims of this study are twofold: (i) to determine the prevalence of obesity and dyslipidemia in individuals with chronic SCI and (ii) to investigate the impact of these factors on mobility.

## MATERIAL AND METHODS

### PARTICIPANTS

A cross-sectional study was conducted after obtaining approval from the Academic Board of the Faculty of Medicine at the University of Health Sciences (date: June 28, 2017, no: 46418926/020). Between July 2017 and May 2018, individuals diagnosed with SCI who were enrolled in an inpatient rehabilitation program at the Ankara Physical Therapy and Rehabilitation Training and Research Hospital were included in the study.

A G*power (version 3.1.9.4, Heinrich Heine University in Düsseldorf, Germany) analysis was performed to calculate the sample size. Based on this analysis, the effect size was set at 0.50, with a type I error (α) value of 0.05 and a type II error (β) value of 0.20. Accordingly, the study power (1-β) was calculated as 0.80, and the sample size was determined to be 102 participants.

Participants were informed about the study's purpose and the tests to be conducted, both verbally and in writing, using a pre-prepared "Informed Consent Form" in accordance with the study protocol. Written consent was obtained from all participants who agreed to participate by signing the "Informed Consent Form". The study was conducted in accordance with the principles of the Declaration of Helsinki.

***Inclusion criteria***

- Age between 18 and 60 years.
- Disease duration of at least 1 year.
- Ability to sit without support for at least 30 seconds.
- Ability to tolerate at least 45 minutes of activity.
- Motor-complete individuals with traumatic or non-traumatic SCI (classified as American Spinal Cord Injury Association (ASIA) Impairment Scale (AIS) A or B).

***Exclusion criteria***

- Postoperative use of thoracolumbar orthosis or cervical braces.
- Secondary musculoskeletal disorders or pain in the trunk or upper extremities.
- Other conditions that could prevent the completion of tests (e.g., postural hypotension, uncontrolled hypertension, uncontrolled hyperglycemia, uncontrolled autonomic dysreflexia, or chronic arterial disease).

## OBESITY ASSESSMENT

Height and weight were measured according to the techniques outlined in the Anthropometric Standardization Reference Manual.[19] The BMI was calculated using the formula of body mass (kg)/[height (m)].[2] Both the World Health Organization (WHO) criteria and SCI population-adjusted data were used to assess obesity. According to the SCI-specific criteria, individuals with a BMI of ≥22 kg/m² were classified as obese, whereas those with a BMI of <22 kg/m² were classified as non-obese.[20,21] Binary groups were then formed. Additionally, based on the WHO criteria, individuals were categorized as underweight (BMI≤18.5 kg/m²), normal weight (BMI=18.5-24.9 kg/m²), overweight (BMI=25-29.9 kg/m²), and obese (BMI≥30 kg/m²).[22]

## ASSESSMENT OF THE LIPID PROFILE

Following a 12-h overnight fast, blood was drawn to assess the serum lipid profiles. An automated chemistry analyzer utilizing colorimetric methodology was employed to measure TC, HDL-C, and TG. LDL-C values were determined using Friedewald's equation: LDL-C=TC HDL-C (TG/5).[23] HDL-C, LDL-C, TG, and TC values were recorded. The values specified in the National Cholesterol Education Program Adult Treatment Panel III (NCEP/ATP III) III guideline were taken as reference.[24] Accordingly, HDL-C below 40 mg/dL was considered low, LDL-C above 130 mg/dL, TG above 150 mg/dL, and TC above 200 mg/dL were considered high, and paired groups were formed.

## ASSESSMENT OF MWPTS

**20-m Propulsion Test ($MWPT_{20m}$):** Participants were positioned with the front bar of their wheelchair at the starting line of the course and were instructed to propel the wheelchair as quickly as possible. The time to complete the 20-m course was measured and recorded in seconds (Figure 1).[18]

**Slalom Test ($MWPT_{SLALOM}$):** Participants navigated their wheelchairs through an 18-m slalom course at their maximum self-selected speed. The course featured seven cones arranged in a linear configuration with varying distances of 3, 2, and 1m between them. The time to complete the course was recorded in seconds (Figure 1).[18]

**6-Minute Propulsion Test ($MWPT_{6min}$):** An 8-shaped track with a length of 25 meters was designed for the test. Participants started at the center of the course and propelled as quickly as possible to the

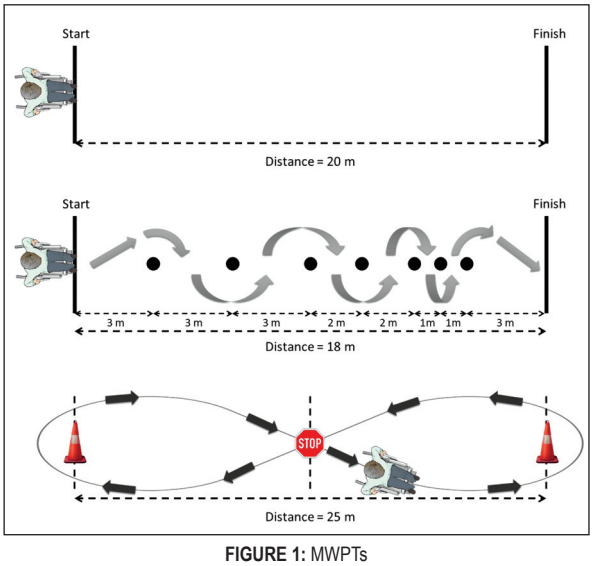


**FIGURE 1:** MWPTs
MWPT: Manual Wheelchair Propulsion Test

cones on the sides, then returned to the center, performed a rapid stop, and continued to the other side. This cycle was repeated for 6 minutes. The distance measured was recorded in meters (Figure 1).[18]

MWPT results were compared in binary groups consisting of SCI-specific BMI≥22 kg/m² (obese) vs. <22 kg/m² (non-obese); HDL-C below 40 mg/dL (low) vs. HDL-C above 40 mg/dL; LDL-C above 130 mg/dL (high) vs. LDL-C below 130 mg/dL; TG above 150 mg/dL (high) vs. TG below 150 mg/dL; and TC above 200 mg/dL (high) vs. TC below 200 mg/dL.

### STATISTICS

Data entry and analysis were performed using the "Statistical Package for Social Sciences" version 23.0 (IBM Corp., Armonk, NY, USA). The conformity of the variables to the normal distribution was checked by the Kolmogorov-Smirnov and Shapiro-Wilk tests. The independent samples t-test was used to compare independent 2-group continuous variables that conformed to the normal distribution. The Mann-Whitney U test was used to compare independent 2-group continuous variables that did not fit the normal distribution. Statistical significance was set at $p<0.05$.

## RESULTS

Table 1 presents the demographic profile of the study participants. The mean age of the cohort was 34.6±11.9 years. The mean time since the date of SCI was 5.36±4.94 years.

### OBESITY

According to the SCI-specific BMI, 28.4% (n=29) of the participants were not obese and 71.6% (n=73) were obese. According to the WHO criteria, 4.9% (n=5) of the participants were underweight, 56.9% (n=58) were of normal weight, 27.4% (n=28) were overweight, and 10.8% (n=11) were obese.

### LIPID PROFILE

The mean HDL-C was 35.7±8.68 mg/dL, LDL-C was 107.4±31.7 mg/dL, TG was 153.7±72.7 mg/dL, and TC was 175±36.8 mg/dL. Among all participants, 69.6% had low HDL-C, 27.4% had high LDL-C, 45.09% had high TG, and 22.5% had high TC.

A comparison of HDL-C, LDL-C, TG, and TC between the AIS-A and AIS-B groups is shown in Table 2. The mean TG level was significantly higher in the AIS-A group than in the AIS-B group (p=0.014). The differences in HDL-C, LDL-C, and TC levels between the groups were not statistically significant (p=0.12, 0.756, and 0.69, respectively).

**TABLE 1:** Characteristics of the participants

| Outcome Measure | | Number (n) | Percentage (%) |
|---|---|---|---|
| Gender | Male | 79 | 77.5 |
| | Female | 23 | 22.5 |
| Age (year) | 18-24 | 28 | 27.3 |
| | 25-31 | 23 | 22.6 |
| | 32-45 | 26 | 25.5 |
| | 46-60 | 25 | 24.6 |
| Etiology | Traumatic | 86 | 84.4 |
| | Non-traumatic | 16 | 15.7 |
| ASIA Impairment Scale | A | 50 | 49 |
| | B | 52 | 51 |
| Neurological injury level | C6 | 3 | 3 |
| | C7 | 2 | 2 |
| | C8-T1 | 1 | 1 |
| | T2-T10 | 57 | 55.8 |
| | T11-L2 | 39 | 38.2 |

ASIA: American Spinal Cord Injury Association

**TABLE 2:** Comparison of lipid profile results according to AIS classification

| | AIS A | | AIS B | | |
|---|---|---|---|---|---|
| | n | X̄±SD | n | X̄±SD | p value |
| HDL-C (mg/dL) | 50 | 34.4±8.16 | 52 | 37.1±9.1 | 0.120 |
| LDL-C (mg/dL) | 50 | 108.4±29.2 | 52 | 106.5±34.1 | 0.756 |
| TG (mg/dL) | 50 | 170.9±80.6 | 52 | 135.8±59.1 | **0.014*** |
| TC (mg/dL) | 50 | 175.1±37.4 | 52 | 171.9±42.5 | 0.692 |

*p<0.05; AIS: American Spinal Cord Injury Association Impairment Scale; SD: Standard deviation; HDL-C: High-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TG: triglycerides; TC: Total cholesterol

**TABLE 3:** MWPT outcome measures in individuals with spinal cord injury

| Wheelchair Propulsion Tests | n | X̄±SD | Minimum-Maximum |
|---|---|---|---|
| 20-m propulsion test (s) | 102 | 13.3±5.18 | 7-35 |
| Slalom test (s) | 102 | 28.6±11.8 | 13-80 |
| 6-min propulsion test (m) | 102 | 625.8±173.6 | 180-1200 |

SD: Standard deviation; s: Second; m: Meter

**TABLE 4:** Effect of HDL-C, LDL-C, TG, TC, and BMI on MWPT results

| | 20-m propulsion test (s) | | | | Slalom test (s) | | | | 6-min propulsion test (m) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | X̄±SD | Minimum-maximum | p value | n | X̄±SD | Minimum-maximum | p value | n | X̄±SD | Minimum-maximum | p value |
| HDL-C< 40 (mg/dL) | 71 | 12.8±4.00 | 7-30 | 0.44 | 71 | 26.5±7.60 | 13-45 | 0.06 | 71 | 646.8±170.7 | 375-1200 | 0.11 |
| HDL-C≥ 40 (mg/dL) | 31 | 14.5±7.15 | 7-35 | | 31 | 33.4±17.3 | 14-80 | | 31 | 577.7±173.6 | 180-900 | |
| LDL-C< 130 (mg/dL) | 74 | 13.4±5.51 | 7-35 | 0.61 | 74 | 29.3±13.0 | 13-80 | 0.69 | 74 | 609.8±160.9 | 180-1050 | 0.30 |
| LDL-C≥ 130 (mg/dL) | 28 | 12.9±4.28 | 8.74-25 | | 28 | 26.9±7.68 | 14-44 | | 28 | 668.2±250 | 300-1200 | |
| TG<150 (mg/dL) | 56 | 12.9±6.04 | 7-35 | **0.014*** | 56 | 28.9±14.7 | 13-80 | 0.196 | 56 | 638.8±189.0 | 180-1200 | 0.49 |
| TG≥150 (mg/dL) | 46 | 13.7±3.93 | 9-30 | | 46 | 28.3±6.8 | 16-45 | | 46 | 610±153.5 | 300-1050 | |
| TC< 200 (mg/dL) | 79 | 13.2±5.39 | 7-35 | 0.60 | 79 | 28.9±12.8 | 13-80 | 0.86 | 79 | 608.2±156.9 | 180-1050 | 0.10 |
| TC≥ 200 (mg/dL) | 23 | 13.6±4.50 | 9-25 | | 23 | 27.7±7.53 | 14-43 | | 23 | 686.3±215.1 | 300-1200 | |
| BMI<22 kg/m$^2$ | 29 | 11.3±4.21 | 7-30 | **0.014*** | 29 | 23.7±5.88 | 13-40 | **0.008*** | 29 | 683.1±141.8 | 450-925 | **0.035*** |
| BMI≥22 kg/m$^2$ | 73 | 14.1±5.35 | 8-35 | | 73 | 30.6±12.9 | 15-80 | | 73 | 603±180.7 | 180-1200 | |

*p<0.05; SD: Standard deviation; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; TG: Triglycerides; TC: Total cholesterol; BMI: Body mass index

### MWPTS

The results of the MWPTs for the participants are shown in Table 3.

The effects of obesity and dyslipidemia on the MWPT results are shown in Table 4. Participants were divided into paired groups according to HDL-C, LDL-C, TG, TC, and BMI measurements. The effects of the $MWPT_{20m}$, $MWPT_{SLALOM}$, and $MWPT_{6min}$ were compared between the paired groups. The time to complete the $MWPT_{20m}$ was significantly lower in the group with TG<150 mg/dl compared with the group with TG³150 mg/dl (p=0.014). However, no significant relationship was found between the groups in the $MWPT_{SLALOM}$ and the $MWPT_{6min}$ (p=0.196 and 0.49, respectively). The shorter time to complete the $MWPT_{20m}$ and $MWPT_{SLALOM}$ and the longer distance traveled in the $MWPT_{6min}$ were statistically significant in the group with BMI <22 kg/m$^2$ compared to the group with BMI≥22 kg/m$^2$ (p=0.014; 0.008; 0.035, respectively). The effect of the binary groups for HDL-C, LDL-C, and TC on the MWPTs was not statistically significant (p>0.05).

## DISCUSSION

Our study revealed the effect of obesity and dyslipidemia on mobility using MWPTs in individuals with chronic motor complete SCI. It was shown that the SCI-specific BMI criteria diagnosed obesity more frequently than the WHO criteria. Regarding dyslipidemia, low HDL-C and high TG were found to be at the forefront. TG levels were statistically significantly higher in individuals with AIS-A compared with those with AIS-B. Regarding the effect of obesity on mobility, obese individuals demonstrated slower performance in the $MWPT_{20m}$ and $MWPT_{SLALOM}$ and covered a shorter distance in the $MWPT_{6min}$. Regarding the effect of dyslipidemia on mobility, it was shown that individuals with hypertriglyceridemia demonstrated slower performance in the $MWPT_{20m}$.

In SCI, neurogenic obesity may develop as a result of physical inactivity due to motor impairment, negative energy balance, and progressive loss of muscle and bone mass due to interruption of neuronal stimulation below the level of injury.[25-27] In the first study on the need for a separate classification for the assessment of obesity, Laughton et al. showed that the WHO classification may miss 73.9% of individuals with SCI and that their body fat percentage was 8-18% higher than that of healthy controls. They suggested that those with a BMI ≥22 kg/m² in individuals with chronic SCI should be considered to be at high risk of obesity and related chronic diseases.[21] In a study conducted by Hatchett et al. on 222 individuals with chronic SCI, 13.5% were found to be obese

according to the WHO classification and 44.1% according to SCI-specific BMI.[28] In a study including 136 individuals with SCI with an obesity limit of 22 kg/m$^2$, 77.9% were shown to be obese.[29] In our study, we used both the WHO and SCI-specific BMI criteria. Consistent with the literature, we evaluated 10.8% of individuals as obese according to the WHO criteria and 71.6% of individuals as obese according to the SCI-specific BMI criteria.

Dyslipidemia is a common cardiometabolic risk factor after SCI.[30] It is frequently characterized by low HDL-C and elevated TG.[19,23,31] Wahman et al. compared wheelchair-dependent chronic paraplegics (n=135) with age-gender matched healthy individuals (n=1,488) in terms of cardiovascular risk factors. Accordingly, the prevalence of dyslipidemia was 11.1% in individuals with SCI, whereas it was 1.8% in healthy controls, and the difference was found to be statistically significant.[32] Bauman et al. compared the HDL-C levels of 320 paraplegic patients with those of relatively sedentary healthy controls. The serum HDL-C level was reduced in the SCI group compared with the control group.[33] In a study conducted on 253 individuals with chronic SCI, low HDL-C was found to be 85%.[34] A study by Paim et al. showed that the mean LDL-C was 114.6±9.1 mg/dl in sedentary individuals with SCI and 101.9±5.5 mg/dl in physically active individuals with SCI.[35] In a study conducted by Vichiansiri, the TC value was found to be 175.1±41.3 mg/dl.[36] In our study, similar to the literature, the mean HDL-C value was 35.7±8.68 mg/dl and 69.6% of the participants had low HDL-C; the mean LDL-C value was 107.4±31.7 mg/dl, and 27.4% had high LDL-C, and the mean TC value was 175±36.8 mg/dl and 22.5% had high TC. This may be due to the reduced physical activity and immobilization associated with SCI, which can alter lipid metabolism. Decreased mobility generally results in lower energy expenditure and muscle mass, potentially contributing to dyslipidemia, including low HDL-C and altered LDL-C and TC levels.

In a study, it was shown that TG value was positively correlated with visceral adipose tissue in individuals with chronic SCI.[37] In studies, mean TG values were found to be 202.4±119.8 mg/dl and 128.6±65.6 mg/dl.[38,39] In our study, the mean TG of all participants was 153.7±72.7 mg/dl and was found to be high in 45.1% of patients. We believe that the different results in the literature may be related to the diet and exercise habits of individuals. The risk of developing cardiometabolic diseases such as dyslipidemia and obesity has been found to be higher in complete SCIs than in incomplete SCIs due to less physical activity.[11] Although no correlation has been found between serum TG levels and AIS in studies, in our study, we found significantly higher TG levels in patients with AIS-A compared with those with AIS-B.[14,36] We associated this with higher physical inactivity in individuals with complete SCI.

MWPTs are inexpensive and simple tests used to assess mobility. It has been shown that tasks such as going back and forth and changing direction in these tests can be applied to improve the ability to use the wheelchair.[40-43] In the $MWPT_{20m}$, it is necessary to apply symmetrical force to the wheelchair; energy is generated during linear displacement. In the $MWPT_{SLALOM}$, asymmetric force is applied to change directions along the trajectory, with energy production and consumption.[44] The $MWPT_{20m}$ and $MWPT_{SLALOM}$ assess anaerobic capacity, whereas $MWPT_{6min}$ evaluates endurance, i.e., aerobic capacity.

Obesity causes limitations in daily living activities, transfers, and manual wheelchair mobilization in individuals with SCI.[3,4] A person's body weight affects their ability to maneuver a wheelchair, with individuals having a higher BMI generally unable to push as far as those with a lower BMI.[45] It has been shown that individuals who experience at least a 5% increase in body weight have greater difficulty propelling a wheelchair. Their propulsion speed was lower than that of individuals who did not gain weight, and the total distance covered was also significantly negatively affected. It has been reported that obese individuals with SCI propel their wheelchairs 19% less per day on average.[28] Remarkably, in a study of veterans who had recently developed SCI, BMI increased over 5 years despite a decrease in muscle and bone mass, suggesting that weight changes mainly reflect an increase in adipose tissue.[3,46] In individuals with incomplete paraplegia, obese patients showed smaller improvements in FIM self-care and mobility scores compared with those of

normal weight. For those with complete paraplegia, obese patients experienced significantly lower gains in self-care and mobility scores. However, in cases of both incomplete and complete tetraplegia, there were no significant differences in FIM self-care and mobility scores between obese and normal-weight patients.[47] Consistent with the literature, our study revealed statistically significant differences, with obese patients demonstrating longer $MWPT_{20m}$ and $MWPT_{SLALOM}$ times and shorter $MWPT_{6min}$ distances compared to non-obese patients. A study by the Human Engineering Research Laboratories revealed that manual wheelchair users with a BMI greater than 25 kg/m² propelled at a notably slower speed compared to those with a BMI below 25 kg/m² when navigating surfaces commonly encountered in daily life, such as ramps, thick carpet, and grass.[48] The findings of our study demonstrate that obesity limits mobility in individuals with SCI, causing difficulties in wheelchair mobilization and negatively affecting physical performance. Consistent with the literature, obese individuals demonstrated shorter wheelchair propulsion distances and longer propulsion times compared with those with lower BMI. This could be due to the increased body weight and adipose tissue, which restrict the mobilization abilities. Additionally, these patients may have experienced reduced mobility due to lower muscle strength and endurance, with decreased physical activity levels potentially contributing to the development of obesity.

Following SCI, dyslipidemia is linked to significant physical deconditioning and systemic inflammation, both of which are connected to the rapid development of neurogenic obesity, insulin resistance, and type 2 diabetes mellitus. Compared with non-disabled individuals, those with SCI face a higher risk of developing these health complications.[49] Comparisons between physically active and inactive men with SCI showed notable differences in total body weight, BMI, and fat mass.[7,50] Men and women with SCI who are physically active have total body fat percentages ranging from 16% to 24% for men and 24% to 32% for women. In contrast, sedentary individuals with SCI exhibit "at-risk" fat mass levels, exceeding 25% in men and 32% in women.[7] Farkas et al. identified notable associations between visceral fat measured by MRI and the ratio of visceral-to-subcutaneous fat with TG, HDL-C, and the TC:HDL-C ratio, specifically in individuals with paraplegia. However, these connections were not present in cases of tetraplegia.[51] Similarly, Gorgey et al. found multiple significant relationships between lipid metabolism markers and abdominal obesity measured via magnetic resonance imaging (MRI) in individuals with SCI. The study noted that HDL-C, the TC: HDL-C ratio, and TG levels were associated with both upper and lower visceral fat, subcutaneous fat, and the ratio between these two fat depots.[37] No study examining the effect of lipid profile on the MWPTs was found in the literature. In our study, the effect of HDL-C, LDL-C, and TC binary groups on the MWPTs were not statistically significant. The $MWPT_{20m}$ duration was 13.8±3.93 seconds in the group with TG≥150 mg/dL and 13±6.04 seconds in the group with TG<150 mg/dL, and this difference was statistically significant. Although we did not evaluate it in our study, it has been shown in previous studies that hypertriglyceridemia is associated with visceral adipose tissue.[36] The results of our study may reflect the complex relationship between TG levels and physical inactivity in individuals with SCI. High TG levels are associated with central obesity, which may result from decreased physical activity and subsequent accumulation of visceral adipose tissue. Since visceral fat is associated with lipid metabolism disorders, it is likely that individuals with higher TG levels experience increased fat accumulation, which may prolong the $MWPT_{20m}$ completion times. Furthermore, the physical inactivity of these individuals may have intensified the metabolic disorders seen in SCI and led to longer propulsion times.

## LIMITATIONS

The lack of a healthy control group was a limitation of our study. We thought that healthy individuals may affect the results of the MWPTs because they are not accustomed to the use of a manual wheelchair. The absence of a healthy control group may have limited our ability to determine a definitive cause-and-effect relationship. Additionally, the cross-sectional design of our study may have further constrained this as-

sessment. Another limitation is that body fat and muscle mass were not evaluated.

## CONCLUSION

In our study investigating the effect of obesity and dyslipidemia on wheelchair mobilization in individuals with chronic motor complete SCI, hypertriglyceridemia, and obesity were shown to have a negative effect. The use of SCI-specific BMI criteria may be preferred over WHO criteria to avoid missing cases of obesity in individuals with SCI. In the future, randomized controlled trials are needed to evaluate the effect of the effectiveness of treatments for obesity and dyslipidemia on mobilization in individuals with SCI.



## REFERENCES


1. Rajan S, McNeely MJ, Warms C, et al. Clinical assessment and management of obesity in individuals with spinal cord injury: a review. J Spinal Cord Med. 2008;31:361-72. PMID: 18959353; PMCID: PMC2582426.
2. de Groot S, Post MW, Postma K, et al. Prospective analysis of body mass index during and up to 5 years after discharge from inpatient spinal cord injury rehabilitation. J Rehabil Med. 2010;42:922-8. PMID: 21031288.
3. Crane DA, Little JW, Burns SP. Weight gain following spinal cord injury: a pilot study. J Spinal Cord Med. 2011;34:227-32. PMID: 21675361; PMCID: PMC3066508.
4. Gater DR Jr. Obesity after spinal cord injury. Phys Med Rehabil Clin N Am. 2007;18:333-51, vii. PMID: 17543776.
5. Eriks-Hoogland I, Hilfiker R, Baumberger M, et al. Clinical assessment of obesity in persons with spinal cord injury: validity of waist circumference, body mass index, and anthropometric index. J Spinal Cord Med. 2011;34:416-22. PMID: 21903016; PMCID: PMC3152814.
6. Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999-2000. JAMA. 2002;288:1723-7. PMID: 12365955.
7. Spungen AM, Adkins RH, Stewart CA, et al. Factors influencing body composition in persons with spinal cord injury: a cross-sectional study. J Appl Physiol (1985). 2003;95:2398-407. PMID: 12909613.
8. Wilmet E, Ismail AA, Heilporn A, et al. Longitudinal study of the bone mineral content and of soft tissue composition after spinal cord section. Paraplegia. 1995;33:674-7. PMID: 8584304.
9. Gorgey AS, Gater DR. Regional and relative adiposity patterns in relation to carbohydrate and lipid metabolism in men with spinal cord injury. Appl Physiol Nutr Metab. 2011;36:107-14. PMID: 21326384.
10. Bauman WA, Spungen AM, Zhong YG, et al. Depressed serum high density lipoprotein cholesterol levels in veterans with spinal cord injury. Paraplegia. 1992;30:697-703. PMID: 1448297.
11. Bauman WA, Spungen AM. Carbohydrate and lipid metabolism in chronic spinal cord injury. J Spinal Cord Med. 2001;24:266-77. PMID: 11944785.
12. Bauman WA, Spungen AM. Metabolic changes in persons after spinal cord injury. Phys Med Rehabil Clin N Am. 2000;11:109-40. PMID: 10680161.
13. Gilbert O, Croffoot JR, Taylor AJ, et al. Serum lipid concentrations among persons with spinal cord injury - a systematic review and meta-analysis of the literature. Atherosclerosis. 2014;232:305-12. PMID: 24468143.
14. Koyuncu E, Nakipoğlu Yüzer GF, Yenigün D, et al. The analysis of serum lipid levels in patients with spinal cord injury. J Spinal Cord Med. 2017;40:567-72. PMID: 27735233; PMCID: PMC5815153.
15. Peterson MD, Berri M, Lin P, et al. Cardiovascular and metabolic morbidity following spinal cord injury. Spine J. 2021;21(9):1520-7. PMID: 34023517; PMCID: PMC9645293.
16. Tallqvist S, Kauppila AM, Vainionpää A, et al. Prevalence of comorbidities and secondary health conditions among the Finnish population with spinal cord injury. Spinal Cord. 2022;60:618-27. PMID: 34511604; PMCID: PMC9287167.
17. Goktepe AS, Tugcu I, Yilmaz B, et al. Does standing protect bone density in patients with chronic spinal cord injury? J Spinal Cord Med. 2008;31:197-201. PMID: 18581668; PMCID: PMC2565474.
18. Gagnon DH, Roy A, Verrier MC, et al. Do performance-based wheelchair propulsion tests detect changes among manual wheelchair users with spinal cord injury during inpatient rehabilitation in quebec? Arch Phys Med Rehabil. 2016;97:1214-8. PMID: 26987621.
19. Harrison GG, Buskirk ER, Carter JE. Skinfold thicknesses and measurement technique. In: Lohman T, Roche A, Martorell R, eds. Anthropometric Standardization Reference Manual. Champaign: Human Kinetics Books; 1988. p. 55-80.
20. Silveira SL, Ledoux TA, Robinson-Whelen S, et al. Methods for classifying obesity in spinal cord injury: a review. Spinal Cord. 2017;55:812-7. PMID: 28695902.
21. Laughton GE, Buchholz AC, Martin Ginis KA, et al; SHAPE SCI Research Group. Lowering body mass index cutoffs better identifies obese persons with spinal cord injury. Spinal Cord. 2009;47:757-62. PMID: 19350042.
22. World Health Organization. (10.6. 2024c). A healthy lifestyle - WHO recommendations: Body Mass İndex. https://www.who.int/europe/news-room/fact-sheets/item/a-healthy-lifestyle---who-recommendations
23. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18:499-502. PMID: 4337382.
24. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation. 2002;106:3143-421. PMID: 12485966.
25. Svircev JN. Cardiovascular disease in persons with spinal cord dysfunction-an update on select topics. Phys Med Rehabil Clin N Am. 2009;20:737-47. PMID: 19781509.
26. Groah SL, Nash MS, Ward EA, et al. Cardiometabolic risk in community-dwelling persons with chronic spinal cord injury. J Cardiopulm Rehabil Prev. 2011;31:73-80. PMID: 21045711.
27. Gater DR Jr, Farkas GJ, Tiozzo E. Pathophysiology of neurogenic obesity after spinal cord injury. Top Spinal Cord Inj Rehabil. 2021;27:1-10. PMID: 33814879; PMCID: PMC7983633.
28. Hatchett PE, Mulroy SJ, Eberly VJ, et al. Body mass index changes over 3 years and effect of obesity on community mobility for persons with chronic spinal cord injury. J Spinal Cord Med. 2016;39:421-32. PMID: 26781601; PMCID: PMC5102291.

29. Pelletier CA, Miyatani M, Giangregorio L, et al. Sarcopenic obesity in adults with spinal cord injury: a cross-sectional study. Arch Phys Med Rehabil. 2016;97:1931-7. PMID: 27282328.

30. Jörgensen S, Hill M, Lexell J. Cardiovascular risk factors among older adults with long-term spinal cord injury. PM R. 2019;11:8-16. PMID: 29964213.

31. Solinsky R, Betancourt L, Schmidt-Read M, et al. Acute spinal cord ınjury ıs associated with prevalent cardiometabolic risk factors. Arch Phys Med Rehabil. 2022;103:696-701. PMID: 34062117.

32. Wahman K, Nash MS, Westgren N, et al. Cardiovascular disease risk factors in persons with paraplegia: the Stockholm spinal cord injury study. J Rehabil Med. 2010;42:272-8. PMID: 20419873.

33. Bauman WA, Adkins RH, Spungen AM, et al. Is immobilization associated with an abnormal lipoprotein profile? observations from a diverse cohort. Spinal Cord. 1999;37:485-93. PMID: 10438115.

34. Köseoğlu BF, Safer VB, Öken Ö, et al. Cardiovascular disease risk in people with spinal cord injury: is there a possible association between reduced lung function and increased risk of diabetes and hypertension? Spinal Cord. 2017;55:87-93. PMID: 27377303.

35. Paim LR, Schreiber R, Matos-Souza JR, et al. Oxidized low-density lipoprotein, matrix-metalloproteinase-8 and carotid atherosclerosis in spinal cord injured subjects. Atherosclerosis. 2013;231:341-5. PMID: 24267248.

36. Vichiansiri R, Saengsuwan J, Manimmanakorn N, et al. The prevalence of dyslipidemia in patients with spinal cord lesion in Thailand. Cholesterol. 2012;2012:847462. PMID: 22848801; PMCID: PMC3400296.

37. Gorgey AS, Farkas GJ, Dolbow DR, et al. Gender dimorphism in central adiposity may explain metabolic dysfunction after spinal cord ınjury. PM R. 2018;10:338-48. PMID: 28827208.

38. Nash MS, Jacobs PL, Mendez AJ, et al. Circuit resistance training improves the atherogenic lipid profiles of persons with chronic paraplegia. J Spinal Cord Med. 2001;24:2-9. PMID: 11587430.

39. Akbal A, Kurtaran A, Selçuk B, et al. H-FABP, cardiovascular risk factors, and functional status in asymptomatic spinal cord injury patients. Herz. 2013;38:629-35. PMID: 23483223.

40. Fliess-Douer O, Vanlandewijck YC, Lubel Manor G, et al. A systematic review of wheelchair skills tests for manual wheelchair users with a spinal cord injury: towards a standardized outcome measure. Clin Rehabil. 2010;24:867-86. PMID: 20554638.

41. Kilkens OJ, Dallmeijer AJ, De Witte LP, et al. The wheelchair circuit: construct validity and responsiveness of a test to assess manual wheelchair mobility in persons with spinal cord injury. Arch Phys Med Rehabil. 2004;85:424-31. PMID: 15031828.

42. Kilkens OJ, Post MW, van der Woude LH, et al. The wheelchair circuit: reliability of a test to assess mobility in persons with spinal cord injuries. Arch Phys Med Rehabil. 2002;83:1783-8. PMID: 12474187.

43. Kirby RL, Swuste J, Dupuis DJ, et al. The wheelchair skills test: a pilot study of a new outcome measure. Arch Phys Med Rehabil. 2002;83:10-8. PMID: 11782826.

44. Gagnon D, Verrier M, Masani K, et al. Effects of trunk impairments on manual wheelchair propulsion among individuals with a spinal cord injury: a brief overview and future challenges. Topics in Spinal Cord Injury Rehabilitation. 2009;15:59-70. https://doi.org/10.1310/sci1502-59

45. Vives Alvarado JR, Felix ER, Gater DR Jr. Upper extremity overuse injuries and obesity after spinal cord injury. Top Spinal Cord Inj Rehabil. 2021;27:68-74. PMID: 33814884; PMCID: PMC7983631.

46. Gater DR, Farkas GJ. Alterations in body composition after SCI and the mitigating role of exercise. In: Taylor JA, eds. The Physiology of Exercise in Spinal Cord Injury. Boston: Springer; 2016. p.175-98.

47. Stenson KW, Deutsch A, Heinemann AW, et al. Obesity and inpatient rehabilitation outcomes for patients with a traumatic spinal cord injury. Arch Phys Med Rehabil. 2011;92:384-90. PMID: 21276960.

48. Mercer JL, Boninger ML, Koontz AM, Pearlman J, Boninger D, Cooper RA. Effect of weight on wheelchair propulsion over various surfaces. In: Proceedings of the 28th Annual RESNA Conference [on CD-ROM]; June 25- 27, 2005; Atlanta, GA.

49. Nash MS, Farkas GJ, Tiozzo E, et al. Exercise to mitigate cardiometabolic disorders after spinal cord injury. Curr Opin Pharmacol. 2022;62:4-11. PMID: 34864560.

50. D'Oliveira GL, Figueiredo FA, Passos MC, et al. Physical exercise is associated with better fat mass distribution and lower insulin resistance in spinal cord injured individuals. J Spinal Cord Med. 2014;37:79-84. PMID: 24090139; PMCID: PMC4066554.

51. Farkas GJ, Gorgey AS, Dolbow DR, et al. The influence of level of spinal cord injury on adipose tissue and its relationship to inflammatory adipokines and cardiometabolic profiles. J Spinal Cord Med. 2018;41:407-15. PMID: 28758566; PMCID: PMC6055972.