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Mind Matters: Examining the Relationship between Mental Lifestyle Determinants and Muscle Mass

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ABSTRACT

Introduction: There was evidence of a relationship between changes in muscle mass and mental health. Depression, stress, anxiety, sleep quality, and smoking are all aspects of one's mental lifestyle. The current study aimed to evaluate the relationship between muscle mass and intangible mental lifestyle variables in a population of Mashhad University of Medical Sciences employees.

Methods: This study used PERSIAN cohort data from 4572 Mashhad University of Medical Sciences employees aged between 30 to 70 who volunteered for the employee health-monitoring plan.

Results: The results showed that factors such as DASS21 (P=0.002, β=-0.047), PSQI (P<0.001, β=-0.064), and smoking (P<0.001, β=-0.207) had a significant negative relationship with skeletal muscle mass index. The structural model of the final hypothesis showed remarkable validity with a high R^2.

Conclusions: The results demonstrated a strong correlation between skeletal muscle mass and mental lifestyle factors, such as smoking, sleep hygiene, and mental health. In the current study, the most significant relationship is related to smoking.

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Introduction

The term "lifestyle" refers to a person's, a group's, or a culture's habits, attitudes, tastes, moral standards, and other components (1). Intangible characteristics are related to an individual's psychological aspects, whereas tangible components are directly related to demographic variables (2-4). Muscular mass and strength are influenced by tangible lifestyle determinants and intangible psychological variables (5). Smoking, diet, and physical activity are examples of lifestyle variables, which significantly affect strength and muscle mass (6). Mental health and lifestyle issues like sleep and smoking are inextricably related. Individuals suffering from mental health issues such as anxiety, depression, and so on frequently have major sleep disturbances, including insomnia, nightmares, and excessive daytime drowsiness (7), which can increase mental illness symptoms and result in lower overall health outcomes (7). Smoking, a typical coping method for stress and anxiety, might exacerbate these problems by reducing sleep quality and quantity (8). Addressing sleep issues and quitting smoking should be included in complete mental health treatment regimens to promote overall well-being and prevent the perpetuation of this vicious cycle (8).

A robust correlation between mental health disorders such as depression, anxiety, and stress and changes in muscle mass and strength has been considered (9). Individuals with mental health issues frequently have decreased muscle mass as a result of variables such as diminished physical activity, changing hormone levels, and inflammatory processes (10).

Stress, anxiety, and depression have been shown in studies to impair muscle growth, implying mental health can influence muscle mass (11, 12). Stress negatively impacts muscle mass through various mechanisms, including altered gene expression, inflammation, and protein turnover. Daily acute psychological stress is

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associated with myostatin-dependent muscular atrophy and increased expression of atrophic genes, both of which lead to a decrease in body and muscle mass (13). Hypercortisolism, low-grade systemic inflammation, and prolonged stress can potentially have a negative impact on muscle mass (10).

Sleep is essential for both physiological and cognitive functions. Inadequate sleep causes the blood to have more catabolic hormones, such as cortisol, and less anabolic hormones. According to a recent notion, manipulating the sleep-wake cycle may be detrimental to the health of skeletal muscles (14). There are multiple mechanisms via which the quality of sleep affects muscle mass. Lack of sleep can negatively impact muscle mass because stress and stress hormones affect strength and muscle protein turnover. Muscular strength may decrease due to muscle protein catabolism brought on by stress hormones generated during restless nights (15). Long-term stress can increase cortisol levels, which can hinder the body's ability to secrete chemicals necessary for muscular growth, such as testosterone, and slow down the growth and repair of muscle. Muscle mass can be negatively impacted by low-grade systemic inflammation, persistent stress, and the hypercortisolism that results from these factors (10).

There are negative effects of smoking on muscle mass and strength. Research has indicated that smoking is associated with decreased muscle mass and strength and increased discomfort in the muscles (16). Smoking can negatively affect muscle mass by increasing systemic inflammation and oxidative stress, reducing the synthesis of muscle proteins, and speeding up the breakdown of muscle proteins. Additionally, smoking may affect metabolism and muscle performance by reducing the amount of oxygen delivered to muscles through altered vasoconstriction. Therefore, the negative effects of smoking on muscle hypertrophy are mediated by a combination of increased inflammation, reduced muscle protein turnover, and enhanced oxidative stress (10).

Additional studies can yield essential information for creating all-encompassing interventions considering health's psychological and physical components. Most studies have focused on a single factor, and there is a lack of research examining multiple variables simultaneously, particularly in Iran. This study innovatively employs the Partial Least Squares (PLS) statistical model to investigate the simultaneous relationships between muscle mass and essential intangible lifestyle factors like sleep quality, mental health, and smoking among Mashhad University of Medical Sciences employees.

Materials & Methods

Research Design

Data from the PERSIAN cohort—a group of 4572 Mashhad University of Medical Sciences employees aged 30 to 70—were used in this cross-sectional investigation. Study participants were asked to understand the relationship between mental lifestyle determinants and muscle mass by examining the dependent variable, skeletal muscle mass index (SMI), and independent variables, Depression Anxiety Stress Scale-21 (DASS21), Pittsburgh Sleep Quality Index (PSQI), and smoking. Out of the initial cohort (5622), 4572 participants were included after applying inclusion and exclusion criteria. Additionally, the "ten times rule," which states that the minimum sample size should be at least ten times the highest number of pathways in the structural or formative measurement models, was frequently applied in PLS-SEM. Partial Least Squares Structural Equation Modeling (PLS-SEM) has emerged as a widely accepted method for examining intricate relationships between observed and hidden variables. Based on these guidelines, a minimum sample size of 390 was considered appropriate for this investigation (17, 18). A thorough analysis of various variables was made possible by a sample size of 4572, as demonstrated by applying the PLS-SEM method for error inflation compensation. A conceptual model was created, and latent variables were generated after carefully cleaning the data to remove missing values and compute the required indicators.

Measures and Covariates

The data required for this study (PERSIAN Cohort) came from Mashhad University of Medical Sciences' Health Monitoring Centre. This information was collected by skilled workers, including nurses, doctors, nutritionists, and interviewers, and the validity and reliability of the surveys were assessed (19, 20).

The necessary indicators were identified when the data was carefully cleaned using the natural range of the variables. The total score of the SMI,

DASS21, and PSQI was then calculated using the information from each person's completed questionnaires.

$SMI(m)=ASM/height^2$

Theoretical Framework

Reading several studies first created a figurative map of the assumptions. After the latent variables were defined, a conceptual assumptions model was developed using PLS-SEM (partial least squares structural equation modeling) version 3, 2, 8 (Figure 1). Once a non-significant relationship was eliminated, several conceptual models were developed, and each new model was constructed. The final conceptual model was developed based on P-values less than 0.001. The final hypothesis is depicted in Figure 1.

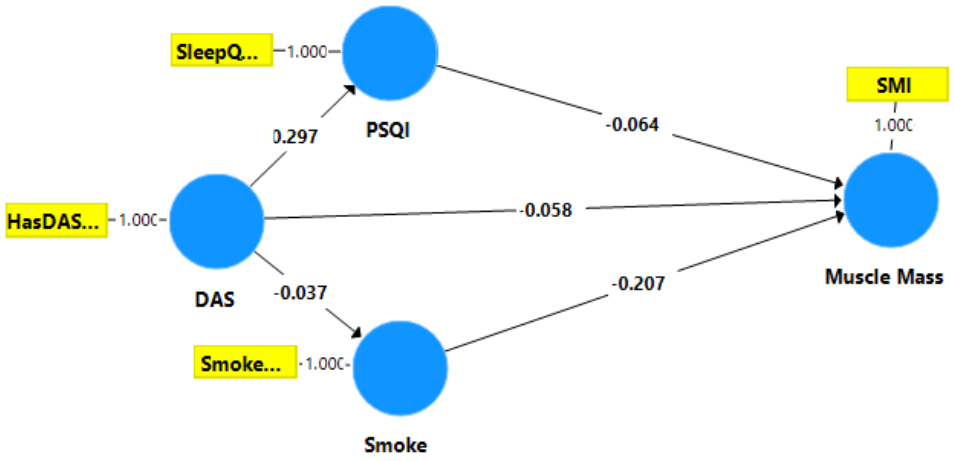

Figure 1. Final partial least squares model (Hfinal).

Analytic Procedure

Index computation was part of the statistical analysis with SPSS version 25. The study utilized partial least squares structural equation modeling (PLS-SEM) to examine the correlation between latent variables. There were several reasons for selecting PLS-SEM, including incremental research (21), the requirement for latent variable scores for further analysis (22), and an assessment of a theoretical framework from a predictive perspective. The data analysis (23), which followed the most recent evaluative PLS-SEM analysis criteria given by Ghasemy, Teeroovengadum et al. (2020) (24), was conducted using SmartPLS 3 (24).

Bootstrapping analyses in PLS-SEM and the algorithm were employed to test hypotheses and evaluate the importance of relationships in the conceptual model. The structural equation model consisted of a measurement model illustrating the links between indicator and latent variables and a structural model showing the relationships between latent variables.

Path coefficient and effect size (f^2) evaluations were part of the internal model quality assessments. Path coefficients ranged from -1 to +1, indicating the strength and direction of the correlations between the variables. The level of influence was evaluated using the effect size (f^2), computed using R^2 and the variance of endogenous variables. Cohen's results showed that values of 0.02, 0.15, and 0.35 indicate weak, medium, and large effects, respectively (25).

Assessment of the Measurement Models

Even though the questionnaires' validity and reliability have previously been assessed (19, 20), it is crucial to reevaluate the questionnaire's psychometric qualities from the perspectives of indicator reliability, internal consistency reliability, convergent validity, and discriminant validity (24-26). The correlations between each item and the construct were assessed by the given standards to determine the reliability of the indicators. The dependability of internal consistency was then evaluated using metrics like Cronbach's alpha, composite reliability (CR), and the recently developed Rho_A metric (27). Convergent validity was then evaluated using the average variance extracted (AVE) measure. Discriminant validity was assessed using the Heterotrait-Monotrait (HTMT) criterion using

evaluative criteria to determine the HTMT values (24, 28, 29).

Assessment of the Structural Model

A variety of factors were examined as part of the process of evaluating the study's structural model under the recommendations given, including the significance and relevance of path coefficients and indirect effects, the in-sample and out-of-sample predictive powers, and the f2 effect sizes of endogenous constructs (Table 3). While the variance inflation factor (VIF) values were first analyzed, collinearity was unaffected by any value less than 2 (Table 1). A 5% significance level and 10,000 subsamples were used in the percentile-bootstrapping test of the hypotheses. A summary of the findings is presented in Table 3.

Results

Assessment of the Measurement Models

According to Table 1, the evaluation showed valid loadings over 0.708, meaning that no non-contributory components needed to be removed. Results above 0.7 for reliability estimates and over 0.5 for AVE values were obtained from the computation of the AVE values and the one-tailed 95% percentile confidence intervals of the reliability statistics, indicating acceptable convergent validity and reliability (Table 1).

According to the evaluation, all HTMT values and their upper confidence intervals fell below 0.85, achieving the HTMT0.85 requirement and demonstrating acceptable discriminant validity (Table 2).

Assessment of the Structural Model

The study found empirical evidence for the H-final hypothesis, suggesting that the suggested conceptual model may predict SMI to an extent of 31.7% with an adequate R^2 value of 0.317. The Pittsburgh Sleep Quality Index, smoking with SMI, and the Depression Anxiety Stress Scale-21 were significantly and negatively correlated. On the other hand, favorable correlations between DASS21 and PSQI were discovered. According to the effect size (f^2), the smoke variable strongly correlated with the muscle mass index (Table 3). According to the structural model, there was a relationship (P=0.002) between the skeletal muscle mass index (SMI) and the Depression Anxiety Stress Scale-21 (DASS21). Furthermore, a statistical model study revealed a strong inverse association (P=0.000) between the Pittsburgh sleep quality score and the skeletal muscle mass index. Lastly, the statistical model's results for the current study demonstrated a more potent than previously significant negative relationship between the smoking variable and skeletal muscle mass index (P=0.000, F^2=0.45). Furthermore, a significant relationship was observed between sleep quality (PSQI) and mental health (DASS21) (P=0.000, F^2=0.161).

Table 1. Reliability Estimates, Convergent Validity Statistics, and Outer VIF Values.

	Alpha	rho_A	CR	AVE	VIF
DASS	1.000*	1.000*	1.000*	1.000*	1.000*
SMI	1.000*	1.000*	1.000*	1.000*	1.000*
PSQI	1.000*	1.000*	1.000*	1.000*	1.000*
Smoke	1.000*	1.000*	1.000*	1.000*	1.000*

*Loading>0.7
*Alpha>0.7
*rho_A>0.7
*CR>0.7
*AVE>0.5
*VIF<3
Depression Anxiety Stress Scales (DASS), Pittsburgh Sleep Quality Index (PSQI), Skeletal Muscle Mass Index (SMI), Cronbach's Alpha (ALPHA), Composite Reliability (CR), Average Variance Extracted (AVE)

Table 2. Discriminant Validity Based on HTMT.

	DASS	SMI	PSQI	Smoke
DASS				
SMI	0.058*			
PSQI	0.297*	0.072*		
Smoke	0.037*	0.204*	0.029*	

*HTMT<0.85
Epression Anxiety Stress Scales (DASS), Pittsburgh Sleep Quality Index (PSQI), Skeletal Muscle Mass Index (SMI)

Table 3. Structural Model Evaluation Results (final hypothesis).

	R^2	Path Coefficients (β)	F^2	T Statistics (|O/STDEV|)	P Values	CI95%	
						2.5%	97.5%
DASS -> SMI		-0.047	0.002	3.076	0.002*	-0.074	-0.013
DASS -> PSQI		0.297	0.161*	21.305	0.000**	0.270	0.325
DASS -> Smoke	0.317*	0.037	0.001	2.664	0.008*	0.009	0.062
PSQI -> SMI		-0.064	0.004	4.351	0.000**	-0.096	-0.037
Smoke -> SMI		-0.207	0.45**	14.375	0.000**	-0.235	-0.182

*R^2>0.2
*P-value<0.05
**P-value<0.001
*f^2>0.15
**f^2>0.35
. Depression Anxiety Stress Scales (DASS), Pittsburgh Sleep Quality Index (PSQI), Skeletal Muscle Mass Index (SMI)

Discussion & Conclusion

The fundamental objective of this study was to determine the relationship between muscular mass and the mental lifestyle of Mashhad University of Medical Sciences employees. Therefore, the Pittsburgh Sleep Quality Index, smoking, the Depression Anxiety Stress Scale-21, and the dependent variable, skeletal muscle mass index, were all evaluated. This study's main discovery showed how muscle mass (SMI) and intangible mental lifestyle factors relate.

The results showed a relationship between muscle mass and stress, anxiety, and depression ratings. According to the structural model, there is an average 4.7% drop in muscle mass (SMI) for every unit increase in the Depression Anxiety Stress Scale-21 (DASS21) score. Few research have looked at the relationship between mental health and muscle mass, even though numerous studies have looked at the effects of sarcopenia and muscle wasting on mental health (11, 30). Diabetes patients with a lower SMI score had a significantly lower Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) score, per a study by Low et al. (P = 0.030) (31).

The results of the current study showed that sleep quality (PSQI) was related to muscle mass in the structural model evaluating the relationship between intangible mental lifestyle elements and muscle mass. According to the structural model, there was an average 6.4% drop in muscle mass (SMI) for every unit increase in the Pittsburgh Sleep Quality Index (PSQI). Hayashi et al. discovered that a high PSQI total score significantly correlated with decreased muscle strength (P=0.027), even after confounding variables were considered (P=0.011). The adjusted analysis, however, did not find a significant relationship between the skeletal muscle mass and the PSQI total score (P=0.363). Relationships with PSQI subscores showed that muscle strength was significantly connected with sleep latency, sleep efficiency, and daytime dysfunction scores (9). Buchmann et al. indicated a significant (P<0.03) relationship between parameter adequacy, sleep quality, and muscle mass thickness. The study's findings support the hypothesis that sleep and muscle mass are related but call for more long-term research (32). There are multiple ways in which the quality of sleep affects muscle mass. Lack of sleep can negatively impact muscle mass because stress hormones affect strength and muscle protein turnover. Muscle-derived protein Muscular strength can be lost as a result of catabolism brought on by stress hormones generated during restless nights (15). Long-term stress can elevate cortisol levels, which may hinder the body's production of testosterone and other chemicals necessary for muscle building, thus impeding muscle growth and recovery. Muscle hypertrophy can be negatively impacted by persistent stress, low-grade systemic inflammation, and the ensuing hypercortisolism (10).

The results of the present investigation showed a relationship between muscle mass and smoking. Structural models indicate that muscle mass (SMI) declines by 20.7% for every unit increase in smoking. Xu showed a relationship between smoking and a person's loss of muscle mass (33). Additionally, Buchmann observes that regular smoking and the thickness of one's muscle mass are related (32). In addition, Atkins et al. demonstrated an association between smoking and muscle mass (MAMC and FFMI) (34). According to Rom et al., quitting smoking is associated with increased bone density, muscle mass, and muscle strength (35). Smoking

increases the potential for systemic inflammation and oxidative stress, which can lead to the atrophy and malfunctioning of muscles. Moreover, smoking has been related to a reduction in muscle protein synthesis and an increase in muscle protein breakdown, both of which are deleterious to muscle mass (10). Furthermore, smoking's vasoconstrictive effects may worsen muscular performance and metabolism by lowering the quantity of oxygen that reaches the muscles. Consequently, the negative effects of smoking on muscle hypertrophy were mediated by increased inflammation, reduced muscle protein turnover, and enhanced oxidative stress (10).

The final hypothesis's structural model has a high R^2, indicating significant validity. According to the current study, smoking, sleep quality, and mental health are significantly correlated with skeletal muscle mass.

Declarations

Strengths

The significant statistical population in this study makes it possible for various data ranges to be measured. Thus, this study can be received with good validity and reliability. The simultaneous evaluation and analysis of the investigated aspects (muscle mass and intangible mental lifestyle determinants) are drawn based on conceptual models using the Smart PLS software, which is the strength of this study because one of the positive features of this method is to eliminate the inflation of errors.

Limitations and Recommendations

Two of the study's weaknesses are personal recollection and reporting bias in the survey replies. The research team also decided to remove the alcohol use component from the study because of report bias, and more research is required in this field.

Authorship Contribution Statement

Here is the authorship contribution statement:

Sara Telikani: First Author, Main Author, and Data Analyst. Sara Telikani was responsible for designing the study, collecting and analyzing the data, and drafting the manuscript.

Dr. Seyyed Reza Sobhani: Supervisor. Dr. Sobhani supervised the study, providing guidance and oversight throughout the research process.

Dr. Jamshid Jamali: Statistical Supervisor. Dr. Jamali was responsible for designing and implementing the statistical analysis plan, ensuring the accuracy and reliability of the results.

Mohammad Masoumvand: Co-Analyzer. Mohammad Masoumvand assisted in the data analysis and contributed to interpreting the results.

Dr. Reza Rezvani: Article Supervisor. Dr. Rezvani reviewed and edited the manuscript, ensuring its clarity, accuracy, and overall quality.

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Declaration of Conflicting Interests

The authors declare no conflict of interest related to this manuscript.

Ethical Issues

The Mashhad University of Medical Sciences Ethics Committee (IR.MUMS.MEDICAL.RE.C.1401.585) authorized the procedures performed under its ethical guidelines. Before the data collection, the participants also signed an informed consent form, and PERSIAN Cohort protocols were adhered to for data confidentiality and anonymization.

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