

Associations Between Sleep Deprivation and Salivary Testosterone Levels in Male University Students: A Prospective Cohort Study

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Abstract

Sleep deprivation is a common health problem that is growing rapidly worldwide and it is associated with short- and long-term impacts on health. The aim of this study was to detect potential predictors of salivary testosterone (sT) association with sleep deprivation in Arab male university students. In this prospective cohort study, 77 university male students in the age range of 18 to 26 years were divided into two groups, sleep-deprived (SD) participants and non-sleep-deprived (NSD) participants. Sleep deprivation was defined as sleeping less than 5 hr per night. Blood samples and sT were collected from fasting participants to measure serum levels of glucose, lipid profile, leptin, serotonin, sT, and body mass index (BMI) values. The multiple linear correlation model of high-density lipoprotein cholesterol (HDL-C), BMI, and serotonin was positively correlated with sT ($r = .977, p < .05$) in the SD group. No correlations were identified with sT in the NSD group. In the SD study group, the multiple linear regression model of HDL-C, BMI, and serotonin was significantly influenced by sT ($R^2 = .955, p < .05$). These predictors together explained approximately 96% of the variance in sT levels in the SD study group. No predictive variables for sT were reported in the NSD group. Results indirectly confirmed the presence of a positive association between sT and sleep deprivation in young men. This association is mediated by three factors, HDL-C, BMI, and serum serotonin, which are collectively considered as part of a significant physiological adaptation to sleep deprivation in young men.

Keywords

salivary testosterone, sleep deprivation, BMI, leptin, lipid, serotonin

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Sleep pattern changes across the life span from infancy to old age and may additionally be modulated by sex, race/ethnicity, and mental or physical health conditions (Orzel-Gryglewska, 2010). Experimental sleep deprivation protocols conducted in healthy young adults led to profound functional changes in cognition and behavior (Hajali et al., 2015). Sleep deprivation is a changeable behavior and its consequences increase stress levels and accelerate biological aging (Choi et al., 2016). Human behavior including stress, physical activity, alcohol consumption, obesity, and sleep deprivation may influence serum testosterone (T) levels (Alvarenga, Hirotsu, Mazaro-Costa, Tufik, & Andersen, 2015). T is a sex steroid hormone that affects primarily male reproductive activities and has

anabolic effects on muscle mass formation (Reynolds et al., 2012). T is involved in the sleep–wake cycle and may modulate individual susceptibility to subjective symptoms of sleep deprivation. Low T may affect overall

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sleep quality and this is particularly proved by the decline in T levels among individuals with sleep deprivation. The paradox is that large doses of exogenous T are associated with abnormalities of sleep duration (Collomp et al., 2016). Salivary testosterone (sT) (Clifton et al., 2016; Crewther, Obminski, Orysiak, & Al-Dujaili, 2017) was measured in many previous studies to predict cardiovascular disease (CVD) correlations (Miller et al., 2010) as well as mental health (Dabbs Jr, 1993). It was concluded that circulatory androgen levels were affected by time and duration of sleep (Arnal et al., 2016). Although the majority of authors have confirmed an inverse correlation between sleep deprivation and T (Akerstedt, Palmblad, de la Torre, Marana, & Gillberg, 1980; Axelsson, Ingre, Akerstedt, & Holmback, 2005; Gonzalez-Santos, Gaja-Rodriguez, Alonso-Uriarte, Sojo-Aranda, & Cortes-Gallegos, 1989; Luboshitzky, Shen-Orr, & Herer, 2003; Luboshitzky, Zabari, Shen-Orr, Herer, & Lavie, 2001), the variability of T levels in different age groups remains poorly understood (Liu, Swerdloff, & Veldhuis, 2004; McLachlan & Allan, 2004; Stellato, Feldman, Hamdy, Horton, & McKinlay, 2000). Elevated T levels were reported in young men during partial sleep deprivation, after exercise (Abdelmalek et al., 2013). Another study (Wittert, 2014) identified that less T was secreted by middle-aged men at night compared to healthy young men. The effect of sleep deprivation on T levels may be age dependent (Maheshwari et al., 2017; Penev, 2007) rather than independent. Accordingly, large individual differences based on age (Axelsson et al., 2005) may reflect the variability in morning T levels (Penev, 2007). Changes of some CVD- and depression-associated predictors in sleep deprivation, such as lipid profile and serotonin, have been given more attention in recent studies (Giagulli, Guastamacchia, Licchelli, & Triggiani, 2016; Hylander & Lehtihet, 2015). Lipid profile changes are viewed as early potential predictors for stepwise progression of pathogenesis of atherosclerosis (Killick et al., 2015; Zimberg, Fernandes Junior, Crispim, Tufik, & De Mello, 2012). Serum serotonin predicts mood changes and psychiatric disorders including depression (Dallaspezia & Benedetti, 2015). Association studies of sleep deprivation with lipid profile or serum serotonin were conducted in human and animal models (Giagulli et al., 2016; Hylander & Lehtihet, 2015).

Using saliva as a biological fluid for analysis of steroid hormones, particularly androgen, in sports medicine, psychology, and stress research is increasing (Groschl, 2008). The main reason for this vast use is the current development in enzyme-linked immunoassays, which leads to simplicity, high sensitivity and low cross-reactivity, as well as reliability (Al-Dujaili & Sharp, 2012). sT levels represent the biologically active hormone and correlate with total blood T in healthy and clinical

populations (Al-Dujaili & Sharp, 2012; Groschl, 2008; Vittek, L'Hommedieu, Gordon, Rappaport, & Louis Southren, 1985).

The aim of the current study was to clarify the effect of sleep deprivation on sT by detecting some of the implemented potential predictors in young Arab men.

Materials and Methods

Study Design and Participants

The present work is a prospective cohort study carried out at the Applied Science Private University (ASU) in Amman, Jordan, during the period from January to April 2014. The protocol of this study was based on a chronic model of sleep deprivation (Tassi et al., 2012) according to participants' sleep behavior during the 3 months prior to the day of the study. To avoid some anticipated variations in the study sample, only Jordanian students who live in Amman city participated in the study. The participants filled out a questionnaire including anthropometric, clinical parameters, and sleep duration patterns. Healthy male nursing students in the age range of 18 to 26 years were invited to participate in this study and their samples were taken for analysis.

Healthy adults typically require 6 to 10 hr of sleep in a 24-hr day. The average need of sleep for healthy adults is around 8 hr daily (Bonnet, 2005). Adults who get less than 5 hr of sleep will demonstrate a decline in peak alertness and for that reason are considered sleep deprived (Carskadon & Dement, 1982). Based on this sleep duration pattern, which was confirmed through interviewing the participants and their roommates, participants were categorized into two groups, a sleep-deprived group (SD) and a non-sleep-deprived group (NSD; Figure 1).

Informed Consent and Health Screening

This study was performed using a protocol for the protection of human subjects approved by the ASU ethical committee under reference DRGS-2013/2014-8. Approval for all the conducted studies was obtained from ASU's ethics committee. All participants were provided with an information sheet, which contained details of the experimental protocol. Participants fully understood the purpose of the study as well as the risks involved. Participants were informed of being free to withdraw from the investigation at any stage. All participants provided written consent prior to the commencement of the study and were asked to complete a health screen questionnaire prior to their participation in the investigation. To avoid confounding factors known to affect leptin and sT levels, participants with diagnosed chronic diseases such as CVDs, diabetes, hepatic, renal, or endocrine disorders and those who had

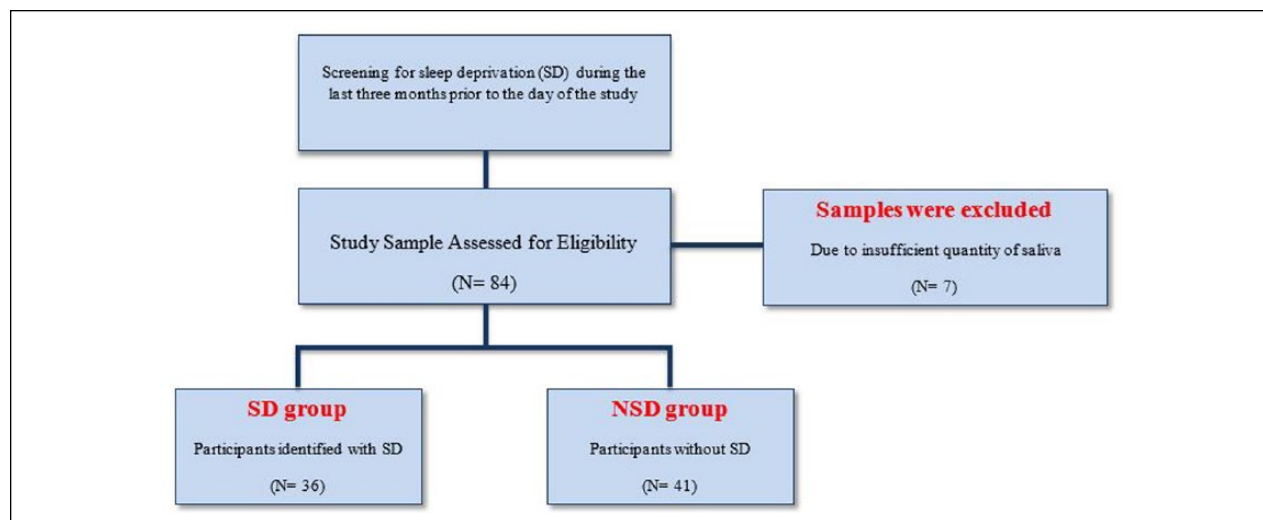


Figure 1. Schematic diagram of study design

Note. SD = sleep-deprived group; NSD = non-sleep deprived group; N = sample size.

been taking any kind of medication for the past two months prior to the study were excluded.

Salivary Testosterone

sT has been widely used as an indicator of the activity of the hypothalamic-pituitary-gonadal axis activity (Elloumi, Maso, Michaux, Robert, & Lac, 2003; Kraemer et al., 2001; Sallinen et al., 2004). sT was collected from the participants between 8 and 9 in the morning. To collect salivary samples, participants were provided with a *Salivette*[®] sampling device (cotton) along with both verbal and written instructions for usage. The instructions stated that participants were to collect saliva themselves. Participants were asked to drool passively through a straw into a tube, which was then kept on ice to precipitate mucins and then centrifuged ($10,000 \times g$, 15 min, 4°C). The supernatant (1 mL) was collected and stored at -20°C until assayed 2 weeks later. sT samples were measured using an enzyme-linked immunosorbent assay (ELISA, SLV-3013, DRG[®] International, Inc., NJ, USA) at Ibn Al-Haytham Hospital laboratories in Amman, Jordan. The range of the assay is between 1.9 and 5,000 pmol/L and the limits of detection of this assay is 1.9 pmol/L at a 95% confidence limit and the normal range for sT is between 21.2 and 801.2 pmol/L.

Serum Glucose and Lipid Profile

Fasting venous blood samples were obtained, centrifuged, and stored at -20°C until being assayed. Fasting blood glucose samples were collected at 8 a.m. Blood glucose was confirmed by using *One Touch*[®] test strips (LifeScan; Johnson & Johnson, Palmitas, CA, USA).

Triglycerides, total cholesterol, and high-density lipoprotein cholesterol (HDL-C) were determined using enzymatic colorimetric kits (Linear Chemicals, Barcelona, Spain). Low-density lipoprotein cholesterol (LDL-C) was calculated from the equation recorded in a previous study (Friedewald, Levy, & Fredrickson, 1972).

Serum Leptin and Serotonin

Fasting serum leptin and serotonin samples were liquated and stored in polypropylene vials at -20°C until being analyzed 2 weeks later at Al-Khalidi Hospital and Medical Centre Laboratories in Amman, Jordan. Commercial serotonin enzyme immunoassay (EIA) kits were obtained for quantitative determination of serum serotonin (Labor Diagnostika Nord GmbH & Co, KG, Nordhorn, Germany). Analytical sensitivity, as reported by the manufacturer, was 5 ng/mL. Leptin samples were assayed with an EIA kit (DRG Diagnostics, Marburg, Germany) with analytical sensitivity of about 1.0 ng/mL, according to the manufacturer.

Body Mass Index

On the evaluation day, height (cm) and weight (kg) of participants were recorded. Then, body mass index (BMI, kg/m^2) was calculated accordingly.

Statistical Analysis

Statistical analysis was performed using a statistical software package SPSS, version 19.0 for Windows (Chicago, IL, USA). *T*-test statistical analysis was used to compare the differences of demographic and clinical findings

Table 1. Characteristics of the Two Study Groups Subdivided by Their Sleep Deprivation (Mean \pm Standard Deviation).

Variable	SD		NSD		p value
	Mean	Standard deviation	Mean	Standard deviation	
Age (years)	22.39	1.93	21.60	1.49	.251
sT (pmol/L)	274.7	164.8	235.8	131.48	.254
FBG (mg/dL)	86.68	8.96	86.92	7.43	.872
Body weight (kg)	77.50	14.61	80.96	15.38	.243
BMI (kg/m ²)	25.16	4.45	26.16	4.43	.253
Leptin (ng/mL)	9.43	8.87	11.38	9.92	.270
TG (mg/dL)	123.6	52.8	134.68	58.73	.321
Total cholesterol (mg/dL)	176.05	34.05	176.87	32.71	.902
HDL-C (mg/dL)	50.11	8.66	51.25	8.14	.506
LDL-C (mg/dL)	105.94	35.38	100.29	29.27	.387
Serum serotonin (ng/mL)	241.14	94.96	268.22	97.73	.142

Note. BMI = body mass index; FBG = fasting blood glucose; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; NSD = non-sleep-deprived; SD = sleep-deprived; sT = salivary testosterone; TG = triglycerides.

between the means of the two studied groups. The Pearson analysis was used to find if there was any correlation between the participants' characteristics and serum leptin levels. Stepwise multiple regressions and univariate analysis were used to evaluate the effects of independent variables (IDVs), serum lipid profile, BMI, and serotonin, on sT levels as a dependent variable (DV) in SD and NSD groups.

Results

Study Design and Participants

One hundred and twenty-two healthy male nursing students in the age range of 18 to 26 years were invited to participate in this study. Samples were taken from only 84 participants who committed to follow the study protocol as presented in Figure 1. Seven samples were excluded due to insufficient quantity of saliva. Participants were categorized into two groups, a SD group ($n = 36$, 45.4%) and a NSD group ($n = 41$, 54.6%).

Descriptive Characteristics

As reported in Table 1, *t*-test was used to study the difference in all variables between the SD and NSD groups. No significant differences were noted between the SD and NSD groups for all variables. However, the comparison identified higher levels of sT in the SD group (274.7 ± 164.8) compared with NSD group (235.8 ± 131.5) without significant difference in the mean ($p > .05$). The mean age for SD participants was $22.38 (\pm 1.9)$ years, whereas it was $21.6 (\pm 1.5)$ years for NSD participants. Values for mean BMI were higher in the NSD group compared with SD group (26.16 ± 4.4 vs. 25.16 ± 4.5). The mean obesity

marker, serum leptin, was higher in the NSD group (11.38 ± 9.9) versus (9.4 ± 8.7) in the SD group. These results may predict the effect of obesity on BMI differences in the two study groups.

The Univariate Analysis

Univariate analysis was used to study the association between all variables and sT. As reported in Tables 2 and 3, there were no significant univariate associations between any of the study variables and sT in both the SD and NSD groups.

Correlations of sT in SD Group

Table 2 presents sT correlation coefficients for three selected variables in the SD study group. The multiple linear correlation model of HDL-C, BMI, and serum serotonin is significantly and positively correlated with sT ($r = .977$, $p < .05$). No correlations were reported between HDL-C, BMI, serum serotonin, and sT in the NSD group.

Stepwise Regression Analysis

Stepwise regression analysis was performed to investigate which IDVs accounted for these associations with sT changes. In the SD study group, the multiple linear regression model of HDL-C, BMI, and serum serotonin was significantly influenced by sT ($R^2 = .955$, $p < .05$). These predictors together explained approximately 96% of the variance in sT levels in the SD study group. No predictive variables for sT were identified in the NSD group (Table 3).

Table 2. The Univariate Association Between Factors and salivary testosterone in the Sleep-Deprived Group Using Simple Linear Regression.

Univariate effects estimates	Coefficients						
	N	B	LCI	UCI	R	R ²	p value
Serum serotonin (ng/mL)	36	0.431	-0.115	0.976	.269	.073	.118
Salivary morning cortisol (nmol/L)	36	6.831	-6.092	19.754	.200	.040	.288
Salivary night cortisol (nmol/L)	36	15.216	-3.475	33.908	.306	.94	.106
Salivary morning dehydroepiandrosterone (nmol/L)	36	0.922	-2.907	4.750	.095	.009	.625
Salivary night dehydroepiandrosterone (nmol/L)	36	0.020	-0.134	0.174	.051	.003	.791
FBG (mg/dL)	36	-1.012	-8.094	6.070	.050	.002	.773
Leptin (ng/mL)	36	3.145	-4.757	11.048	.140	.019	.424
Vitamin B12 (pg/mL)	36	-0.259	-0.795	0.276	.173	.030	.331
Folic acid (ng/mL)	36	-6.032	-22.333	10.269	.132	.017	.457
TG (mg/dL)	36	0.064	-0.875	1.002	.025	.001	.891
Total cholesterol (mg/dL)	36	-0.374	-2.312	1.563	.072	.005	.696
HDL-C (mg/dL)	36	5.415	-3.219	14.049	.236	.056	.209
LDL-C (mg/dL)	36	-0.493	-2.357	1.371	.098	.010	.593
BMI (kg/m ²)	36	42.135	-31.392	115.662	.196	.038	.252
Body weight (kg)	36	1.793	-2.196	5.783	.165	.027	.366
Height (cm)	36	-3.409	-13.293	4.476	.128	.016	.487
WBC (10 ⁹ /L)	36	13.849	-24.616	52.315	.138	.019	.467
RBC (×10 ¹² /L)	36	-55.003	-128.579	18.573	.300	.090	.136
Hemoglobin (g/dL)	36	3.951	-39.056	46.958	.032	.001	.853
PCV (%)	36	0.266	-11.729	12.260	.008	.000	.964
MCV (fL)	36	2.446	-7.636	12.528	.084	.007	.625
MCH (pg)	36	12.407	-3.773	28.587	.307	.094	.127
MCHC (g/dL)	36	11.575	-9.734	32.883	.223	.050	.273
Lymphocytes number (×10 ⁹ /L)	36	-0.352	-12.531	11.828	.012	.000	.953
Monocytes (%)	36	-14.376	-53.936	25.184	.159	.025	.459
Granulocytes number (×10 ⁹ /L)	36	2.044	-8.389	12.476	.086	.007	.688
Platelets (×10 ⁹ /L)	36	-0.167	-0.981	0.647	.095	.009	.674

Note. B = slope; BMI = body mass index; FBG = fasting blood glucose; HDL-C = high-density lipoprotein cholesterol; LCI = lower 95% confidence interval; UCI = upper 95% confidence interval; LDL-C = low-density lipoprotein cholesterol; MCH = mean cell hemoglobin; MCHC = mean cell hemoglobin concentration; MCV = mean cell volume; N = sample size; PCV = packed cell volume; R = Pearson linear correlation coefficient; R² = determinant coefficient; RBC = red blood cell; TG = triglycerides; WBC = white blood cell.

Discussion

Most of the studies on the association of salivary or serum T with sleep deprivation were conducted on the elderly (Akerstedt et al., 1980; Axelsson et al., 2005; Jauch-Chara, Schmid, Hallschmid, Oltmanns, & Schultes, 2013). Few reports are available so far on sT in healthy young or adolescent males (Clifton et al., 2016; Crewther et al., 2017). The current results reported an increase of sT in SD men compared with an NSD group. In addition, elevated sT in SD participants was significantly positively correlated with HDL-C, BMI, and serum serotonin levels. The effects of T on lipid profile, particularly for HDL-C, are modest and variable (Canoy et al., 2014; Cassimatis, Crim, & Wenger, 2016; Md, Brinton, & Grunfeld, 2017). An inverse association was observed between serum lipid profile and T levels except for

HDL-C (Chillaron et al., 2016; Md et al., 2017). The latter association was positive when adjusted for adiposity. Nevertheless, HDL-C was no longer significant (Canoy et al., 2014). Lu et al. (2016) have noted a positive correlation between obesity-associated markers and age with elevated lipid profile parameters, except for HDL-C. This is consistent with recent studies, which have reported that low T is associated with accelerated atherosclerosis (Gosai et al., 2016).

Further, one quarter of men with coronary artery disease (CAD) were biochemically hypogonadal according to a previous study by Gosai et al. (2016). Accordingly, a preventive potential of T during the progressive mechanisms of CVD was acknowledged (Yeap, 2015). The variations of T predictors such as LDL and HDL in previous associated studies were mostly age dependent (Frederiksen, Hojlund, Hougaard, Brixen, & Andersen,

Table 3. The Univariate Association Between Factors and Salivary Testosterone in Non-Sleep-Deprived Group Using Simple Liner Regression.

Univariate effects estimates	Coefficients						
	N	B	LCI	UCI	R	R ²	p value
Serum serotonin (ng/mL)	41	0.178	-0.294	0.651	.123	.015	.450
Salivary morning cortisol (nmol/L)	41	10.008	-2.269	22.285	.273	.075	.107
Salivary night cortisol (nmol/L)	41	19.614	-9.913	49.141	.245	.060	.185
Salivary morning dehydroepiandrosterone (nmol/L)	41	35.726	-38.076	109.528	.166	.028	.332
Salivary night dehydroepiandrosterone (nmol/L)	41	-0.016	-0.128	0.096	.053	.003	.777
FBG (mg/dL)	41	1.874	-3.523	7.271	.112	.012	.487
Leptin (ng/mL)	41	-.822	-4.834	3.191	.066	.004	.681
Vitamin B12 (pg/mL)	41	0.015	-0.355	0.385	.013	.000	.936
Folic acid (ng/mL)	41	-6.533	-20.008	6.942	.159	.025	.332
TG (mg/dL)	41	0.413	-0.322	1.148	.192	.037	.261
Total cholesterol (mg/dL)	41	0.076	-1.485	1.637	.017	.000	.921
HDL-C (mg/dL)	41	-0.305	-6.335	5.724	.018	.000	.919
LDL-C (mg/dL)	41	0.122	-1.565	1.809	.025	.001	.884
BMI (kg/m ²)	41	-13.473	-63.791	-63.791	.086	.007	.591
Body weight (kg)	41	-0.969	-3.775	1.836	.118	.014	.488
Height (cm)	41	-4.531	-10.929	1.867	.236	.056	.159
WBC (10 ⁹ /L)	41	-18.782	-44.761	7.196	.241	.058	.151
RBC (×10 ¹² /L)	41	13.712	-53.757	81.181	.071	.005	.682
Hemoglobin (g/dL)	41	-1.046	-30.277	28.186	.012	.000	.943
PCV (%)	41	-0.164	-7.941	7.612	.007	.000	.966
MCV (fL)	41	1.856	-8.159	11.870	.060	.004	.710
MCH (pg)	41	0.595	-20.069	21.259	.010	.000	.954
MCHC (g/dL)	41	-4.872	-27.788	18.044	.074	.005	.668
Lymphocytes number (×10 ⁹ /L)	41	1.031	-4.558	6.621	.066	.004	.710
Monocytes (%)	41	9.514	-12.045	31.072	.162	.026	.375
Granulocytes number (×10 ⁹ /L)	41	-0.310	-5.105	4.485	.024	.001	.896
Platelets (×10 ⁹ /L)	41	-0.022	-0.852	0.808	.010	.000	.957

Note. B = slope; BMI = body mass index; FBG = fasting blood glucose; HDL-C = high-density lipoprotein cholesterol; LCI = lower 95% confidence interval; UCI = upper 95% confidence interval; LDL-C = low-density lipoprotein cholesterol; MCH = mean cell hemoglobin; MCHC = mean cell hemoglobin concentration; MCV = mean cell volume; N = sample size; PCV = packed cell volume; R = Pearson linear correlation coefficient; R² = determinant coefficient; RBC = red blood cell; sT = salivary testosterone, TG = triglycerides; WBC = white blood cell.

Table 4. The Multivariate Association Between Factors and Salivary Testosterone in Sleep-Deprived Group by Using Multiple Liner Regression (Stepwise Method).

Univariate effects estimates	Coefficients						
	N	B	LCI	UCI	R	R ²	p value
HDL-C (mg/dL)	36	14.356	9.944	18.769	.773	.597	.009
BMI (kg/m ²)	36	78.767	43.316	114.219	.896	.802	.003
Serum serotonin (ng/mL)	36	0.730	0.334	1.126	.977	.955	<.00

Note. LCI = lower 95% confidence interval; UCI = upper 95% confidence interval; N = sample size; B = slope; R = Pearson linear correlation coefficient; R² = determinant coefficient.

2012; Haymana et al., 2017; Maheshwari et al., 2017). In this context, the current findings can be considered as evident during young age. Inconsistent results were identified due to therapy with high doses of T (Md et al., 2017) or due to the fact that they were case studies (Hylander & Lehtihet, 2015).

It is well known that T is one of the anabolic hormones that mediates regulation of skeletal muscle protein balance (Rossetti, Steiner, & Gordon, 2017) and increases muscle mass (Haymana et al., 2017). A particular problem with BMI, as an index of obesity, is that it does not differentiate between body lean mass and body fat mass,

whereby a person can have a high BMI but still have a very low fat mass and vice versa (Ljungvall, Gerdtham, & Lindblad, 2015; Nuttall, 2015). For instance, Holliday, Gupta, and Vibhute (2016) considered that BMI is a reliable predictor of subcutaneous fat thickness. To evaluate this dilemma, serum levels of leptin were compared as an obesity predictor. Serum leptin levels were higher in the NSD group in comparison to the SD group. Comparable results in previous studies have reported a significant decrease in total T serum levels in obese young men compared to men with normal BMI (Jastrzebska et al., 2014).

Frederiksen et al. (2012) demonstrated similar T effects on muscle mass in aging men. These findings confirmed anabolic effects of T on muscle mass rather than obesity.

On the other hand, a few previous studies have mentioned that sleep deprivation was negatively associated with serum serotonin in older people (Roman, Hagewoud, Luiten, & Meerlo, 2006). In young men, the results were ambiguous. Partial similarity with this study's results about the association of elevated T with serum serotonin in the SD group was observed in animal models. Although elevated serum serotonin levels were noted in sleep deprivation-induced rats (Yang, Wu, Wu, Lin, & Tsai, 2015), luteinizing hormone (LH) secretion was inhibited. Lopez-Rodriguez, Wilson, Maidment, Poland, and Engel Jr. (2003) reported that sleep deprivation increased extracellular serotonin in the rat hippocampus. These findings suggested that decreased serum levels in SD rats may be mediated by a 5-HT-related mechanism (Wu et al., 2011).

To our knowledge, there are no clinical reports supporting this potential mechanism. Cassimatis et al. (2016) and Gettler and Oka (2016) concluded that men with low T are at greater risk of depression or cognitive impairment functions (Giagulli et al., 2016). This study's results indirectly confirmed these findings, where the association between sT and serum serotonin was positive in SD young men. These two factors could be independent of each other. However, decline of serum serotonin and T with advanced age (Wang et al., 2014) are well known as early predictors for depression and CVD.

Finally, there are some limitations to this study, which can be summarized in the following three points. First, the sT sample size (= number of measurements in a sample) was performed only once due to limited funding. Second, only sleep duration was obtained and no other questions detailing sleep quality were raised. Third, there was a lack of data collection of time interval between participants' waking up and sampling. However, throughout the entire study period, the authors made the necessary arrangements for participants to be present for sampling at 7 a.m.

Taken together, these observations conclude that positive correlations of sT with HDL-C and serum serotonin predict significant physiological adaptation to sleep deprivation in young men.

Declaration of Conflicting Interests

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