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The Isokinetic and Electromyographic Assessment of Knee Muscles Strength in the Short- and Long-Term Type 2 Diabetes

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Abstract

Background: Type 2 diabetes (T2DM) patients are subject to muscle weakness.
Objectives: The aim of the study was an assessment of electromyographic (EMG) activity of knee muscles during isometric maximal voluntary contraction in the different disease durations of T2DM.
Methods: Eighteen patients with less than 10 years and twelve patients with more than 10 years of T2DM were compared with nineteen matched healthy control subjects. EMG of flexor and extensor muscles of knee concurrently with isometric maximal peak torque of knee flexion and extension at 75 degrees of knee flexion were recorded in three groups.
Results: Isometric maximal peak torque of extension and root mean squared (RMS) of vastus lateralis and medial hamstring in the healthy control was significantly higher than both patient groups. Whenever the maximal isometric peak flexion torque was not significantly different between groups, the mean power frequency (MPF) of flexor muscles especially medial hamstrings were higher in the short-term T2DM than healthy control groups. The two factors, gender and age, had significant effect on maximal peak torque and RMS of knee muscles.
Conclusions: EMG could show the effect of T2DM, gender and age on knee muscles activity. It seems that the medial hamstring was the most sensitive muscle of knee compartment to show the effect of T2DM and difference of short and long-term T2DM in EMG study.

Keywords: Type 2 Diabetes Mellitus, Isometric Maximal Peak Torque, Electromyography, Knee

1. Background

Several studies have demonstrated that type 2 diabetes (T2DM) causes poor muscle quality and strength especially in the lower limb (1-6). Strength reduction has been associated positively with increase of neuropathy, glycated hemoglobin A1c (HbA1c) and duration of diabetes (1, 2, 4, 7). Muscular weakness in T2DM could be due to insulin resistance, intracellular lipid accumulation, mitochondria dysfunction, sarcopenia and neuromuscular impairments (8-15). The electromyographic (EMG) assessment of muscle as a sarcolemma output provides more information about the properties of motor units to control neuromuscular function (16). EMG frequency, an indicator of muscle fiber conduction velocity, and EMG amplitude, a sign of neural excitation (17).

Rare studies assessed the EMG properties of diabetic muscles (7, 18, 19). The most electrophysiological assessments in T2DM were limited to measure nerve conduction velocity (20) and compound muscle action potentials that showed reduction in nerve conduction velocity and density of fibers and motor unit in the diabetic polyneuropathy (6, 15, 16). Watanabe et al. compared the root mean squared (RMS) activity of vastus lateralis in 10% MVC for 120 seconds between healthy and T2DM by 64 multi-channel EMG. They found that the modified entropy as a sign of heterogeneity of muscle was more in T2DM than healthy subjects. The results also indicated that synchronization of motor units significantly decreased in T2DM during low-level sustain contraction (19). The anterior tibialis and gastrocnemius activated later during stance phase of gait in the diabetic patients than healthy subjects. The study concluded when diabetic patients faced a new difficult situation that needed a higher muscle performance, the necessary range of motion and neuromuscular control around knee and ankle joints were insufficient (21).

Whereas exercise appears to play an important role in

controlling the diabetes, the study of muscle activity can be helpful to design a better strength exercise protocol (22). Many studies have been previously reported on the relationship between the surface EMG amplitude and muscle force. Then surface EMG is used to quantify muscle activity to improve exercise protocol design (23).

2. Objectives

To clarify the changes of muscle strength due to diabetes, the present study aims to assess the kinesiological EMG concurrently with torque of knee flexors and extensors during isometric maximal voluntary contraction in patients with less or more than 10 years of T2DM compared to healthy subjects that were matched with patients in terms of sex, body mass index (BMI), physical activity index (PAI) and ankle brachial index (ABI). The effect of gender and age were considered in comparison too. Previously it was demonstrated that these factors affected the muscle strength (5, 24-27).

3. Methods

3.1. Subjects

Thirty T2DM patients between 25 - 70 years-old were referred by internal specialists from medicine and endocrine clinics. They did not have severe or uncontrolled cardiac disease, intermittent claudication of leg and ulcers of feet, myopathy or rheumatoid arthritis. The ABI was between 0.9 - 1.3. Patients according to the duration of diagnosed diabetes were categorized into two groups. Eighteen patients that their diabetes were diagnosed less than ten years. They were placed in the short-term T2DM group. Twelve patients with equal or more than ten years of diabetes were placed in the long-term T2DM group. The healthy control group were twenty subjects who were matched with both diabetic groups in terms of sex, BMI, ABI and PAI. The added inclusion criterion for the healthy control group was that their HbA1c was less than 6%. All participants signed the written informed consent and the protocol was approved by the medical ethics committee of the Tarbiat Modares University.

3.2. Procedure

Firstly HbA1c and fasting blood sugar (FBS) were measured from blood sampling. Then ABI were measured by Doppler ultrasound from the dominant leg in the supine position. Those who entered the study had an ABI between 0.91 - 1.3 (28). The level of physical activity was measured by scoring of PAI. The method of PAI measurement has been explained in previous papers (7). The score has been categorized into the sedentary, poor and fair physical activity groups (29). The patients did not stop the prescribed diabetic medications. The tests were done in the afternoons. The finger blood glucose and brachial pressure were measured before the test.

3.3. Isometric Recording

Isometric maximum peak torque (IMPT) [Newton meter] of knee extensors and flexors was performed with an isokinetic dynamometer (HUMAC NORM, USA). The dominant leg which determined as the preferred leg for kicking a ball was tested. All subjects were instructed about the procedure of study and performed a warm-up and training trials. The adjustment of isokinetic machine and positioning of subjects for the test were explained previously (30). They were asked to push the fixed lever arm maximally to extension and then to flexion for three times at 75 degrees of knee flexion (30), each trial was done for 3 seconds and a rest period of 30 seconds was given between consecutive contractions. The trial which had maximal value of three trials was considered as IMPT. All IMPT were divided by weight for normalization.

3.4. EMG Recording and Analysis

Surface EMG signals concurrently with the isokinetic test were recorded from the vastus lateralis, vastus medialis, long head of biceps femoris and medial hamstring muscles of dominant leg. The electrodes were positioned according to surface EMG for non-invasive assessment of muscles' (SENIAM's) recommendations (31). The skin was first shaved and then cleaned with an 70% alcohol solution and two recording circle Ag/AgCl sensors (Telectrode, Bio Protech Inc., Korea, Diameter of 23 mm) for each muscle placed 20 mm apart (center to center distance) on the skin. A reference electrode was attached to the ipsilateral styloid process of wrist. A bipolar multi-channel EMG amplifier (Bio-Signal Pack, Bayamed Co.www.bayamed.com) (CMMR: 120 dB, Input Impedance: 10M Ω, bandwidth 200 KHz, gain:1000) was used to register the surface EMG activity. The signals were sampled with a frequency of 2460 Hz and analogue-to-digital converted and stored with 12-bit computer. LabVIEW (version 10.0.0, National Instruments) programming software was used to perform the signal processing.

Then the sampled EMG signals were digitally band-pass filtered from 10 to 500 Hz. The method of calculation the mean power frequency (MPF) and root mean square (RMS) (μV) were explained in the previous paper (7).

3.5. Statistical Analysis

Chi-square was used to assess differences of distribution of gender and PAI between groups. The ANOVA procedure was used to determine the baseline differences of age, weight, length, BMI, ABI, HbA1c, FBS and blood glucose test between groups. Test of normality and homogeneity of variances were performed for all variables. Two-way ANCOVA was used to compare the normalized IMPT, RMS in the three groups and two genders while the age was considered as covariate. Two-way ANOVA was used for MPF in different genders and groups and no covariance variable was proved. Bonferroni post hoc was used to follow up pairwise comparison. P-value less than 0.05 was considered as significantly. The SPSS software version 21 was used for analysis.

4. Results

We included 12 long-term T2DM and 18 short-term T2DM patients that were matched with 20 healthy control subjects. Demographic and blood parameters are presented in Table 1, diabetic subjects with good and moderate blood glucose control were included in this study. The three groups were matched according to sex, BMI, PAI and ABI. The long-term T2DM group was 10 years older than the healthy control group ($P = 0.02$). HbA1c, FBS and glucose tests of the two diabetic groups were higher than the healthy control group ($P < 0.000$), while the two diabetic groups were not significantly different.

Table 2 showed the extension IMPT and MPF and RMS of the VL and VM muscles. Table 3 showed the flexion IMPT and MPF and RMS of The BF and MH muscles. Both extension (EXT) and flexion (FLEX) IMPT and related RMS of women were significantly lower than men. The EXT IMPT of both diabetic groups were lower than that of healthy control group. The RMS of VL and MH were significantly lower in both diabetic groups than healthy controls. The MPF of both extensor muscles did not differ between groups or genders. However the MPF of MH muscle of short-term T2DM group was significantly more than healthy control group while only the MPF of BF muscle of both diabetic women was more than healthy women.

5. Discussion

As the isometric maximal peak torques of healthy controls was more than both T2DM groups, the RMS of agonist muscles, especially VL and MH of healthy control group was higher than both diabetic groups. The MPF of flexor muscles showed significantly more value in patients. EMG recording in T2DM showed that MH was more sensitive to effects of diabetes than other knee muscles. Because the diabetic medial hamstring, a small and single-joint muscle, had significantly lower RMS and slightly higher MPF than the healthy control group especially in the short-term T2DM.

The previous studies indicated that the main confounding factor in the strength such as age, sex, BMI and fitness should be controlled to make reliable comparison (7, 30). This study carefully controlled them. The results of the study were consistent with those of previous studies which found the T2DM had weaker muscles than matched healthy subjects in the lower limb (1, 4-7, 30). Our findings also showed that peak torque and RMS followed the same pattern of change in 100% maximal voluntary contraction. Gabriel et al. showed that the RMS in according force increases but median frequency slightly decreases at 100% MVC. They showed an increase in synchronization of motor units to be the source of these changes (32, 33). Moreover frequency- domain features are sensitive to variations in shape and peak to peak activation and synchronization of motor units (34). Then it might be suggested that the reason for lower RMS and higher MPF of weaker diabetic muscle in comparison to healthy muscle was low ability of motor units to produce synchronization around 100% MVC. It is noticeable that the behavior of mean frequency and median frequency is always similar, and they are two kinds of averages in statistics (35).

The decrease in muscle strength in T2DM is a result of muscle quality decline. It is dependent on loss of muscle fibers especially type 1 and an increase in intermuscular lipids, decrease in oxidative enzymes and secondary increase in glycolytic muscle enzymes that produced more metabolic residuals (9, 11, 36-39). Then the muscle fibers of diabetic muscle in the long term shift to type 2 because of loss of muscular capillary and oxidative enzymes (11, 40). These changes affect the quality of EMG (41).

The hamstring in comparison with quadriceps has a higher proportion of type 2 muscle fibers (42, 43). It is well known that the axon of fast motor units is thicker and has more conduction velocity (44). Therefore, it seems that higher MPF of knee flexors was associated with the existence of more fast motor units in addition to the effect of diabetes on muscle. To confirm that, the current study also showed that knee flexors had higher MPF than knee extensors although we did not analyze it. In contrast to the anticipation that MPF of women is less than that of men, the MPF of flexor muscles especially BF of short-term diabetic women was shown to be significantly more than that of men of the same group and others.

The limitations of the study included the selection of a nonspecific position to test knee flexors as well as lack of measurement of neuropathy intensity in T2DM patients.

Table 1. Demographic and Blood Characters of the Three Groups and the P Value[a]

	Healthy Control (N = 20)	Short-Term T2DM (N = 18)	Long-Term T2DM (N = 12)	P Value
Number of cases (women/men)	20 (10 / 10)	18 (9 / 9)	12 (6 / 6)	1
Age (years)	49.55 ± 10	52.11 ± 9.2	59.17 ± 7.1	0.02[b]
Weight (Kg)	73.68 ± 7.7	77.61 ± 12.5	77.72 ± 12.4	0.47
Height (cm)	167.89 ± 9.2	164.33 ± 8.3	166.09 ± 11	0.51
BMI	26.25 ± 3	28.71 ± 4.1	28.54 ± 3.6	0.09
Duration of diabetes (years)		4.8 ± 2	15.5 ± 7	
Medication (insulin + drugs / drugs) (N of cases)		1 / 17	3 / 9	
PAI (sedentary/poor/fair) (%)	63.2, 26.3, 10.5	66.7, 27.8, 5.6	45.5, 54.5,0	0.83
ABI	1.17 ± 0.08	1.22 ± 0.09	1.14 ± 0.14	0.43
HbA1c (%)	4.7 ± 0.8	7.02 ± 1.5	7.3 ± 1.4	0.000[c]
FBS (mmol/L)	91 ± 12.6	141.65 ± 36.9	160.91 ± 31.4	0.000[c]
Blood sugar (mmol/L)	114.05 ± 17.7	167.39 ± 44.23	205.20 ± 84.24	0.000[c]

[a] Data is mean ± SD.
[b] Post Hoc between health and long-term T2DM groups.
[c] Post Hoc between healthy control and long-term T2DM and short-term T2DM groups. FBS: fasting blood sugar.

Table 2. The mean (lower and upper level of 95% confidence interval) of males and females in three groups of IMPT of extension and EMG variables of vastus lateralis and medialis are described. The P value of two-way ANCOVA for IMPT and RMS of muscles and two-way ANOVA for MPF of muscles between three groups, two gender and interaction effect of them are in the last three columns.[a]

	Gender	Healthy Control (N = 20)	Short-Term T2DM (N = 18)	Long-Term T2DM (N = 12)	P Value		
					Groups	Gender	Gender × Groups
IMPT of extension	Male	3.01 (2.5-3.5)	2.4 (1.8-2.8)	2 (1.7-2.4)	0.018[b]	0.000	0.57
	Female	1.9 (1.5-2.3)	1.7 (1.1-2.1)	1.1 (1-1.2)			
MPF of vastus lateralis	Male	60.59 (55- 65)	64.2 (54-81))	68.6 (58-78)	0.47	0.97	0.92
	Female	60.2 (54-66)	65.6 (56-77)	68.1 (55-79)			
MPF of vastus medialis	Male	67.2 (60-75)	68.3 (59-76)	69.4 (65-73)	0.69	0.33	0.50
	Female	73.5 (67-81)	76.1 (67-86)	66.9 (56-72)			
RMS of vastus lateralis	Male	328 (280-382)	260.5 (196-336)	234.7 (146-332)	0.017[b]	0.001	0.82
	Female	231 (160-315)	142.1 (70-228)	85.2 (64-106)			
RMS of vastus medialis	Male	196.5 (148-250)	184.6 (119-270)	149.1 (116-195)	0.36	0.000	0.81
	Female	98.6 (69-119)	79.1 (46-110)	85.1 (50-129)			

[a] Post Hoc was significant between healthy control and short-term T2DM. IMPT: normalized isometric maximal peak torque, RMS: root mean square, MPF: mean power frequency.
[b] Post Hoc was significant between healthy control and both diabetic groups.

Not matching the age between groups and small sample size were other limitations.

This study concluded that both short and long-term T2DM patients 1had lower knee IMPT extension and VL and MH RMS than healthy control group. No significant difference was seen between the two diabetic groups in strength features such as maximal torque or RMS contrast endurance features that were shown previously (30, 45). MPF of MH muscle showed significant increase in the short-term T2DM in comparison with the healthy control group. It may be worth noting that the MH muscle was the most sensitive muscle to show the effect of diabetes in the

Table 3. The mean (lower and upper level of 95% confidence interval) of males and females in three groups of IMPT of flexion and EMG variables of biceps femoris and medial hamstring are described. The P value of two-way ANCOVA for IMPT and RMS of muscles and two-way ANOVA for MPF of muscles between three groups, two gender and interaction effect of them are in the last three columns.

	Gender	Healthy Control (N = 20)	Short-Term T2DM (N = 18)	Long-Term T2DM (N = 12)	P Value		
					Groups	Gender	Gender × Groups
IMPT of flexion	Male	0.98 (0.7-1.1)	0.79 (.7-.8)	0.84 (.6-.9)	0.12	0.000	0.37
	Female	0.5 (.3-.6)	0.49 (.3-.6)	0.39 (.3-.4)			
MPF of biceps femoris	Male	96.6 (92-101)	106.2 (93-119)	129.8 (103-166)	0.000	0.016	0.003[a]
	Female	103 (93-111)	153.4 (139-167)	128 (113-143)			
MPF of medial hamstring	Male	90.4 (77-106)	111.8 (83-143)	106.7 (92-120)	0.031[b]	0.81	0.92
	Female	90.3 (79-105)	122.6 (110-134)	102.6 (88-117)			
RMS of biceps femoris	Male	113.5 (82-142)	102.7 (76-130)	121.4 (65-196)	0.43	0.001	0.94
	Female	77 (60-93)	64 (33-104)	68.7 (57-79)			
RMS of medial hamstring	Male	225.4 (170-285)	175.7 (136-224)	163.2 (106-209)	0.016[a]	0.000	0.78
	Female	152 (102-208)	94.7 (52-151)	91.3 (55-119)			

[a] Post Hoc was significant between healthy control and both diabetic groups.
[b] Post Hoc was significant between healthy control and short-term T2DM. IMPT: normalized isometric maximal peak torque, RMS: root mean square, MPF: mean power frequency.

knee region.

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Footnotes

Authors' Contribution: Boshra Hatef contributed to all parts of study and manuscript preparation; Others contributed to data analysis and edition and preparation of manuscript.

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References

1. Andersen H, Nielsen S, Mogensen CE, Jakobsen J. Muscle strength in type 2 diabetes. *Diabetes.* 2004;**53**(6):1543–8. [PubMed: 15161759].
2. Andersen H, Poulsen PL, Mogensen CE, Jakobsen J. Isokinetic muscle strength in long-term IDDM patients in relation to diabetic complications. *Diabetes.* 1996;**45**(4):440–5. [PubMed: 8603765].
3. Bokan V. Muscle weakness and other late complications of diabetic polyneuropathy. *Acta Clin Croat.* 2011;**50**(3):351–5. [PubMed: 22384769].
4. Park SW, Goodpaster BH, Strotmeyer ES, de Rekeneire N, Harris TB, Schwartz AV, et al. Decreased muscle strength and quality in older adults with type 2 diabetes: the health, aging, and body composition study. *Diabetes.* 2006;**55**(6):1813–8. doi: 10.2337/db05-1183. [PubMed: 16731847].
5. I. Jzerman TH , Schaper NC, Melai T, Meijer K, Willems PJ, Savelberg HH. Lower extremity muscle strength is reduced in people with type 2 diabetes, with and without polyneuropathy, and is associated with impaired mobility and reduced quality of life. *Diabetes Res Clin Pract.* 2012;**95**(3):345–51. doi: 10.1016/j.diabres.2011.10.026. [PubMed: 22104262].
6. Chisari C, Piaggesi A, Baccetti F, Licitra R, Rossi B. Muscle modification in asymptomatic diabetic neuropathy: a surface electromyographic study. *Basic Appl Myol.* 2002;**12**(5):177–81.
7. Hatef B, Ghanjal A, Meftahi GH, Askary-Ashtiani A. Isokinetic and Electromyographic Properties of Muscular Endurance in Short and Long-Term Type 2 Diabetes. *Glob J Health Sci.* 2016;**8**(8):210–9. doi: 10.5539/gjhs.v8n8p210. [PubMed: 27045412].
8. Gaster M, Staehr P, Beck-Nielsen H, Schroder HD, Handberg A. GLUT4 is reduced in slow muscle fibers of type 2 diabetic patients: is insulin resistance in type 2 diabetes a slow, type 1 fiber disease?. *Diabetes.* 2001;**50**(6):1324–9. [PubMed: 11375332].
9. Halvatsiotis P, Short KR, Bigelow M, Nair KS. Synthesis rate of muscle proteins, muscle functions, and amino acid kinetics in type 2 diabetes. *Diabetes.* 2002;**51**(8):2395–404. [PubMed: 12145150].
10. Hawley J, Zierath J. Physical activity and type 2 diabetes: therapeutic effects and mechanisms of action. 1 ed. Human Kinetics; 2008.
11. Marin P, Andersson B, Krotkiewski M, Bjorntorp P. Muscle fiber composition and capillary density in women and men with NIDDM. *Diabetes Care.* 1994;**17**(5):382–6. [PubMed: 8062604].
12. Roden M. Muscle triglycerides and mitochondrial function: possible mechanisms for the development of type 2 diabetes. *Int J Obes (Lond).* 2005;**29 Suppl 2**:S111–5. [PubMed: 16385762].
13. Wood RJ, O'Neill EC. Resistance Training in Type II Diabetes Mellitus: Impact on Areas of Metabolic Dysfunction in Skeletal Muscle and Potential Impact on Bone. *J Nutr Metab.* 2012;**2012**:268197. doi: 10.1155/2012/268197. [PubMed: 22474580].

14. Batsis JA, Buscemi S. Sarcopenia, sarcopenic obesity and insulin resistance. Croatia: INTECH Open Access Publisher; 2011.
15. Andreassen CS, Jakobsen J, Flyvbjerg A, Andersen H. Expression of neurotrophic factors in diabetic muscle–relation to neuropathy and muscle strength. *Brain.* 2009;**132**(Pt 10):2724–33. doi: 10.1093/brain/awp208. [PubMed: 19696031].
16. Christie A, Greig Inglis J, Kamen G, Gabriel DA. Relationships between surface EMG variables and motor unit firing rates. *Eur J Appl Physiol.* 2009;**107**(2):177–85. doi: 10.1007/s00421-009-1113-7. [PubMed: 19544067].
17. Wang R, Fukuda DH, Stout JR, Robinson EH, Miramonti AA, Fragala MS, et al. Evaluation of Electromyographic Frequency Domain Changes during a Three-Minute Maximal Effort Cycling Test. *J Sports Sci Med.* 2015;**14**(2):452–8. [PubMed: 25983596].
18. Butugan MK, Sartor CD, Watari R, Martins MC, Ortega NR, Vigneron VA, et al. Multichannel EMG-based estimation of fiber conduction velocity during isometric contraction of patients with different stages of diabetic neuropathy. *J Electromyogr Kinesiol.* 2014;**24**(4):465–72. doi: 10.1016/j.jelekin.2014.04.007. [PubMed: 24845169].
19. Watanabe K, Miyamoto T, Tanaka Y, Fukuda K, Moritani T. Type 2 diabetes mellitus patients manifest characteristic spatial EMG potential distribution pattern during sustained isometric contraction. *Diabetes Res Clin Pract.* 2012;**97**(3):468–73. doi: 10.1016/j.diabres.2012.03.004. [PubMed: 22483576].
20. Sacchetti M, Balducci S, Bazzucchi I, Carlucci F, Scotto di Palumbo A, Haxhi J, et al. Neuromuscular dysfunction in diabetes: role of nerve impairment and training status. *Med Sci Sports Exerc.* 2013;**45**(1):52–9. doi: 10.1249/MSS.0b013e318269f9bb. [PubMed: 22843109].
21. Gomes AA, Onodera AN, Otuzi ME, Pripas D, Mezzarane RA, Sacco IC. Electromyography and kinematic changes of gait cycle at different cadences in diabetic neuropathic individuals. *Muscle Nerve.* 2011;**44**(2):258–68. doi: 10.1002/mus.22051. [PubMed: 21755508].
22. Colberg SR, Sigal RJ, Fernhall B, Regensteiner JG, Blissmer BJ, Rubin RR, et al. Exercise and type 2 diabetes: the American College of Sports Medicine and the American Diabetes Association: joint position statement. *Diabetes Care.* 2010;**33**(12):147–67. doi: 10.2337/dc10-9990. [PubMed: 21115758].
23. Schellenberg F, Oberhofer K, Taylor WR, Lorenzetti S. Review of Modelling Techniques for In Vivo Muscle Force Estimation in the Lower Extremities during Strength Training. *Comput Math Methods Med.* 2015;**2015**:483921. doi: 10.1155/2015/483921. [PubMed: 26417378].
24. Harbo T, Brincks J, Andersen H. Maximal isokinetic and isometric muscle strength of major muscle groups related to age, body mass, height, and sex in 178 healthy subjects. *Eur J Appl Physiol.* 2012;**112**(1):267–75. doi: 10.1007/s00421-011-1975-3. [PubMed: 21537927].
25. Lanza IR, Nair KS. Muscle mitochondrial changes with aging and exercise. *Am J Clin Nutr.* 2009;**89**(1):467S–71S. doi: 10.3945/ajcn.2008.26717D. [PubMed: 19056588].
26. Lindle RS, Metter EJ, Lynch NA, Fleg JL, Fozard JL, Tobin J, et al. Age and gender comparisons of muscle strength in 654 women and men aged 20-93 yr. *J Appl Physiol (1985).* 1997;**83**(5):1581–7. [PubMed: 9375323].
27. Sreekumar RNK. Skeletal muscle mitochondrial dysfunction and diabetes. *Indian J Med Res.* ;**123**(3):339–410.
28. Ena J, Lozano T, Verdu G, Argente CR, Gonzalez VL. Accuracy of ankle-brachial index obtained by automated blood pressure measuring devices in patients with diabetes mellitus. *Diabetes Res Clin Pract.* 2011;**92**(3):329–36. doi: 10.1016/j.diabres.2011.02.015. [PubMed: 21397352].
29. Smith KA, Gallagher M, Hays AE, Goss FL, Robertson R. Development of the physical activity index as a measure of total activity load and total kilocalorie expenditure during submaximal walking. *J Phys Act Health.* 2012;**9**(6):757–64. [PubMed: 21952161].
30. Hatef B, Bahrpeyma F, Vaziri P. Muscle isokinetic strength and endurance in short-and long-term type 2 diabetes. *Isokinetics and Exercise Science.* 2014;**22**(4):295–301.
31. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. *J Electromyogr Kinesiol.* 2000;**10**(5):361–74. [PubMed: 11018445].
32. Gabriel DA, Christie A, Inglis JG, Kamen G. Experimental and modelling investigation of surface EMG spike analysis. *Med Eng Phys.* 2011;**33**(4):427–37. doi: 10.1016/j.medengphy.2010.11.010. [PubMed: 21146442].
33. Gabriel DA, Kamen G. Experimental and modeling investigation of spectral compression of biceps brachii SEMG activity with increasing force levels. *J Electromyogr Kinesiol.* 2009;**19**(3):437–48. doi: 10.1016/j.jelekin.2007.10.009. [PubMed: 18083563].
34. Hermens HJ, Bruggen TA, Baten CT, Rutten WL, Boom HB. The median frequency of the surface EMG power spectrum in relation to motor unit firing and action potential properties. *J Electromyogr Kinesiol.* 1992;**2**(1):15–25. doi: 10.1016/1050-6411(92)90004-3. [PubMed: 20870523].
35. Farina D, Fosci M, Merletti R. Motor unit recruitment strategies investigated by surface EMG variables. *J Appl Physiol (1985).* 2002;**92**(1):235–47. [PubMed: 11744666].
36. Gaster M, Poulsen P, Handberg A, Schroder HD, Beck-Nielsen H. Direct evidence of fiber type-dependent GLUT-4 expression in human skeletal muscle. *Am J Physiol Endocrinol Metab.* 2000;**278**(5):910–6. [PubMed: 10780948].
37. He J, Watkins S, Kelley DE. Skeletal muscle lipid content and oxidative enzyme activity in relation to muscle fiber type in type 2 diabetes and obesity. *Diabetes.* 2001;**50**(4):817–23. [PubMed: 11289047].
38. Axelson HW, Melberg A, Ronquist G, Askmark H. Microdialysis and electromyography of experimental muscle fatigue in healthy volunteers and patients with mitochondrial myopathy. *Muscle Nerve.* 2002;**26**(4):520–6. doi: 10.1002/mus.10249. [PubMed: 12362418].
39. Jansen R, Ament W, Verkerke GJ, Hof AL. Median power frequency of the surface electromyogram and blood lactate concentration in incremental cycle ergometry. *Eur J Appl Physiol Occup Physiol.* 1997;**75**(2):102–8. doi: 10.1007/s004210050133. [PubMed: 9118974].
40. Oberbach A, Bossenz Y, Lehmann S, Niebauer J, Adams V, Paschke R, et al. Altered fiber distribution and fiber-specific glycolytic and oxidative enzyme activity in skeletal muscle of patients with type 2 diabetes. *Diabetes Care.* 2006;**29**(4):895–900. [PubMed: 16567834].
41. Giombini A, Menotti F, Laudani L, Piccinini A, Fagnani F, Di Cagno A, et al. Effect of whole body vibration frequency on neuromuscular activity in ACL-deficient and healthy males. *Biol Sport.* 2015;**32**(3):243–7. doi: 10.5604/20831862.1163369. [PubMed: 26424928].
42. Dahmane R, Djordjevic S, Smerdu V. Adaptive potential of human biceps femoris muscle demonstrated by histochemical, immunohistochemical and mechanomyographical methods. *Med Biol Eng Comput.* 2006;**44**(11):999–1006. doi: 10.1007/s11517-006-0114-5. [PubMed: 17024467].
43. Garrett WE, Califf JC, Bassett FH. Histochemical correlates of hamstring injuries. *Am J Sports Med.* 1984;**12**(2):98–103. [PubMed: 6234816].
44. Farina D, Merletti R, Enoka RM. The extraction of neural strategies from the surface EMG. *J Appl Physiol (1985).* 2004;**96**(4):1486–95. doi: 10.1152/japplphysiol.01070.2003. [PubMed: 15016793].
45. Hatef B, Bahrpeyma F, Mohajeri Tehrani MR. The comparison of muscle strength and short-term endurance in the different periods of type 2 diabetes. *J Diabetes Metab Disord.* 2014;**13**(1):22. doi: 10.1186/2251-6581-13-22. [PubMed: 24476108].