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Original Article

Health indicators and costs among outpatients according to physical activity level and obesity

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ABSTRACT

Objective: How biochemical variables influence the costs of the Brazilian National Healthcare System, according to body composition and physical activity.
Methods: Participated in this study 168 patients. Biochemical variables were glucose, triglycerides, total cholesterol, high, low, very low density lipoprotein and C-reactive protein (CRP). For the cost analysis the medical records was analyzed. Physical activity was assessed through questionnaire. Body adiposity was assessed by body mass index. Four groups were defined according body adiposity and physical activity.
Results: The active obese group had higher values of very low density lipoprotein and triglycerides when compared to the inactive obese. The non-obese inactive group had lower values of non-high density lipoprotein compared to the inactive obese. The non-obese active group presented lower insulin value when compared to the inactive obese. The inactive obese group presented higher values in the CRP when compared to the non-obese active and inactive groups when compared to non-obese and active obese group. There was a positive correlation between insulin, glucose, CRP and drug and total costs.
Conclusions: Biochemical variables were different according to body composition and physical activity. Insulin, glucose and CRP were related to cost in drugs and total costs.

1. Introduction

The prevalence of obesity has been considered the worldwide epidemic of the new millennium [1]. Around 2.8 million adults in the world die of events related to overweight and obesity [2], and this health indicator is strongly associated with several chronic non-communicable diseases (NCD) [3].

Obesity is characterized by an excessive accumulation of body fat reserves [4], and its etiology is multifactorial and complex, caused by genes interactions, lifestyle, environmental, and emotional factors [5]. According to the Brazilian Institute of Geography and Statistics [6], 50% of the Brazilian population over 20 years old is overweight. The World Health Organization [4] reported 2.3 billion overweight adults and more than 700 million obese in 2015.

Regarding the physiological aspects, the literature points out, obese individuals have a higher propensity to present altered biochemical parameters, such as total cholesterol, low-density lipoprotein, triglycerides [7], high-density lipoprotein [8] and C-reactive protein (CRP) [9], values that result in the acquisition of NCD such as diabetes, cardiovascular diseases, and cancer [10].

In the financial framework, obesity demands high costs. Bahia and colleagues [11] estimated that the Brazilian National Healthcare System spent 3.6 billion dollars with obesity treatment

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between 2008 and 2010, specifically 1.2 billion with outpatient treatment and 2.4 billion with hospital treatment.

The majority of the adult population gains weight over a long period of time, so it is relevant to determine how physical activity attenuates undesirable risk factors for NCD [12]. One study examined the association between sedentary behavior and television viewing on the risk of obesity and type 2 diabetes over a 6-year period. Physical activity was associated with a 9% reduction in obesity and a 12% reduction in type 2 diabetes, and walking 1 h per day was associated with a 24% reduction in obesity and a 34% reduction in type 2 diabetes [13].

When aiming to decrease expenditures with obesity, studies suggest non-pharmacological interventions for treatment and prevention, such as physical exercise, a method with numerous benefits [14] and effective for health management [15].

Aoyagi and Shepard [16] carried out a study including 5000 elderly people diagnosed with several NCD, applied an exercise and pedometer intervention, and concluded that if 5% of the elderly population increased 2000 steps per day, it would result in 3.7% reduction of medical expenditures. Thus, the objective of this study was to analyze how biochemical variables influence the costs of outpatients of the Brazilian National Healthcare System, according to body composition and level of physical activity.

2. Methods

The sample was composed of 168 patients aged 50 years or older attending two different Basic Healthcare Units (BHU) of Presidente Prudente, SP, Brazil. The inclusion criteria adopted for the recruitment of patients were: i) patients enrolled at BHU for at least one year, actively using health service; ii) 50 years or older; iii) signed consent form.

Biochemical variables were collected and analyzed by an enabled laboratory who removed 10 ml of blood from the peripheral puncture of the forearm vein, after a 12 h fasting. For the analysis of glucose, triglycerides (TG), total cholesterol (TC), high and low-density lipoprotein (HDL, LDL) were used the colorimetric method (Labtest®, Brazil). For cytokine and hormone (CRP and insulin) dosage, the ELISA method was used according to R&D Systems®, USA and ALPCO®, USA.

To classify the insulin resistance, Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated using the following equation: HOMA-IR= (insulin fasting (μUI/mL) x fasting glucose (mg/dL))/22,5 [17].

Medical records of the last 12 months of the interview date [18–20] were used to access the treatment cost of each patient. The information provided was the number of consultations, medications, and laboratory tests. It was also calculated costs with other health services, such as materials, employers (nurses and administrative) payrolls, and water, electricity and telephone bills [18,19].

The level of physical activity was assessed using Baecke's questionnaire [21], validated in Brazil by Florindo [22], which evaluates the level of habitual physical activity according to three domains, i) occupational, ii) physical exercise and iii) leisure and locomotion, in the last 12 months. Each domain results in a score and the sum determines the level of physical activity of the patient. For final score was classified in quartiles: $P \leq 25$ for inactive patients and $P \geq 75$ active patients.

Body adiposity was classified according to body mass index (BMI) in kg/m^2, and measurements were taken according to Lohman and colleagues protocol [23]. The obese/overweight group was characterized by patients who presented values above 25 kg/m^2. The non-obese group consisted of patients with BMI between 17 kg/m^2 and 24.9 kg/m^2.

For statistical analyses, the four groups were defined: non-obese active, non-obese inactive, obese active, and obese inactive. For the comparison between groups, we used the analysis of variance (ANOVA) with Tukey's Post-Hoc test, and the Spearman test was used for correlations. The level of significance was set at $p < 0.05$ and the software used was BioStat 5.0.

3. Results

The sample was composed of 168 patients (53 males and 115 females). Regarding the levels of physical activity, there was a significant difference between non-obese inactive, obese-active, and obese-inactive groups ($p = 0.001$). For BMI and waist circumference, the highest values were found in obese-active and obese-inactive groups ($p = 0.001$). There were differences regarding fat percentage between all groups ($p = 0.001$). Finally, there were no significant differences between groups for age, weight, height, systolic, and diastolic blood pressure (see Table 1).

Table 2 shows the biochemical variables according to the groups. The active obese group showed higher values of VLDL ($p = 0.001$) and TG ($p = 0.017$) when compared to the non-obese active group. The inactive non-obese group had lower values of no-HDL ($p = 0.034$) when compared to the inactive obese group. The active non-obese group presented a lower value of insulin ($p = 0.048$) when compared to the inactive obese group. The inactive obese group presented higher values of CRP ($p = 0.007$) when compared to the active and inactive non-obese groups. The inactive obese group presented higher values of HOMA-IR ($p = 0.001$) when compared to the non-obese and active obese groups. The variables of total cholesterol, HDL, LDL, and glucose were not statistically different.

Table 3 shows the cost variables (consultations, tests, medications, and total cost) according to groups, and we did not find statistical differences between groups.

Table 4 shows the correlations between biochemical variables and costs. The non-obese inactive (glucose and drugs and total cost), obese-active (glucose and CRP and total cost) and obese-inactive (TG and lab test and CRP and total cost) groups presented a positive correlation between tests results and health cost. The non-obese active group presented a negative correlation between medication cost and insulin results.

4. Discussion

The objective of the present study was to analyze the influence of biochemical variables on patients' costs according to body composition and physical activity level.

Initially, we found statistical differences between groups regarding habitual physical activity level, BMI, waist circumference, and fat percentage. As predicted, the level of habitual physical activity and BMI were different between groups because these variables were used to divide the groups. Additionally, it makes sense that waist circumference and body fat were also different between groups because higher BMI is associated with increased waist circumference and fat percentage [24].

We also found differences in VLDL and TG, with higher values in the active obese group when compared to the non-obese active group. VLDL is a member of the LDL family, it is involved in remaining lipoprotein particles in peripheral tissues and in cooperation with lipoprotein lipase, thus VLDL is directly related to the development of atherosclerosis and insulin resistance. However, low values of VLDL have been suggested as a target to improve the disease [25]. A study conducted by Oh Yoen, Kim et al. [26] found an association of VLDL with elevated TG levels, and an inverse relationship with the amounts of adiponectin (a protein hormone). One of the adiponectin functions is to control energy metabolism and

Table 1
Descriptive variables according to the presence of obesity and physical activity level.

	Non-obese active n = 21	Non-obese inactive n = 42	Obese active n = 50	Obese inactive n = 55	p-value
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	
Age (years)	64.51 (7.99)	65.72 (10.07)	61.78 (7.59)	64.08 (8.29)	0.169
Weight (Kg)	61.62 (14.06)	62.91 (10.41)	78.16 (12.54)	79.72 (15.46)	0.790
Height (cm)	158.32 (9.15)	159.32 (9.63)	158.32 (8.35)	155.24 (7.57)	0.139
Level of PA (score)	6.80 (1.19)	5.75 (1.34)	7.23 (1.76)[b]	5.89 (1.56)[b]	**0.001**
SP (mmHg)	129.95 (15.74)	130.05 (21.85)	127.28 (17.32)	132.69 (20.47)	0.485
DP (mmHg)	76.19 (10.98)	74.48 (11.29)	76.78 (8.65)	77.20 (12.48)	0.665
IMC (kg/m^2)	24.35 (3.89)	24.62 (2.09)	31.20 (4.74)[a,b]	32.89 (5.23)[a,b]	**0.001**
WC (cm)	82.17 (17.10)	85.83 (8.79)	101.51 (9.16)[a,b]	103.29 (10.62)[a,b]	**0.001**
Fat %	31.83 (5.93)	30.78 (6.12)	41.01 (6.52)[a,b]	44.41 (6.26)[a,b,c]	**0.001**

PA: physical activity, SP: systolic blood pressure, DP: diastolic blood pressure, WC: waist circumference, IMC: body mass index, a = difference non-obese active, b = difference non-obese inactive, c = difference active obese.

Table 2
Biochemical variables according to groups.

Variable	Group (N)	Analysis of variance (ANOVA)		
		Mean (SD)	F	p-value
TC	Non-obese active (n = 20)	197.66 (38.53)	2.197	0.092
	Non-obese inactive (n = 40)	186.65 (33.57)		
	Active obese (n = 25)	200.93 (27.97)		
	Obese inactive (n = 37)	206.58 (38.47)		
HDL	Non-obese active (n = 20)	45.25 (8.41)	1.430	0.238
	Non-obese inactive (n = 40)	46.01 (10.88)		
	Active obese (n = 25)	46.95 (12.16)		
	Obese inactive (n = 37)	41.94 (10.14)		
VLDL	Non-obese active (n = 20)	27.91 (12.48)	7.801	**0.001**
	Non-obese inactive (n = 40)	22.84 (10.60)		
	Active obese (n = 25)	31.24 (15.99)[a]		
	Obese inactive (n = 37)	37.76 (15.01)		
LDL	Non-obese active (n = 20)	124.50 (32.25)	0.545	0.653
	Non-obese inactive (n = 40)	117.13 (28.66)		
	Active obese (n = 25)	122.73 (23.01)		
	Obese inactive (n = 37)	125.56 (35.55)		
No-HDL	Non-obese active (n = 20)	152.41 (35.92)	2.978	**0.034**
	Non-obese inactive (n = 40)	140.64 (32.54)		
	Active obese (n = 25)	153.97 (28.66)		
	Obese inactive (n = 37)	164.64 (41.33)[b]		
TG	Non-obese active (n = 20)	139.57 (62.42)	3.534	**0.017**
	Non-obese inactive (n = 40)	126.31 (92.61)		
	Active obese (n = 25)	156.16 (79.98)[a]		
	Obese inactive (n = 37)	206.91 (162.04)		
Glucose	Non-obese active (n = 20)	109.60 (63.02)	2.303	0.081
	Non-obese inactive (n = 40)	90.73 (14.89)		
	Active obese (n = 25)	114.90 (43.88)		
	Obese inactive (n = 37)	113.00 (51.43)		
Insulin	Non-obese active (n = 20)	6.74 (3.26)	2.713	**0.048**
	Non-obese inactive (n = 40)	6.93 (3.76)		
	Active obese (n = 25)	13.80 (8.59)		
	Obese inactive (n = 37)	22.91 (48.03)[a]		
CRP	Non-obese active (n = 20)	1.36 (1.34)	4.244	**0.007**
	Non-obese inactive (n = 40)	2.46 (4.84)		
	Active obese (n = 25)	5.26 (5.59)		
	Obese inactive (n = 37)	6.15 (8.20)[a,b]		
HOMA-IR	Non-obese active (n = 20)	1.81 (1.25)	13.32	**0.001**
	Non-obese inactive (n = 40)	1.56 (0.87)		
	Active obese (n = 25)	3.91 (2.91)		
	Obese inactive (n = 37)	4.52 (3.23)[a,c]		

TC: Total cholesterol HDL: High-Density Lipoprotein, VLDL: Very Low-Density Lipoprotein, LDL: Low-Density Lipoprotein, N-HDL: No-High Density Lipoprotein, TG: Triglycerides, CRP: C-reactive protein, HOMA-IR: homeostatic model assessment. a = denotes difference compared to Non-obese active; b = denotes difference compared to Non-obese inactive; c = denotes difference compared to Active obese.

Table 3
Cost variables according to the groups.

Cost	Group (N)	Analysis of variance (ANOVA)		
		Mean (SD)	F	p-value
Consultations	Non-obese active (n = 21)	22.20 (20.53)	0.519	0.670
	Non-obese inactive (n = 42)	23.88 (16.85)		
	Active obese (n = 29)	27.55 (16.79)		
	Obese inactive (n = 39)	25.92 (14.30)		
Exams	Non-obese active (n = 21)	13.46 (32.27)	0.536	0.659
	Non-obese inactive (n = 42)	9.35 (23.38)		
	Active obese (n = 29)	9.41 (21.16)		
	Obese inactive (n = 39)	15.93 (29.91)		
Drug	Non-obese active (n = 21)	58.69 (94.70)	1.135	0.338
	Non-obese inactive (n = 42)	46.49 (78.47)		
	Active obese (n = 29)	74.43 (94.18)		
	Obese inactive (n = 39)	42.29 (44.33)		
Total	Non-obese active (n = 21)	108.60 (107.72)	0.810	0.419
	Non-obese inactive (n = 42)	97.53 (99.61)		
	Active obese (n = 29)	130.06 (100.07)		
	Obese inactive (n = 39)	102.00 (58.93)		

weight loss and this is because adiponectin levels are lower in the non-obese active group.

The inactive obese group presented a higher value of no-HDL than the non-obese inactive group. The study by Lu Liu et al. [27] pointed to No-HDL as an increase in discriminatory power together with higher levels of BMI, stressing that obese women should have high serum lipid levels, which is part of the present study because most of the sample is women. Still, Robinson et al. [28] showed that abdominal obesity and age influence levels of No-HDL, total cholesterol, and TG.

It is known that obesity causes insulin resistance through inflammatory cytokines underlying TNF-α [29], also that the origin of these cytokines are migrations of macrophages to adipocytes [30]. Thus, as expected, insulin in this study had higher values in the inactive obese group when compared to the non-obese active group.

Obesity has been associated with increased pro-inflammatory biomarkers and systemic inflammation, and it should be noted that inflammation is an important indicator of cardiovascular disease [31]. However, chronic inflammation can cause increased cell stress and increase the release of cytokines, which can develop elevated levels of systemic inflammation characterized by CRP [32]. Our finding confirms the high CRP values in the inactive obese group when compared to the non-obese active and non-obese inactive groups. A study by Briggs et al. [33] and by Robinson, colleagues [28] showed CRP values above 3.0 mg/dL in obese

Table 4
Correlation between costs and biochemical variables.

Cost	Group	TC	HDL	VLDL	LDL	N-HDL	TG	Glucose	Insulin	CRP	HOMA-IR
Non-obese active											
Doctor's appointments		0.048	0.341	−0.026	0.014	−0.056	−0.032	−0.319	−0.327	−0.190	−0.424
Exams		−0.073	−0.166	0.095	−0.009	0.003	0.095	−0.035	0.191	0.131	0.085
Drug		−0.007	−0.024	0.226	−0.016	−0.039	0.216	0.181	**−0.479***	0.120	−0.271
Total		0.066	−0.038	0.243	0.114	0.053	0.235	0.101	−0.359	0.116	−0.248
Non-obese inactive											
Doctor's appointments		−0.002	0.290	−0.157	0.028	0.053	−0.153	0.378	−0.054	0.163	−0.015
Exams		0.255	0.146	−0.001	0.264	0.292	0.007	−0.164	−0.344	0.147	−0.415
Drug		−0.251	0.122	−0.412	−0.208	−0.226	−0.411	**0.539***	−0.033	0.074	0.071
Total		−0.199	0.161	−0.303	−0.142	−0.142	−0.297	**0.497***	−0.098	0.255	−0.021
Obese-active											
Doctor's appointments		0.070	−0.056	−0.119	0.090	0.081	0.139	0.420	−0.054	0.330	0.152
Exams		0.112	−0.299	−0.258	0.194	0.261	0.156	0.125	0.174	0.365	0.111
Drug		−0.049	−0.085	0.254	−0.165	0.140	0.315	0.430	0.070	0.183	0.283
Total		0.053	−0.196	0.077	0.064	0.280	0.294	**0.478***	0.118	**0.489***	0.307
Obese inactive											
Doctor's appointments		0.136	−0.057	0.001	0.099	0.278	0.234	0.011	−0.026	0.240	−0.020
Exams		0.292	−0.143	0.030	0.172	0.402	**0.448***	−0.296	−0.294	0.328	−0.420
Drug		−0.172	0.018	0.199	−0.068	−0.163	−0.193	0.296	−0.035	0.433	0.066
Total		0.130	0.060	0.109	0.017	0.171	0.093	0.086	−0.055	**0.583****	−0.026

*p = 0,05 **p = 0,001.

patients, which corroborates with the present study where the CRP values reached 6.15 mg/dL.

The results also showed higher HOMA-IR levels in the inactive obese group when compared to the non-obese active and obese active groups. Independently of the level of physical activity, the values are high, however, the study by Nieuwpoort et al. [34] found a relationship between HOMA-IR and BMI, Glucose, Insulin, Leptin, and Resistin, thus, the values of these variables can alter HOMA-IR levels in obese patients. Bousier and colleagues [35] carried out a study with obese people (n = 498), of which 27.9% were grade I, 37.3% grade II and 34.7% grade III, and presented mean values of HOMA-IR of 2.9, which makes it different from the values of the present study, because the inactive obese group that obtained a higher value reached 4.5 in this variable, showing the importance of considering resistant and insulin sensitive patients.

Regarding the costs of consultations, lab tests, and medications, we did not find differences, however, the literature presents other results [11,36–39].

There was a moderate and inverse correlation between medication costs and insulin in the non-obese active group, i.e., lower use of medication, higher insulin level. This result makes sense if we think of type 2 diabetics with insulin resistance, a process defined as the lower uptake of glucose by the muscles and adipose tissues, and an increased release of glucose in the liver [40], consequently, the patient needs medication to lower insulin rates.

In the non-obese inactive group, there was a moderate correlation between glucose, medication costs, and total costs. In this study, independent of the group, it was assessed the prevalence rate of diabetes, however, it is understood that glucose is linked to this group due to the sedentary lifestyle and bad eating habits, causing higher demand of medication to lower the levels of glucose [41].

The active obese group showed a moderate correlation between glucose, CRP and total costs. The inactive obese group showed a moderate correlation between total costs and CRP. The correlations of the obese groups can be explained due to the inflammations caused by the dysfunctions in the adipose tissues that alter the secretion of adiponectin, altering its level, increasing levels of retinol binding protein 4 (RBP4), insulin resistance, also modifying CRP values, interleukin-6, monocyte chemotactic protein (MCP-1) and tumor necrosis (TNF-α) [42]. These changes reflect higher demand for health services provided by the Brazilian National Healthcare System and higher total costs.

5. Conclusion

It can be concluded that the biochemical variables VLDL, N-HDL, TG, Insulin, CRP and HOMA-IR were different in relation to body composition and levels of physical activity. Insulin, glucose and CRP were related to cost in drugs and total costs, but no influence was found in the costs of patients with different body composition and levels of physical activity.

Conflicts of interest

The authors state no conflict of interest.

Ethical approval

All procedures performed in this study involving human participants were in accordance with the ethical standards of the Ethics Research Committee of the Sao Paulo State University approved the study (CAAE 47386715.9.0000.5402).

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References

[1] Roberto CA, Swinburn B, Hawkes C, et al. Patchy progress on obesity prevention: emerging examples, entrenched barriers, and new thinking. Lancet 2015;385:2400–9.
[2] Singh GM, Danaei G, Farzadfar F, et al. The age-specific quantitative effects of metabolic risk factors on cardiovascular diseases and diabetes: a pooled analysis. PLoS One 2013;8(7). e65174.
[3] Emerging risk factors collaboration, Wormser D, Kaptoge S, et al. Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. Lancet 2011;377:1085–95.
[4] WHO. Obesity: Health topics. World Health Organization; 2016.

[5] Diretrizes Brasileiras de Obesidade, 4.ª edição, Associação Brasileira para o Estudo da Obesidade. Cap. Ganho de peso e obesidade 2016, São Paulo, p. 31-52.
[6] Instituto Brasileiro de Geografia e Estatística (IBGE), Pesquisa Nacional de Saúde. Percepção do estado de saúde, estilos de vida e doenças crônicas. 2013. p. 15.
[7] Stadler TAC, Moretti M, Poretti MP, Sakae TM, Araujo D. Associação dos níveis de dislipidemia entre obesidade tipo I, II e III. Arq Catarinenses Med 2011;40(3).
[8] Schmidt MI, Duncan BB, Watson RL, Shannett AR, Blancati FL. Heiss GA metabolic syndrome in whites and african-americans: the atherosclerosis risk in communities (ARIC) baseline study. Diabetes Care 1996;19(5):414–8.
[9] Rexrode KM, Pradhan A, Mansos JE, Buring JE, Ridker PM. Relationship of total and abdominal adiposity with CRP and IL-6 in women. Ann Epidemiol 2003;13:1–9.
[10] Schmidt MI, Duncan BB, Silva GA, Menezes AM, Monteiro CA, Barreto SM, et al. Chronic noncommunicable diseases in Brazil: burden and current challenges. Lancet 2011 Jun;9(11):60135–9.
[11] Bahia L1, Coutinho ES, Barufaldi LA, Abreu Gde A, Malhão TA, de Souza CP, Araujo DV. The costs of overweight and obesity-related diseases in the Brazilian public health system: cross-sectional study. BMC Public Health 2012 Jun 18;12:440.
[12] Donnelly JE, Blair SN, Jakicic JM, Manore MM, Rankin JW, Smith BK. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. American College of Sports Medicine; 2009. p. 459–71.
[13] Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. J Am Med Assoc 2003;289:1785–91.
[14] Bueno DR, Marucci MFN, Codogno JS, Roediger MA. Os custos da inatividade física no mundo: estudo de revisão. Ciência Saúde Coletiva 2016;21(4):1001–10.
[15] Bielemann RM, Knuth AG, Hallal PC. Atividade física e redução de custos por doenças crônicas para o Sistema Único de Saúde. Rev. Bras. Ativ. Fís. Saúde 2010;15(1):9–14.
[16] Aoyagi Y, Shephard RJ. Model to estimate the potential for a physical activity-induced reduction in Healthcare costs for the elderly, based on pedometer/accelerometer data from the nakanojo study. Sports Med 2011 Sep 1;41(9):695–708.
[17] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985 Jul;28(7):412–9.
[18] Codogno JS, Fernandes RA, Monteiro HL. Prática de atividades físicas e custo do tratamento ambulatorial de diabéticos tipo 2 atendidos em unidade básica de saúde. Arq Bras Endocrinol Metab 2012 Feb;56(1):06–11.
[19] Codogno JS, Fernandes RA, Rosa CSC, Bueno DR, Monteiro HL. Custo com tratamento e indicadores de risco em pacientes diabéticos, segundo esquema terapêutico. Rev. Ed. Fís 2011;22(1):115–22.
[20] JS1 Codogno, Turi BC, Kemper HC, Fernandes RA, Christofaro DG, Monteiro HL. Physical inactivity of adults and 1-year health care expenditures in Brazil. Int. J. Public Health 2015 Mar;60(3):309–16.
[21] Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. Am J Clin Nutr 1982 Nov;36(5):936–42.
[22] Florindo AA, Latorre MRDO. Validation and reliability of the Baecke questionnaire for the evaluation of habitual physical activity in adult men. Rev Bras Med Esporte 2003 June;9(3):129–35.
[23] Lohman TG, Roche AE, Martorell R. Anthropometric standardization reference manual. Illioes: Human Kinetics Book; 1988. p. 90.
[24] Hayakawa YK, Sasaki S, Takao H, Yoshikawa T, Hayashi N, Mori H, Kunimatsu A, Aoki S, Ohtomo K. The relationship of waist circumference and body mass index to grey matter volume in community dwelling adults with mild obesity. Obes.Sci.Pract. 2017:97–1005.
[25] Takahashi S, Sakai J, Fujino T, Miyamori I, Yamamoto TT. The very low density lipoprotein (VLDL) receptorda peripheral lipoprotein receptor for remnant lipoproteins into fatty acid active tissues. Mol Cell Biochem 2003;248:121–7.
[26] Oh YK, Seung-Min L, Ji HC, Hyun JD, Jiyoung M, Min-Jeong S. Arginase I and the very low-density lipoprotein receptor are associated with phenotypic biomarkers for obesity. Nutrition 2012;28:635–9.
[27] Lu L, Qiu L, Zhongshang Y, Meng Z, Xu Z, Haiqing Z, et al. Non-high-density lipoprotein cholesterol is more informative than traditional cholesterol indices in predicting diabetes risk for women with normal glucose tolerance. J Diabetes Investig 2018:1–8.
[28] Robinson JG, Ballantyne CM, Hsueh WA, Rosen JB, Lin J, Shah AK, et al. Age, abdominal obesity, and baseline high-sensitivity C-reactive protein are associated with low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein B responses to ezetimibe/simvastatin and atorvastatin in patients with metabolic syndrome. J.Clin. Lipidol. 2013;7:292–303.
[29] Waki H, Tontonoz P. Endocrine functions of adipose tissue. Annu Rev Pathol 2007;2:31–56.
[30] Tsukumo DM, Carvalho-Filho MA, Carvalheira JB, Prada PO, Hirabara SM, Schenka AA. Loss-of-function mutation in toll-like receptor 4 prevents diet-induced and insulin resistance. Diabetes 2007;56(8):1986–8.
[31] Stienstra R, Tack CJ, Kanneganti TD, Joosten LA, Netea MG. The inflammasome puts obesity in the danger zone. Cell Metab 2012;15:10–8.
[32] Barbe MF, Barr AE. Inflammation and the pathophysiology of workrelated musculoskeletal disorders. Brain Behav Immun 2006;20:423–9.
[33] Briggs MS, Givens DL, Schmitt LC, Taylor CA. Relations of C-reactive protein and obesity to the prevalence and the odds of reporting low back pain. Arch Phys Med Rehabil 2013;94:745–52.
[34] Nieuwpoort CV, Twisk JWR, Curfs LMG, Lips P, Drent ML. Body composition, adipokines, bone mineral density and bone remodeling markers in relation to IGF-1 levels in adults with Prader-Willi syndrome. Int J Pediatr Endocrinol 2018;2018(1).
[35] Boursier G, Sultan A, Molinari N, Maimoun L, Boegner C, Picandet M, et al. Triglycerides and glycated hemoglobin for screening insulin resistance in obese patients. Clinical Biochemistry. Clin Biochem 2018 Mar;53:8–12.
[36] Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer- and service-specific estimates. Health Aff 2009;28(5):w822–31.
[37] Denasus. Departamento Nacional de Auditoria do Sistema Único de Saúde. 2013. Disponível em, http://portalsaude.saude.gov.br/index.php/oministerio/principal/secretarias/sgep/denasus-departamento-nacional-de-auditoria-do-sistema-unico-de-saude.
[38] Malta DC, Cezário AC, Moura L, Morais Neto OL, Silva Júnir JB. Construção da vigilância e prevenção das doenças crônicas não transmissíveis no contexto do sistema único de saúde. Epidemiol. Serv. Saúde. 2006 Set;15(3):47–65.
[39] Moreira MM, Ikegami EM, Mesquita IMR, Amaro EA, et al. Impacto da inatividade física nos custos com internações hospitalares para doenças crônicas no Sistema Único de Saúde. Arq. Cien. Esp 2017;5(1):16–9.
[40] Wilding JPH. The role of the kidneys in glucose homeostasis in type 2 diabetes: clinical implications and therapeutic significance through sodium glucose cotransporter 2 inhibitors. Metabolism 2014;63:1228.
[41] Kahn SE, Cooper ME, Del Prato S. Pathophysiology and treatment of type 2 diabetes: perspectives on the past, present, and future. Lancet 2014;383:1068.
[42] Kahn SE, Zinman B, Haffner SM, O'Neill MC, Kravitz BG, Yu D, et al. Obesity is a major determinant of the association of C-reactive protein levels and the metabolic syndrome in type 2 diabetes. Diabetes 2006;55(8):2357–64.