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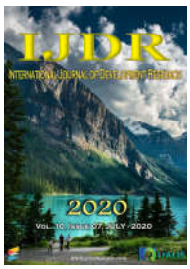

RESEARCH ARTICLE OPEN ACCESS

DEPRESSION/ANXIETY AND METABOLIC SYNDROME: ASSOCIATION BETWEEN MINOR MENTAL DISORDERS AND METABOLIC SYNDROME

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ABSTRACT

Metabolic syndrome (MS) is a group of metabolic, anthropometric and homodynamic modifications, including intolerance to glucose, abdominal obesity, dyslipidemia and hypertension. Either isolated or mainly combined, these factors increase the risk of atherothrombotic disease and type 2 diabetes *mellitus*. The psychological and emotional aspects are also important determining factor of diseases, including the cardiovascular ones, because of their risk failures. Studies with depression, anxiety and MS are still rare in literature and present controversial results. In order to contribute to this gap, this study tested the association between depression/anxiety and MS in a cross-sectional study encompassing adult men and women. MS was defined by the criteria of the International Federation of Diabetes, being characterized in the presence of abdominal obesity (≥84 cm of waist circumference for women and ≥ 99 cm of waist circumference for men), besides two of the following criteria: hypertension (≥ 130/85 mmHg), hyperglycemia (≥ 100 mg/dl), hypertriglyceridemia (≥150 mg/dl) and hypoalphalipoproteinemia (HDL cholesterol less than 40 mg/dl for women and less than 50 for men). Depression/anxiety was evaluated by the combination of the variables, employing the sub-scale (PSAD) of 12 depression and anxiety items of the Questionnaire of Psychiatric Morbidity in Adults (QPMA). All the items referred to the current situation, as well as to the one of the last 12 months. The state of anxiety was defined for the ones who presented a score higher or the same as 18 in the PSAD/QPMA and that referred to at least one specific symptom of anxiety. The state of depression was defined for the ones who presented a score the same or higher than 18 of the PSAD/QMPA and who related at least one specific symptom of depression. The final sample was of 1333 adults. Amongst women, 22.5% presented anxiety and/or depression, while for men these values were of 9.1% (p= 0.000). In the logistic regression, the adjusted prevalence ratio (PR) for depression/anxiety and MS for women was 0.95 (CI 95% 0.64-1.38**).** For men, the adjusted prevalence ratio was 2.35 (CI 95% 1.05-3.77) with statistical significance. For both genders, the confounding factors were age and marital situation, especially for black/mixed blood men and the ones who lead a sedentary life. This study highlights the association between depression/anxiety for men and presents a new confounding factor referring to mixed blood and black men. Further studies will incorporate this form of evaluating minor mental disorders.

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INTRODUCTION

Metabolic syndrome (MS) is a group of metabolic, anthropometric and homodynamic modifications, including intolerance to glucose, abdominal obesity, dyslipidemia and hypertension. Either isolated or mainly combined, these factors increase the risk of cardiovascular diseases(Motillo *et al*., 2010). The connections between social factors and cardiovascular diseases are complex, involving interrelated

processes, encompassing macro determination, for example, the work and family spheres, as well as more proximal or biological risk factors, such as MS. Despite being established that genetic and lifestyle factors are involved in the etiology of this syndrome, the basic mechanisms have not yet been clearly elucidated (Bouchard, Despres, & Mauriege, 1993). Psychological features, related to the personality, chronic stress and labor situations, anxiety and depression may also be involved (Björntorp, 2001). Ever since the nineties studies on MS components have demonstrated that socio-emotional aspects are associated to abdominal obesity, dysglycemia, dyslipidemiaand to arterial hypertension(Rosmond, Eriksson, & Björntorp, 1999; Feldman, & Steptoe, 2003; Knox *et al*., 1996; Jones-Webb *et al*., 1996). In 2004, Rosenren& col. (in the *INTERHEART study*), demonstrated a strong association between psychosocial risk and acute myocardial infarction, in a case-control study, in 52 countries, the *Frankenhaeuser*bio-psychosocial model tries to comprise these three aspects (Rosengren *et al*., 2004; Frankenhaeuser *et al*., 1989). In such proposal, validated empirically in its different aspects, the model manages to explain the complex organic response related to the environment, interfering in a direct manner on the health-illness process. This system, in a simplified manner, is triggered when the cerebral cortex perceives a challenge or a demand; the brain assesses its importance and the capacity of the individual in solving them or not. This perception triggers electrochemical signals to regions of the hippocampus and of the hypothalamus and, through the autonomic nervous system (ANS), noradrenaline (NA) and adrenaline (A) are released by the adrenal glands. These stress hormones mobilize all the body reserves for the known response of fight-or-flight. This sensation of insecurity, dissatisfaction and thread makes the cerebral cortex to release adrenocorticotropic hormone (ACTH), which in turn, stimulates the cortical region of the adrenal glands releasing a third stress hormone, cortisol. The dosages of these hormones are objective indicators of the individualized response to stress(Kautzky-Willer, Harreiter, & Pacini, 2016).

The psycho-emotional stress study encompasses various factors considered as external stressors, either at work or at home, through theoretical models of requirements (demands) and/or decision-making, prioritization or negotiation (control) capabilities(das Merces *et al*., 2019). Adverse events in life, such as bereavement, loss of employment, important material loss, or overload of daily activities can also trigger an increase or functional alterations to the mentioned hormones. Adrenalineand noradrenaline are, for example, responsible for the acute cardiovascular responses, leading to systemic or regional arterial hypertension or to cerebrovascular accidents (stroke). Cortisol has a more chronic action and acts on the cellular metabolism. It best represents the alterations to the immune system, and for diseases, such as cancer and diabetes *mellitus,* to the cognitive cerebral alterations(das Merces *et al*., 2019). With reference to serum alterations, this hormone is the mediator for the release of free fatty acids (lipolysis) and for the incorporation of fat by the young adipocytes, located in the abdominal viscera. These adipocytes, of great importance in visceral obesity, have a large quantity of receptors for cortisol and some of its metabolites. Cortisol is a potent antagonist of insulin and of low serum concentrations of hdl-cholesterol, and to the appearance of glucose intolerance.This intolerance has been associated to increased basal secretions of cortisol(Thomas, Kalaria, & O´Brien, 2004).

Thus, it seems plausible for stress to be associated to MS. One of the greatest difficulties in measuring these hormones or the alterations directly produced by them is the higher or lower capacity of the individuals to react to these external stressors, leading to different endogenous responses, with equal or very similar stimuli. Accordingly, there is a great difficulty to obtain an objective evaluation in population studies(Kautzky-Willer, Harreiter, & Pacini, 2016). At present robust studies to assess relations between depression/anxiety and MS are still scarce in literature. In 1997, Brunner & col., on analysis of the social distribution and its determinants, in 7063 workers, using the *WhitehallI*cohort,demonstrated an inverse social gradient, in other words, the least qualified the worker, the higher is the prevalence to the main MS component, abdominal obesity(Brunner *et al*., 2002; Brunner, & Marmot, 2005). Namely, the occurrence of central obesity was inversely associated to the socioeconomic situation of these workers. In this same cohort, five years later, a nested case-control study, with 183 men, evidenced that the individuals with MS had a higher activation of the stress neuroendocrine axis, assessed by the cortisol and catecholamine, when compared to those that do not fulfill the MS criteria(Brunner *et al*., 2002). Raikkönen& col.and Kinder & col. demonstrated an association between depression and MS (Räikkönen, Matthews, & Kuller, 2002; Kinder *et al*., 2002). The first was an exclusive cohort study with 425 women; those with high levels of tension, depression or rage, in the basal phase of the study, presented a high risk of developing MS (p=0.04) at follow-up. In the second study, a cross-sectional design representative of the population of North America (*NHANES*) demonstrated a four-time greater prevalence for the development of MS in women with episodes of depression. In another small study, from a convenience sample of 116 individuals, Petrlova& col. (Petrlova , Rosolva, & Ai, 2004)found, in depressed people, a four-time higher risk of MS when compared to those without depression. However, the study by Herva& col. (Herva *et al*., 2006), in a birth cohort of young Caucasians, no association was demonstrated between depression and MS. National studies on the metabolic syndrome or its components, related to psycho-emotional aspects, are still scarce. Licínio *et al*.(Licinio, Yildiz, & Wong, 2002), in a clinical trial with outpatients in the clinical pharmacological department in Los Angeles, U.S.A., revealed an association between depression and obesity, with the latter being considered as a risk factor for the development of MS. Merces& col. (das Merces *et al*., 2016)in a study with Primary Healthcare Nursing in Bahia, Brazil, evidenced an association between exacerbated occupational stress and the increase in abdominal adiposity. Thus, the effort to assess depression/anxiety and MS in a population-based study, encompassing adult men and women, of different ethnical groups, seems to be very pertinent in our midst.

METHODS

The study is part of the Project for Monitoring Cardiovascular Diseases ("Monite"), developed by the *Instituto de SaúdeColetiva*, of *Universidade Federal da Bahia*, with the support of *Cenepi – Centro Nacional de Epidemiologia*/Ministry of Health. The study is characterized as a cross sectional study, population based, performed between 1999 and 2000, in a sample of adults, of both genders, of ages equal to or over 20 years, residing in permanent domiciles in the city of Salvador, Bahia. Pregnant women were excluded. The area of influence was defined based on a sample

of a study on health conditions performed in Salvador, described previously (Teixeira *et al*., 2002). A sample was selected by clusters in two stages:

First stage: simple random sampling of 2,305 residences without replacement, based on the register of 16,592 domiciles of the mentioned study(Teixeira *et al*., 2002).

Second stage: after registering all the adults, one woman and one man were selected, by draw, of ages equal to or over 20 years. The theoretical sample size for the domiciles was estimated according to expected prevalence of arterial hypertension of 15%, of arterial coronary disease of 5%, obesity of 30% and diabetes of 7%. A variation coefficient of 5% was used, for a confidence interval of 95%. In view of the existence of partially performed interviews and unrealized interviews in the domiciles, the weights of the respective domiciles were calculated. Nevertheless, these were not considered in the statistical analysis once some units presented negative weights, due to the differentiated allocation of the domiciles in relation to the sample definition. Data production occurred through face-to-face interviews with the application of a structured instrument and measurement procedures for biological measurements. The questionnaire encompassed sociodemographic, physical activity, food, smoking, alcohol consumption and personal health information. Blood pressure was measured using previously validated OMRON HEM 705 CP digital equipment and "adult and large standard" arm cuffs (O'Brien *et al*., 1996). Measurements were taken from the left arm, with the individual seated, divided into two blocks of three (total six measurements): the first block, after at least 5 minutes of rest, and the second block with at least 20 minutes of interval from the first measurement, with the individual remaining seated during the interval. The material for blood exams was collected in selected health centers, using standard techniques (Myers *et al*., 1989) as follows: glycaemia, Labtest technique; hdl-cholesterol; Labtest technique; triglycerides, modified Soloni technique.

Anthropometric measurements were performed, with the individual barefoot and wearing light clothing. To measure waist circumference, defined as the midpoint between the bottom edge of the bottom rib and the top of the iliac crest using a standard tape measure, of synthetic material. The interviews were performed by interviewers with minimum schooling level of complete secondary education and prior experience in field research, selected and trained for this purpose. For quality control a subsample of 10% of the interviewees was selected for retesting of the questionnaire and measurements.For the formation of the databank the EPIINFO version 6.04b software was used and for analysis the STATA version 8. In the analysis, the dependent variable is the Metabolic Syndrome, defined in accordance with the criteria of the International Diabetes Federation. Accordingly, the metabolic syndrome was considered when, in the presence of abdominal obesity(José *et al*., 2006) – two or more of the following criteria were fulfilled: arterial hypertension, hyperglycemia; hypertriglyceridemia; hypoalphalipoproteinemia. Hypertension was defined by the average of the six systolic arterial pressure (SAP) measurements, greater or equal to 130 mmHg, or of the diastolic arterial pressure (DAP), greater or equal to 85 mmHg; hyperglycemia was defined by the fasting glycaemia of 9 hours or more, of values equal to or greater than 100 mg/dl; hypertriglyceridemia by blood triglycerides, obtained after 12 hour or more fasting, of values greater than or equal to 150mg/dl; and hypoalphalipoproteinemia due to low hdl-cholesterol, of values for men lower than 40 mg/dl and for women lower than 50 mg/dl. Values below these of reference were included for individuals under regular treatment for hypertension, hyperglycemia and dyslipidemia.

The main independent variable was depression/anxiety – a composite indicator, defined from the combination of the variables that assess the presence of depression and/or anxiety, under the following categories: 1 –lack of anxiety and depression; 2- presence of anxiety or depression, or both. For its definition the subscale Psychosomatic-Anxiety-Depression (PSAD) was used, of 12 items of depression and anxiety from the Adult Psychiatric Morbidity Survey (APMS). All of the items referred to the situation, at the time of the study, and those related to the prior 12 months. Cases of anxiety were defined as those presenting scores equal of superior to 18, on the PSAD/APMS, and who report at least one of the specific symptoms for depression. Further details and already validated data have already been published in prior studies (Almeida FIlho et la., 2004; Andreoli *et al*., 1994). The co-variables analyzed were: age, self-referred race/color (white, brown, black and others), schooling, recent migration, marital status, menopause, smoking, alcohol consumption and sedentary lifestyles. Data analysis involved initial description of the variables of interest, with the purpose of characterizing the population of the study. Differences between proportions were tested as to the statistical significance using the Pearson χ^2 with a 5% level. Prevalence was calculated as a measurement of occurrence, and prevalence ratio (PR) as a measurement of association.

In the stratified analysis, the Mantel-Haenszel method was used for estimating the prevalence ratio, confidence intervals and verification of strata differences (considering a p value of ≤ 0.05). Identification was performed of effect modifiers, through the numerical observation of the stratum-specific measurements, in relation to the confidence interval of the opposed strata. For the identification of probable confounding factors, the parameter used was the difference of approximately 10% for comparison of the gross association measurement with the adjusted association measurement using the Mantel-Haenszel method. Unconditional logistic regression was used for obtaining estimates of the association measurements based on maximum likelihood, with the objective of testing the hypothesis under study. The backward strategy was used for selection of the variables; including in the model all of the variables obtaining a p value $\leq 0.25\%$ in the univariate tests. However, to remain in the model the associations must present a level of statistical significance ≤ 0.10.In order to verify the existence of change of effect, the likelihood ratio test was used in the assessment of the differences in deviations, comparing the complete model (with product terms), with the reduced model (without product terms) for an alpha of ≤ 0.05. Confounding variables considered were those than when removed from the complete model, produced a statistically significant difference in the one-off measures of the main association, considering confidence intervals of an alpha ≤ 0.10.. The prevalence ratio was automatically calculated by the Stata software, resulting in a corrected OR for high prevalence(Rothman, Greenland, & Lasch, 2011; Horsten *et al*., 1999). The study fulfilled the requirements of the National Research Ethics Committee (Conepe), including the right to refusal in any phase of the study. Confidentiality and privacy of information was

guaranteed, as well as access to the results of the exams and priority in the attendance of individuals with complaints or abnormal laboratory exams, in the primary healthcare units of reference for the project. The study was approved by the Ethics Committee of the Regional Medical Council for the State of Bahia.

RESULTS

The effective sample comprised 1,503 residential units, with 90.2% of the homes having been visited. Out of the 2,442 eligible individuals, 94.4% were interviewed; of these, 1,333 (58.0%) participated in all the phases, including exams and measurements, comprising the population of this study. The individuals dismissed, compared to those examined, were most frequently young men, white, not working at the time of the interview, without prior diagnosis of arterial hypertension and of hyperglycemia. The prevalence for depression/anxiety was of 22.5% among women and of 9.1% among men (p= 0.000) (graph 1).

In both genders, the bearers of depression/anxiety were most frequently married or widow(er)/separate/divorced. Among the men, inactivity was most frequent ($p \leq 0.05$) (table 1). The gross prevalence ration between depression/anxiety and MS, or its components, among women did not demonstrate any statistical significance, being equitably distributed in the unit For men, despite a trend to the distribution of measures to the right, the interval included the unit, not constituting a statistical significance (Fig.2). In the stratified analysis, for both genders, there was no significant association in relation to the co-variables (table 2). In logistic regression, the association of depression/anxiety, when adjusted by the two variables that were maintained in the model as confounders, did not present statistical significance for women, with a PR of 0.95 (CI 95% 0.64-1.38). However for men, this adjusted prevalence ration was modified with values of 2.35 (CI 95% 1.05 -3.77), becoming statistically significant. Among all of the co-variables analyzed, those that were maintained in the model as confounders were age and marital status and, among men, race/color in addition.

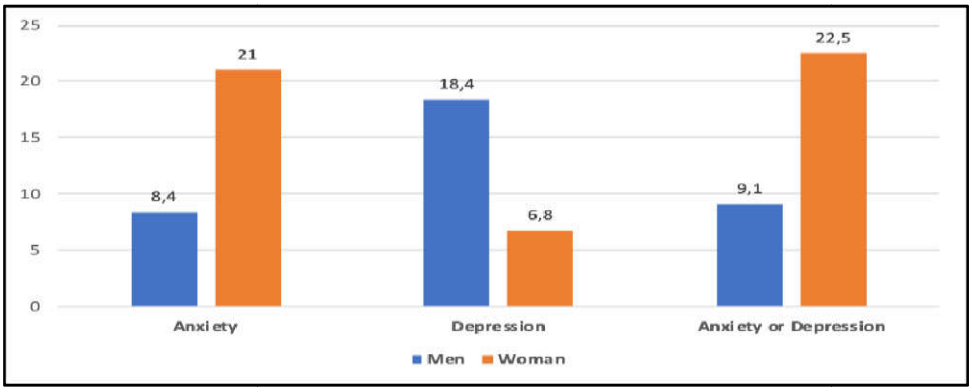


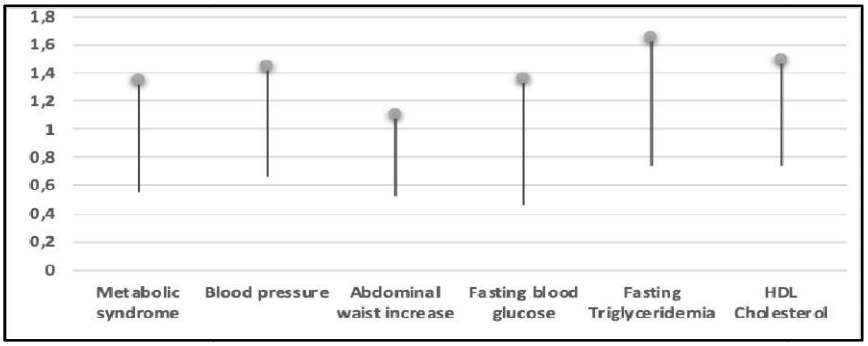
Figure 1. Prevalence of Depression or Anxiety by sex, Salvador, BA, 1999-2000

Table 1. Sociodemographic characteristics of the population of the study in relation to depression or anxiety in men and women. Salvador, BA, 1999-2000

Co-variables	Women		Men	
	No (n=537) (%)	Yes (n=158) (%)	No (n=49) (%)	Yes (n=481) (%)
Age	*p=0.090		*p=0.162	
20 – 49	71.7	78.5	78.2	69.4
50 and over	28.3	21.5	21.8	30.6
Race/Color	*p=0.081		*p=0.070	
White	14.3	7.6	12.1	10.2
Mixed	65.0	70.9	64.2	79.6
Black	20.7	21.5	23.7	10.2
Schooling	*p=0.325		*p=0.069	
Incomplete primary education	60.2	60.1	56.8	71.4
Complete primary education	17.5	13.3	20.8	8.2
Complete Secondary/Higher education	22.4	26.6	22.4	20.4
Smokers	*p=0.074		*p=0.140	
Yes	16.0	22.2	26.8	36.7
No	84.0	77.8	73.2	63.3
Recent migration	*p=0.646		*p=0.186	
Yes (< 10 years)	8.8	7.6	10.2	16.3
No	91.3	92.4	89.8	83.7
Menopause	*p=0.324			
Yes	32.8	28.6	...	...
No	67.2	71.3	...	...
Marital status	*p= 0.006		*p= 0.007	
Single	21.0	22.8	26.4	6.1
Married	62.8	58.9	70.3	89.8
Widow(er)/ Separate/Divorced	16.2	18.4	3.3	4.0
Alcohol consumption	*p=0.036		*p=0.747	
Light and moderate	40.6	50.0	61.5	59.2
Absent and excessive	59.4	50.0	38.5	40.8
Sedentary lifestyle	*p=0.178		*p=0.050	
Yes	75.9	70.5	74.5	60.9
No	24.1	29.5	25.5	39.1

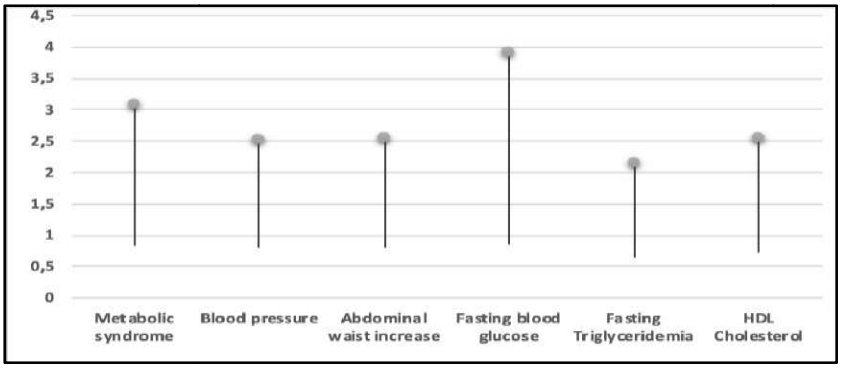
p-valor obtained through the Pearson χ^2 test

Women



Note: Abdominal waist increase ≥ 84

Men



Note: Abdominal waist increase ≥ 88

Figure 2. Prevalence ratio of metabolic syndrome and its constituents by sex according to depression or anxiety, Salvador, BA, 1999-2000

Table 2 Stratified analysis and logistic regression for association between depression or anxiety and the metabolic syndrome, Salvador, BA, 1999-2000

Variables	Women				Men			
	n	Prev. (%)	PR*	PR*$_{Adjusted}$	n	Prev. (%)	PR*	PR*$_{Adjusted}$
Depression or anxiety								
No	537	20.7	1.00	1.00	481	18.5	1.00	1.00
Yes	158	18.4	0.89 (0.61-1.28)	0.95 (0.64-1.38)	49	26.5	1.43 (0.87-2.37)	2.35 (1.05-3.77)
Age (years)								
20 – 49	509	12.9	0.89 (0.53-1.50)	1.00	410	17.6	1.12 (0.52-2.41)	1.00
50 and over	186	38.2	1.06 (0.66-1.70)	2.16 (1.58-2.85)	120	46.7	1.63 (0.88-3.03)	1.63 (1.08-2.35)
Adjusted		0.96 (0.67-1.37)					1.34 (0.82-2.20)	
Race/Color								
White	89	16.7	0.99 (0.25-3.84)		63	60.0	2.9 (1.21-6.96)	1.00
Mixed and Black	606	18.5	0.87 (0.59-1.27)		467	22.7	1.25 (0.70-2.23)	2.54 (1.50-3.73)
Adjusted		0.88 (0.61-1.27)					1.44 (0.87-2.37)	
Schooling								
Complete Secondary/Higher education	162	16.7	1.33 (0.58-3.04)		118	20.0	0.86 (0.24-3.13)	
Complete Primary education	115	9.5	0.99 (0.23-4.27)		104	25.0	1.67 (0.29-9.69)	
Incomplete Primary education	418	21.1	0.78 (0.51-1.20)		308	28.6	1.60 (0.89-2.85)	
Adjusted		0.88 (0.61-1.27)					1.41 (0.85-2.34)	
Smoker								
No	574	17.1	0.86 (0.56-1.32)	1.00	383	22.6	1.19 (0.60-2.36)	
Yes	121	22.9	0.94 (0.46-1.91)	1.43 (0.97-2.00)	147	33.3	1.95 (0.92-4.16)	
Adjusted		0.87 (0.60-1.26)					1.44 (0.87-2.40)	
Recent migration								
No	636	19.2	0.89 (0.62-1.30)		473	22.0	1.13 (0.61-2.07)	
Yes (< 10 years)	59	8.3	0.65 (0.09-4.92)		57	50.0	4.9 (1.66-14.45)	
Adjusted		0.88 (0.61-1.28)					1.46(0.87- 2.45)	
Marital status								
Single	149	13.9	1.43 (0.53-3.83)	1.00	130	0.0	...	1.00
Married	430	14.0	0.72 (0.42-1.26)	1.62 (1.01-2.41)	382	29.5	1.25 (0.76-2.05)	2.09 (1.24-3.20)
Divorced/Separate/Widow(er)	116	37.9	0.94 (0.55-1.60)	2.40 (1.54-3.34)	18	0.0	...	...
Adjusted		0.87 (0.61-1.25)					1.22 (0.74-2.00)	
Menopause								
No	472	10.7	0.76 (0.42-1.37)					
Yes	221	37.8	1.13 (0.73-1.73)					
Adjusted		0.94 (0.66-1.34)						
Alcohol consumption								
Light and moderate	297	16.5	1.12 (0.62-2.02)	1.00	325	27.6	1.57 (0.83-2.98)	
Absent and excessive	398	20.2	0.82 (0.51-1.32)	1.33 (0.95-1.82)	205	25.0	1.25 (0.55-2.82)	
Adjusted		0.92 (0.64-1.34)					1.43 (0.86-2.36)	
Sedentary lifestyle								
No	172	13.0	0.86 (0.37-2.03)		112	33.3	2.85 (1.21-6.71)	1.00
Yes	506	20.9	0.93 (0.62-1.40)		302	25.0	1.00 (0.51-1.98)	2.09 (1.23-3.20)
			0.92 (0.63-1.32)				1.41 (0.84-2.37)	

Adjusted for age, marital status, race/floor and sedentary lifestyle. *CI 95%.

DISCUSSION

No other study was identified with a cross-sectional population base, assessing adults, considering lesser mental disorders, self-referred, measured in a direct manner (anxiety/depression) in association to MS. The studies that assessed as MS, or its components, as an outcome were mostly performed with women, considering as exposure social relations (Horsten *et al*., 1999), family income (Dallongeville *et al*., 2005), schooling levels (Wamala *et al*., 1999), and the quality of the marital status(Radloff, 1997), all demonstrating a strong association between social isolation low family income, low schooling levels and dissatisfaction with the marital relationship with MS. Four studies were found that assessed stress, depression/anxiety, in a direct manner through a subjective questionnaire. The Brunner & col. article demonstrated (Brunner *et al*., 2002), in a case-control study, exclusively with men, nested in a Whitehall II cohort, that chronic stress is associated to MS, validated through saliva cortisol dosage. In other cohorts, Herva& col. (Herva *et al*., 2002)did not identify any association between depression/anxiety and MS, in men or women. However, the research by Raikkonen & col. (Räikkönen, Matthews, & Kuller, 2002), studying only women, demonstrated that the feeling of anger is associated to MS. In the only cross-sectional study, with men and women, Kinder & col.(Kinder *et al*., 2004) demonstrated that among women with episodes of depression there was a greater association to MS.

In the present study, the association between depression/ anxiety and MS was determined only for men. The associations described in literature demonstrate that lesser mental disorders such as depression is the association most found in women and anxiety or chronic stress most found in men (Radloff, 1997; Almeida-Filho*et al*., 2004; Rosenfield, 1980). One of the explanations would be that the responses of the stress hormones are very different in men and women. It seems that this diversity is not linked to biological factors, but to psychological aspects and to the different roles related to gender(Kautzky-Willer, Harreiter, &Pacini, 2016). Women present much less chronic stress, at work, when compare to men(Chandola, Brunner, & Marmot, 2006). This could explain the different magnitudes of association, in this investigation. These deferent aspects were not analyzed in this research. However, maybe in this population there could be more men working or unemployed that would have greater probability of presenting anxiety. Another possibility is of the choice of criteria of FID for the definition of MS. All of the articles described used the ATPIII criteria, which when compared to the FID, decreases in 4.5% to 4.7% the prevalence of MS (Ford, 2005). This would result in lower specificity for the strength of the association in registering significant results. Another issue would be the losses presented, with a higher concentration of older women, concentrated in the MS, directing the association among the women to the null hypotheses. There was an important difference between the prevalence of lesser mental disorders amount men, with 9.1% and among women of 22.5%. This information is in in accordance with various studies(Radloff, 1997; Almeida-Filho *et al*., 2004; Rosenfield, 1980). Longitudinal studies are recognizably those that present greater power of causal interferences, thus, of the four cohort studies that tested lesser mental disorders and/or chronic stress and MS, only one demonstrated contradictory result. In one same cohort obtained a cause-effect relationship between referable psychological risk for chronic stress and MS, respectively (Räikkönen, Matthews, & Kuller, 2002; Chandola, Brunner, & Marmot, 2006; Steptoe, Brunner, & Marmot M, 2004). In accordance with the investigation about social gradient in MS is partially explained by chronic stress at work, and in a large proportion, by health behaviors (smoking sedentary lifestyles, excess alcohol intake and poor diets) (Chandola, Brunner, & Marmot, 2006; Steptoe, Brunner, & Marmot M, 2004). According to these and other authors, these health behaviors may predict the risk of MS for mean and less consistently for women. This fact was also found in this investigation and in the prior article on sedentary lifestyles and MS (Brunner, Marmot, 2005).

On the other hand, in the cohort was not possible do show any association, one of the possibilities for the absence of an association could be the population of younger individuals, than in the present investigation (Herva *et al*., 2006). Nevertheless, there are sufficient trial and clinical bases, as proposed herein. In any event, the difficulty for evidencing associations, as proposed in this investigation, is clear. However, there are sufficient trial and clinical bases association risk factors, diseases and cardiovascular deaths and activation of the neuroendocrine axes, either from the autonomic nervous system (ANS) or from the hypothalamic pituitary adrenocortical system (HPA)(Hemingway, & Marmot, 1999; Stansfeld, & Marmot, 2002). It should rest with subsequent investigations to try and grasp this relation in a population level, as well as the effect of lesser mental disorders, and of lifestyle stresses (Bonnet, Irwing, & Terra, 2005). For example, overweight people, with increased waist circumference, may become sedentary and socially isolated, with the consequence of perceiving life in a more stressful manner, leading to mental disorders. It is known that light and moderate physical activities, performed regularly, release anti-stress hormones and substances such as endorphins and dopamine and serotonin(Hemingway, & Marmot, 1999; das Merces *et al*., 2019).One study demonstrated a block of responses from the serotoninergic system in sedentary people with MS (Bonnet, Irwing, & Terra, 2005; Muldoon *et al*., 2004). It is important to mention that the variables such as marital status and race/color, the confounders found, should be further studied including the role of subjective perception of racial discrimination(Kessler, & Neighbors, 1986). The higher association in black and mixed-race people, when compared to white cardiovascular risk factors among men demonstrated in literature in relation to arterial hypertension(Din-Dzietham *et al*., 2004) and diabetes (Brown et al., 2004). Additionally this association could involve conditions such as less access to healthcare (Clark, & Harrel, 1982).

REFERENCES

Almeida-Filho, N., Lessa, I., Magalhães, L., Araújo, M. J., Aquino, E., & James, S. A., *et al*. (2004). Socialinequality in depressive disorders in Bahia: interactions of gender, ethnicity, and social class. Soc Sci Med, 59(7), 2339–1353.

Almeida-Filho, N., Lessa, I., Magalhães, L., Araújo, M. J., Aquino, E., & Kawachib, I., *et al*. (2004). Alcohol drinking patterns by gender, ethnicity, and social class in Bahia, Brazil. Rev Saúde Pública, 38(1), 45–54.

Andreoli, S. B., Mari, J. J., Blay, S. L., Almeida-Filho, N., Coutinho, E., & França, J., *et al*. (1994). Estrutura fatorial do questionário de morbidade psiquiátrica de adultos

aplicando em amostras populacionais de cidades brasileiras. Rev Saude Publica, 28(4), 249–60.

Björntorp, P. (2001). Do stress reactions cause abdominal obesity and comorbidities ? Obes Rev, 2(2), 73–86.

Bonnet, F., Irwing, K., & Terra, J. (2005). Depressive symptoms are associated wuit unhealthy lifestyles in hypertensive patients with the metabolic syndrome. J Hypertens, 23, 611–7.

Bouchard, C., Despres, J., Mauriege, P. (1993). Genetic and nongenetic determinants of regional fat distribution. Endocr Rev, 14(1), 72–93.

Brown, A. F., Ettner, S. L., Piette, J., Weinberger, M., Gregg, E., & Shapiro, M. F., *et al*. (2004). Socioposition and health among persons with diabetes mellitus: a conceptual framework and review of the literature. Epidemiol Rev, 26, 63–77.

Brunner, E. J., Hemingway, H., Walker, B. R., Page, M., Clarke, P., & Juneja, M., *et al*. (2002). Adrenocortical, Autonomic, and Inflammatory Causes of the Metabolic Syndrome. Circulation, 106(21), 2659–65.

Brunner, E., & Marmot, M. (2005). Social organization, stress, and health. In: Social Determinants of Health. 2a. EUA: Oxford University Press. p. 376.

Chandola, T., Brunner, E., & Marmot, M. (2006). Chronic stress at work and the metabolic syndrome: Prospective study. Br Med J, 332(7540), 521–4.

Clark, V., & Harrel, J. (1982). The relationship among type A behavior, styles used coping with racism, and blood pressure. J Black Psych, 8, 89–99.

Dallongeville, J., Cottel, D., Ferrières, J., Arveiler, D., Bingham, A., & Ruidavets, J. B., *et al*. Household income is associated with the risk of metabolic syndrome in a sex-specific manner. Diabetes Care, 28(2), 409–15.

Das Merces, M. C., Santana, A. I. C., Lua, I., da Silva, D. A. R., Silva, D. S., & Gomes, A. M. T, *et al*. (2019). (Metabolic Syndrome Among Primary Health Care Nursing Professionals: A Cross-1999). Sectional Population-Based Study. Int J Environ Res Public Health, 16(15), 2686.

Das Merces, M. C., Souza, D. D., Lua, I., Oliveira, D. S., Souza, M. C., & Júnior, A. D. O. (2016). Burnout syndrome and abdominal adiposity among Primary Health Care nursing professionals. Psicol Reflexão e Crítica, 29(44). Available at: http://dx.doi.org/10.1186/s41155-016-0051-7

Din-Dzietham, R., Nembhard, W. N., Collins, R., & Davis, S. K. (2004). Perceived stress following race-based discrimination at work is associated with hypertension in Africa-Americans. The Metro Atlanta Heart Study, 1999-2001. Soc Sci Med, 58(3), 449–61.

Feldman, P. J., & Steptoe, A. (2003). Psychosocial and Socioeconomic Factors Associated With Glycated Hemoglobin in Nondiabetic Middle-Aged Men and Women. Heal Psych, 22(4),398–405.

Ford, E. A. (2005). Prevalence of the metabolic syndrome defined by the international diabetes federation among adults in the U.S. Diabetes Care, 28, 2724–49.

Frankenhaeuser, M., Lundberg, U., Fredrikson, M., Melin, B., Tuomisto, M., & Myrsten, A., *et al*. (1989). Stress on and off the job as related to sex and occupational status in white-collar workers. J Org Behav, 10(4), 321–46.

Hemingway, H., & Marmot, M. (1999). Psychosocial factors in the a etiology and prognosis of coronary heart disease: systematic reviews of prospective cohort studies. Br Med J, 318(7196), 1460–7.

Herva, A., Räsänen, P., Miettunen, J., Timonen, M., Läksy, K., & Veijola, J., *et al*. (2006). Co-occurence of metabolic syndrome with depression and anxiety in young adults: the northern Finland 1966 birth cohort study. Psychosom Med, 68(2), 316–22.

Horsten, M., Mittleman, M. A., Wamala, S. P., Schenck-Gustafsson, K., & Orth-Gomér, K. (1999). Social Relations and the Metabolic Syndrome in Middle-Aged Swedish Women. J Cardiovasc Risk, 6(6), 391–7.

Jones-Webb, R., Jr, D. R. J., Flack, J. M., & Liu, K. (1996). Relationships between depressive symptoms, anxiety, alcohol consumption, and blood pressure. Results from the CARDIA study. Alcohol Clin Exp Res, 20(3), 420–7.

José, P., Barbosa, B., Lessa, Í., Filho, N. D. A, Batista, L., & Magalhães, N. C., *et al*. (2006). Criteria for Central Obesity in a Brazilian Population : Impact on the Metabolic Syndrome. Arq Bras Cardiol, 87, 366–73.

Kautzky-Willer, A., Harreiter, J., & Pacini, G. (2016). Sex and Gender Differences in Risk, Pathophysiology and Complications of Type 2 Diabetes Mellitus. Endocr Rev, 37(3), 278–316.

Kessler, R. C., & Neighbors, H. W. (1986). A new perspective on the relationships among race, social class and psychological distress. J Heal Soc Behav, 27(2), 107–15.

Kinder, L. S., Carnethon, M. R., Palaniappan, L. P., King, A. C., & Fortmann, S. P. (2004). Depression and the metabolic syndrome in Young adults: Findings from the third national health and Nutrition examination survey. Psychosom Med, 66(3), 316-322.

Kirkwood, B. R., Sterne, J. (2003). Essentials of Medical Statistics. 2o ed. Publishers B, organizador, p. 501.

Knox, S. S., Jr, D. R. J., Chesney, M. A., Raczynski, J., & McCreath, H. (1996). Psychological factors and plasma lipids in black and white young adults: The coronary artery risk development in young adults study data. Psychosom Med, 58(4):373–465.

Licinio, J., Yildiz, B., & Wong, M. L. (2002). Depression and cardiovascular disease: co-occurence or shared genetic substrates? Mol Psychiatry, 7, 1031–2.

Mottillo, S., Filion, K. B., Genest, J., Joseph, L., Pilote, L., & Poirier, P., *et al*. (2010). The metabolic syndrome and cardiovascular risk: A systematic review and meta-analysis. J Am Coll Cardiol, 56(14), 1113–32. Available at: http://dx.doi.org/10.1016/j.jacc.2010.05.034

Muldoon, M. F., Mackey, R. H., Williams, K. V., Korytkowski, M. T., Flory, J. D., & Manuck. S. B. (2004). Low central nervous system serotonergic responsivity is associated with metabolic syndrome and psysical inactivity. J Clin Endocrinol Metab, 89(1), 266–71.

Myers, G. L., Cooper, G. R., Winn, C. L., & Smith, S. J. (1989). The Centers for Disease Control-National Heart Lung and Blood Institutes Lipid Standardization program. An approach to accurate and precise lipid measurements. Clin Lab Med, 9(1), 105–35.

O'Brien, E., Mee, F., Atkins, N., & Thomas, M. (1996). Evaluation of three devices for self-measurement of blood pressure according to the revised British Hypertension Society Protocol: The Omron HEM-705CP, Philips HP5332, and Nissi DS-175. Blood Pressure Monitoring. Blood Press Monit, 1(1), 55–61.

Petrlova, B., Rosolva, H., & Al, Z. (2004). Depressive disorders and the matabolic syndrome of the insulin resistance. Semin Vas, 4, 161–5.

Radloff, L. S. (1977). Sex differences in depression: the effects of occupation and the marital status. Sex Roles, 1(10), 249–65.

Räikkönen, K., Matthews, K. A., & Kuller, L. H. (2002). The relationship between psychological risk attributes and the metabolic syndrome in healthy women: Antecedent or consequence? Metabolism, 51(12), 1573–7.

Rosenfield, S. (1980). Sex differences in depression: do women always have higher rates? J Heal Soc Behav, 21, 33–42.

Rosengren, A., Hawken, S., Ôunpuu, S., Sliwam, K., Zubaid, M., & Almahmeed, W. A., *et al*. (2004). Association of psychosocial risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the INTERHEART study): case-control. Lancet, 364(938), 953–62.

Rosmond, R., Eriksson, E., & Björntorp, P. (1999). Personality Disorders in Relation to Anthropometric, Endocrine and Metabolic Factors. J Endocrinol Invest, 22(4), 279–88.

Rothman, K. J., Greenland, S., & Lash, T. L. (2011). Epidemiologia Moderna. 3a. Porto Alegre: Artmed, p. 888.

Stansfeld, S., & Marmot, M. (2002). Introduction. In: Books, B., org. Stress and the Heart: Psychosocial Pathways to Coronary Heart Disease. London, p. 304.

Steptoe, A., Brunner, E., & Marmot, M. (2004). Stress-induced inflammatory response and risk of the metabolic syndrome: a longitudinal analysis. Obes Res, 12, A76.

Teixeira, M. G., Barreto, M. L., Costa, M. C. N., Strina, A., Júnior, D. M., & Prado, M. (2002). Sentinel areas : a monitoring strategy in public health Áreas sentinelas : uma estratégia de monitoramento em saúde pública. Cad Saude Publica, 18(5), 1189–95.

Thomas, A. J., Kalaria, R. N., & O´Brien, J. T. (2004). Depresion and vascular disease: What is the relationship? J Affect Disor, 79, 81–95.

Wamala, S. P., Lynch, J., Horsten, M., Mittleman, M. A., Schenck-Gustafsson, K., & Orth-Gomér, K. (1999). Education and the metabolic syndrome in women. Diabetes Care, 22(12), 1999–2003.

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