Received: 6 November 2020 | Revised: 24 December 2020 | Accepted: 3 January 2021

DOI: 10.1111/jch.14190

SHORT REPORT

WILEY

Acute physical and mental stress resulted in an increase in fatty acids, norepinephrine, and hemodynamic changes in normal individuals: A possible pathophysiological mechanism for hypertension—Pilot study

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Abstract

Hypertension is often associated with metabolic changes. The sustained increase in sympathetic activity is related to increased blood pressure and metabolic changes. Environmental stimuli may be related to increased sympathetic activity, blood pressure, and metabolic changes, especially in genetically predisposed individuals. The aim of this study was to evaluate the response of fatty acids to physical and mental stress in healthy volunteers and the hemodynamic, hormonal, and metabolic implications of these stimuli. Fifteen healthy individuals with a mean age of 31 ± 7 years, of both sexes, were evaluated. They were assessed at baseline and after combined physical and mental stress (isometric exercise test, Stroop color test). Blood samples were collected at baseline and after stimulation for glucose, insulin, fatty acid, and catecholamine levels. Blood pressure, heart rate, cardiac output, systemic vascular resistance, and distensibility of the large and small arteries were analyzed. The data obtained at baseline and after stimuli were from the same individual, being the control itself. Compared to baseline, after physical and mental stress there was a statistically significant increase ($p < .05$) in free fatty acids, norepinephrine, diastolic blood pressure, peripheral vascular resistance, and distensibility of the large and small arteries. In conclusion, the combination of physical and mental stress raised fatty acids, norepinephrine, diastolic blood pressure, and peripheral vascular resistance in healthy individuals.

1 | INTRODUCTION

Primary arterial hypertension has as its pathophysiological mechanism the activation of the sympathetic nervous system.[1] Genetically predisposed individuals when interacting with the environment (stress and salt) develop acute and chronic changes that will contribute to increased sympathetic activity and, consequently, to sustained elevation of blood pressure (BP).[2] In some stress situations, adrenergic hormones (norepinephrine, adrenaline) are released and nitric oxide production and activity inhibited, resulting in vasoconstriction,

endothelial dysfunction, and increased BP.[3] In adapting to the environment, the human being encounter physical and mental stress. Handgrip is a type of isometric (physical) exercise that causes hemodynamic changes in normotensive and hypertensive individuals. Training with the handgrip repeatedly during the week can lower BP in normotensive and hypertensive individuals.[4,5] However, isometric exercise with handgrip acutely raises blood pressure in normotensive individuals and in children of hypertensive parents.[6–8] The increase in BP in the children of hypertensive patients in the study by Lopes et al[8] occurred in the presence of increased muscle sympathetic nerve activity. There are different levels of body adaptation to a threat, one of which results in autonomic and endocrine activation. Sympathetic activation (SA) can result in hemodynamic and metabolic changes.[9] In addition to the hemodynamic changes found in primary hypertension, related to SA, metabolic changes are relatively frequent.[10] According to the literature, mental stress is mediated predominantly by beta-adrenergic receptors, while physical stress is mediated predominantly by alpha-adrenergic receptors.[11] The release of fatty acids from visceral fat is mainly related to beta-adrenergic receptors, more specifically the beta 3 receptors.[12] As hypertension has a syndromic characteristic (hemodynamic and metabolic alterations), we understand that the concomitant physical and mental environmental stimuli can activate beta- and alpha-adrenergic receptors resulting in hemodynamic and metabolic changes. The metabolic changes found in hypertensive patients and in children of hypertensive parents may reflect the autonomic imbalance resulting from undesirable stimuli in the environment. We propose that the combination of physical and mental stress on a recurring basis activates the hypothalamic-adrenal pituitary axis resulting in hemodynamic and metabolic changes, especially in individuals predisposed to arterial hypertension. The re-exposure to unpleasant stimuli from the environment can result in greater activation of the autonomic nervous system (ANS), sympathetic component, and consequently in the acute increase of fatty acids. It is known that the acute increase in fatty acids in the circulation (Intralipid® infusion) results in hemodynamic changes such as increased BP. In a previous study, we demonstrated that the acute elevation of free fatty acids (Intralipid® infusion) results in a greater increase of BP in normotensive individuals born to hypertensive parents than those born to normotensive parents.[13]

Given the importance of the ANS in hemodynamic and metabolic modulation, the primary objective of this pilot study was to test whether acute physical and mental stress raises fatty acids and BP in healthy volunteers. The secondary objective was to assess the acute effect of the combination of physical and mental stress on catecholamine levels, glucose metabolism, and basal metabolic rate in healthy volunteers.

O objetivo secundário foi avaliar o efeito agudo da combinação do exercício físico e mental nos níveis de catecolaminas, metabolismo de glicose e taxa metabólica basal.

The secondary objective was to assess the acute effect of the combination of physical and mental stress on catecholamine levels, glucose metabolism, and basal metabolic rate.

2 | POPULATION AND METHODS

2.1 | Study population

Study volunteers were recruited through posters in a public hospital in São Paulo. We initially evaluated 17 male and female volunteers, aged 18–42 years, white and non-white. Two volunteers were excluded for not meeting inclusion criteria. The 15 volunteers (9 female and 6 male) underwent a clinical evaluation that included medical history, complete physical examination, and anthropometric data. All study participants signed an Informed Consent Form (ICF) according to Resolution No. 466/12 which was approved by the Institutional Review Board. The study included volunteers who met the inclusion and exclusion criteria listed below:

2.1.1 | Inclusion criteria

Individuals of both sex, white and non-white, body mass index (BMI) less than 30 kg/m^2, age between 18 and 50 years, BP less than 140/90 mm Hg, LDL cholesterol less than 130 mg/dl, triglycerides less than 150 mg/dl, normal renal function, and no systemic disease.

2.1.2 | Exclusion criteria

Hypertensive volunteers, valvular heart disease, use of cardiac pacemaker, cardiac arrhythmia, patients with hyperthyroidism and hypothyroidism, collagen disease, Crohn and celiac disease, cancer or disabling chronic disease, smokers, diabetics, and BMI greater than or equal to 30 kg/m^2.

2.2 | Methods

After signing an Informed Consent Form (ICF) and initial clinical evaluation, participants were instructed regarding the research procedures. All of them underwent anthropometric evaluation, blood collection for biochemical tests, and noninvasive hemodynamic evaluation. They underwent physical and mental stress after 30 min of rest. Physical and mental stress was performed one immediately after another. Physical stimulus was always before the mental stimulus.

2.3 | Anthropometric assessment

Height was measured using a fully aligned Sanny® anthropometric measuring tape extended on a flat, skirting wall with a flat, rigid floor and angled to the wall to properly support the tape. At the time of obtaining the measure, the volunteer was asked to hold his breath. The value in cm with precision of mm was obtained.

Weight was assessed on a Plenna® digital scale, with a unit of measurement in kg with 100 g accuracy and having a measuring

capacity in the range from 0.0 kg to 150 kg. Body mass index was calculated using the Quételet formula where weight in kilograms is divided by height in meters squared.

2.4 | Body composition assessment—Bioimpedance

Bioimpedance (BIA 405, USA) was used to measure body fat and lean mass, phase anglo and basal metabolic rate in volunteers at baseline and after physical and mental stress (handgrip and color test). The idea was to evaluate the impact of physical and psychic stimuli on the basal metabolic rate.

2.5 | Clinical evaluation and biochemical tests

After anthropometric evaluation, the volunteers underwent clinical evaluation and performed biochemical tests. The volunteers came to the Clinical Research Laboratory after 10- to 12-h fasting for blood collection for laboratory tests and noninvasive testing. Blood glucose, urea, creatinine, sodium, potassium, total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides were measured. Biochemical dosages were performed with commercial kits. Total cholesterol, HDL cholesterol, triglycerides, blood glucose, sodium, and potassium were evaluated by enzymatic colorimetric method in automated equipment (Model BioSystems 310), according to the Labtest® kit manufacturer's recommendations. Homeostasis model of insulin resistance (HOMA-IR) was calculated from blood glucose and insulin levels, and the quantitative insulin sensitivity check index (QUICKI) was calculated from insulin and blood glucose levels. Both indexes were calculated using glucose and insulin levels before and after stimuli.

2.6 | Physical and mental stress

Physical and mental stress was always applied in the same sequence for all individuals. First, individuals performed physical stress with a dynamometer (handgrip test) and then mental stress (Stroop color test). The temperature of the room during the tests was maintained at 22°C.

2.7 | Handgrip test

During isometric exercise, participants were instructed to squeeze a handgrip dynamometer to test the maximum voluntary contraction. After that, participants were instructed to squeeze a handgrip dynamometer with 30% of total strength for the dominant arm over 3 min.

2.8 | Color testing

Stroop color test consists of reading a word written in a color other than what it means. It was applied to each individual lasting approximately 10 min. The test consists of three tasks: word reading, color naming, and identifying the color in which each word is written, regardless of its meaning. Reading each word gives an indication of reading fluency and establishes a point of comparison for the color naming task. The fact that there is a discrepancy between the word name and the color of the ink causes an interference effect on color naming.

2.9 | Hemodynamic evaluation

Casual BP was measured once with patients in the sitting position after 5 min of rest using a calibrated aneroid sphygmomanometer. Noninvasive hemodynamic evaluation was performed with the HDI (Hypertension Diagnosis Incorporation, CR2000, Eagan, USA), and participants were in a supine position. The HDI CR2000 is a noninvasive hemodynamic assessment device that provides cardiac output, total vascular resistance, stroke volume, total vascular impedance, large artery distensibility (vascular compliance), and small artery distensibility (endothelial function). The HDI was validated and used in several clinical studies since the 1990s with good reproducibility for hemodynamic parameters.[14]

Hemodynamic evaluation was performed with the volunteer at rest and after physical and mental stress stimuli.

2.10 | Blood samples during study

The volunteers were maintained with venous access to collect blood samples at rest and after stimulation. Glucose, insulin, fatty acids, noradrenaline, and adrenaline were measured at baseline and after stimuli. Glucose was measured by enzymatic colorimetric method in automated equipment (Model BioSystems 310). Insulin was measured by the CIS BioInternational® INSULIN-CT radioimmunoassay method using Abbott® gamma counter (intra-assay coefficient of variation 2.6%). Fatty acids were measured by the semi-automatic colorimetric method with NEFA-Randox® Kit. Noradrenaline and adrenaline were measured by the HPLC (high-performance liquid chromatography) method.

2.11 | Statistical analysis

GRAPHPAD PRISM 8.2.0 and SPSS version 18.0 software were used for statistical analysis. Shapiro-Wilk's test was used to check the normality of the different variables. Samples will be presented as means and/or medians.

The variables evaluated at two moments with normal distribution were compared with the Student *t* test and those that did not show normal distribution were compared with the Wilcoxon test. Results with two-sided *p* values less than .05 were considered statistically significant.

3 | RESULTS

Clinical, anthropometric, hemodynamic, and biochemical data of the volunteers at baseline are presented in Table 1.

Fatty acids increased significantly after physical and mental stimuli (Figure 1). Plasma norepinephrine increased after physical and mental stress, whereas (Figure 2) epinephrine did not change. Insulin (7.4 ± 3.2 vs. 7.8 ± 3.4 µIU/ml), glucose (4.2 ± 0.3 vs 4.1 ± 0.4 mmol/L), HOMA-IR index (1.37 vs. 1.42), and QUICKI index (0.37 vs. 0.37 did not change significantly after physical and mental stimuli).

Acute physical and mental stress increased diastolic BP and peripheral vascular resistance evaluated noninvasively (HDI) in healthy volunteers. There was also a small increase in distensibility of large and small arteries (Table 2).

Observing the variables related to bioimpedance, there was no difference when comparing the values before and after physical and mental stress (Table 3).

4 | DISCUSSION

In this pilot study conducted on healthy volunteers, physical and mental stress increased plasma fatty acids and norepinephrine levels as well as diastolic blood pressure, and peripheral vascular resistance. Insulin and glucose levels did not increase and metabolic rate did not change after physical and mental stimuli.

Paolisso and collaborators demonstrated that the elevation of fatty acids through the infusion of Intralipid® resulted in sympathetic nervous system activation of healthy participants.[15] In our study, we demonstrated that different forms of stress resulted in increased fatty acids and norepinephrine levels. Fatty acid mobilization during moderate exercise and prolonged fasting is well known.[16,17] Increased fatty acids have also been reported experimentally in rabbits subjected to stress.[18] In the 1970s, a study was conducted with dogs showing an increase in free fatty acids after exposure to low temperatures (physical stress).[19] It is known that the release of fatty acids from adipocytes is related to autonomic modulation, especially SA. In our study, physical and mental stress increased plasma norepinephrine, which points to an increase in sympathetic nervous system activity. Epinephrine levels did not

TABLE 1 Demographic, anthropometric, hemodynamic, and biochemistry data

Variable	Mean ± SD
Age (years)	31 ± 7
Sex—*n* (%)	
Male	6 (40%)
Female	9 (60%)
Race—*n* (%)	
White	10 (66.67%)
Non-white	5 (33.33%)
Weight (kg)	73 ± 15
Height (m)	1.7 ± 12.6
Body mass index (kg/m^2)	25 ± 4
Systolic blood pressure (mm Hg)	110 ± 11
Diastolic blood pressure (mm Hg)	64 ± 7
Total cholesterol (mg/dl)	178 ± 38
LDL cholesterol (mg/dl)	102 ± 31
HDL cholesterol (mg/dl)	54 ± 12
Triglycerides (mg/dl)	91 ± 42
Glucose (mg/dl)	81 ± 6

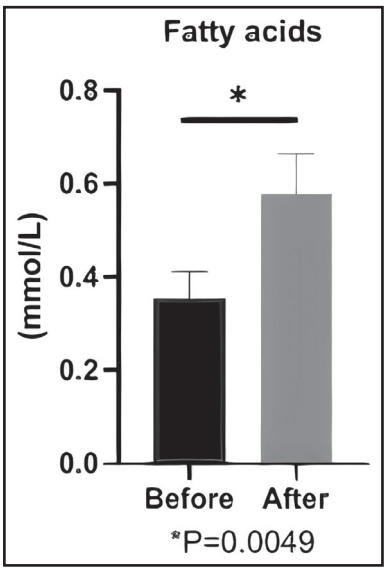

FIGURE 1 Fatty acid levels before and after stimuli (mean ± SE)

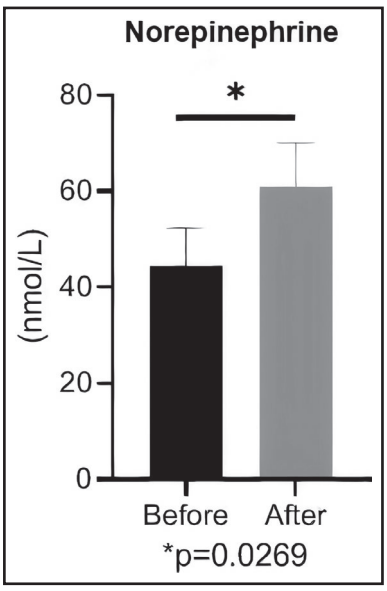

FIGURE 2 Norepinephrine levels before and after stimuli (mean ± SE)

change. This could be interpreted as a possible predominance of physical stress over mental stress, since mental stress further increases adrenaline levels, mediated by the sympatho-adrenal-medullary system.[20] In the study by Meijssen et al,[21] prolonged mental stress increased norepinephrine and epinephrine in healthy individuals. In our study, mental stimulation did not have the same duration and the type of stress was different.

Plasma insulin, glucose, HOMA-IR, and QUICKI index did not change after physical and mental stress. Most studies have so far shown that elevation of fatty acids is related to insulin resistance, that is, after the increase in fatty acids plasma insulin is expected to increase. However, in the study by Pelkonen et al 5- to 7-fold increase in free fatty acid plasma levels did not influence insulin and glucose levels,[22] and we observed the same in our study on participants who were fasting. Catecholamines and insulin are the main regulators of lipolysis. Norepinephrine and epinephrine exert lipolytic effects, and insulin exerts an antilipolytic effect. In the study by Wasserman et al,[23] dogs that performed prolonged exercise, treadmill walking for 150 min, showed increased catecholamines and reduced plasma insulin. In our study, physical and mental stress resulted in increased norepinephrine and insulin levels were unchanged. The increase in norepinephrine, via activation of adipocyte β-adrenoceptors, may explain the increase in fatty acids. The metabolic responses to physical and mental stress are complex and involve many variables. For example, metabolic changes due to physical stress may activate different pathways in the central nervous system and the periphery.

Regarding hemodynamic behavior, physical and mental stress increased diastolic BP, systemic vascular resistance, and slightly increased distensibility of the large and small arteries. Heart rate and cardiac output did not change. Mental stress, as is well known, results in increased heart rate and no change in systemic vascular resistance due to increased adrenaline production as a result of activation of the sympatho-adrenal-medullary system activation.[24] However, our volunteers had increased norepinephrine, which may have been a result of the predominance of physical stress. Seals[25] showed that isometric exercise of different intensities increased sympathetic activity, BP, and systemic vascular resistance in healthy individuals. He also observed an increase in heart rate during isometric exercise. In our study, there was no increase in heart rate. However, hemodynamic variables were recorded after physical and mental stimulation, not during. But, our idea was to assess whether the two types of stimuli could mobilize fatty acids by activating the sympathetic nervous system and whether the increase in fatty acids would result in hemodynamic changes, especially the increase in BP. Previous research has shown that fatty acids augment vascular alpha-adrenergic vasoconstriction.[26,27] In our study, we also observed a slight increase in the distensibility of large and small arteries. We do not have a clear answer to this finding. However, it is possible to speculate the possibility of beta-adrenergic effect in these vessels. In an experimental study, Brawley et al[28] described the relaxation of the rat aorta mediated by β adrenoreceptors.

This study shows, for the first time, the association of physical and mental stress with increased fatty acids, norepinephrine, and increased systemic vascular resistance associated with increased diastolic BP. Although physical and mental stress increased fatty acids, norepinephrine, and slightly altered BP, basal metabolic rate was unchanged. In a recent study of elderly individuals, handgrip exercise was associated with an increase in basal metabolic rate assessed from the Singapore equation.[29] In our study, basal metabolic rate was estimated from a bioimpedance device. The Singapore equation has been tested and valid against other regularly used equations and has shown good accuracy to measure basal metabolic rate in Orientals with different BMI values.[30] In our study, the stimuli applied were sufficient to slightly increase sympathetic activity and may not have been sufficient to increase the basal metabolic rate. In conclusion, physical and mental stress resulted in increased free fatty acids, noradrenaline, diastolic BP, and systemic vascular resistance.

TABLE 2 Hemodynamics data at baseline and after physical and mental stress (mean ± SD)

Variables (n = 15)	Before	After	p value
Systolic BP (mm Hg)	110 ± 11	112 ± 9	.479
Diastolic BP (mm Hg)	64 ± 7	68 ± 6	.021
Pulse pressure (mm Hg)	47 ± 9	44 ± 8	.146
Heart rate (bpm)	71 ± 11	68 ± 13	.308
Cardiac debit (L/min)	5.4 ± 0.6	5.5 ± 0.6	.677
Large arteries distensibility (ml/mm Hg × 10)	14 ± 5	16 ± 4	.049
Small arteries distensibility (ml/mm Hg × 100)	8 ± 3	10 ± 2	.044
Systemic vascular resistance (dyne.sec.cm^{-5})	1209 ± 139	1264 ± 142	.023

TABLE 3 Bioimpedance data before and after physical and mental stress (mean ± SD)

Variable (n = 15)	Before	After	p value
Phase angle (degrees)	6.76 ± 1.14	6.70 ± 1.16	.887
Capacitance (pF)	626 ± 156	625 ± 149	.972
Resistance (ohms)	605.0 ± 95.8	604.7 ± 93.1	.991
Reactance (ohms)	69.9 ± 12.7	70.4 ± 13.7	.905
Lean mass (kg)	51.3 ± 10.0	50.9 ± 9.8	.891
Fat mass (kg)	21.3 ± 5.8	20.8 ± 4.8	.919
Body water (%)	36.7 ± 6.8	36.5 ± 6.7	.953
Basal metabolic rate (cal)	1600 ± 314	1588 ± 309	.919

4.1 | Study limitations

1. Hemodynamic data were recorded immediately after stimuli, not during stimuli. This was probably the reason why we registered small hemodynamic changes.
2. The bioimpedance data were also recorded immediately after the stimuli, and this can perhaps explain no change in the metabolic rate.
3. The study did not include a sham control group.

Considering that low intensity physical and mental stress occur frequently in life, it would be interesting to determine if participants with altered cardiovascular and metabolic regulation, such as metabolic syndrome patients, would exhibit a greater response of neurogenic, metabolic, and hemodynamic response to combined physical and mental stress than healthy volunteers.

AUTHOR CONTRIBUTIONS

JM: Contributed to conception and design of the study, acquisition of data, analysis and interpretation of data, statistical analysis, and draft the manuscript; LS: Contributed to acquisition of data, analysis and interpretation of data; BV: Contributed to acquisition of data, analysis and interpretation of data; HD: Contributed to acquisition of data, analysis, interpretation of data, and draft the manuscript; FC: Contributed to conception and design of the study and draft the manuscript; BE: Contributed to the revision of the manuscript (style) and with different suggestions in the analysis and interpretation of the data. HL: Contributed to conception and design of the study, interpretation of data, and draft the manuscript.

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How to cite this article: Motta e Motta J, Souza LN, Vieira BB, et al. Acute physical and mental stress resulted in an increase in fatty acids, norepinephrine, and hemodynamic changes in normal individuals: A possible pathophysiological mechanism for hypertension—Pilot study. *J Clin Hypertens*. 2021;23:888–894. https://doi.org/10.1111/jch.14190