Effect of psychological stress on blood pressure increase: a meta-analysis of cohort studies

Efeito do estresse psicológico no aumento da pressão arterial: uma metanálise de estudos de coorte

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Abstract

Studies have suggested that chronic exposure to stress may have an influence on increased blood pressure. A systematic review followed by a meta-analysis was conducted aiming to assess the effect of psychological stress on blood pressure increase. Research was mainly conducted in Ingenta, Psycinfo, PubMed, Scopus and Web of Science. Inclusion criteria were: published in any language; from January 1970 to December 2006; prospective cohort design; adults; main exposure psychological/emotional stress; outcome arterial hypertension or blood pressure increase ≥ 3.5mmHg. A total of 2,043 studies were found, of which 110 were cohort studies. Of these, six were eligible and yielded 23 comparison groups and 34,556 subjects. Median follow-up time and loss to follow-up were 11.5 years and 21%. Results showed individuals who had stronger responses to stressor tasks were 21% more likely to develop blood pressure increase when compared to those with less strong responses (OR: 1.21; 95%CI: 1.14-1.28; p < 0.001). Although the magnitude of effect was relatively small, results suggest the relevance of the control of psychological stress to the non-therapeutic management of high blood pressure.

Blood Pressure; Hypertension; Psychological Stress

Introduction

According to the World Health Organization [1], non-transmissible diseases will be the leading cause of functional disability in the next two decades and, among chronic degenerative conditions, arterial hypertension will be the most important cause. Hypertension is a public health concern due to its magnitude, risks, difficulty in management, high medical and social costs and severe cardiovascular and renal complications [2]. The number of deaths due to hypertension as primary cause was estimated to be over 7 million in 2002, approximately 13% of all reported deaths [1]. Hypertensive adults will reach 1.5 billion by 2025, around 30% of the world population [3].

Hypertension management comprises drug and/or non-drug therapeutic approaches. Although there is clear evidence that antihypertensive medications are useful in controlling hypertension and reducing the incidence of stroke and infarction [2], long-term drug treatment can be expensive and side-effects can threaten patients' adherence to drug prescriptions [4]. The identification of non-pharmacological methods to prevent, or significantly delay the onset of hypertension would represent an important advance in the prevention of cardiovascular disease [2]. Among non-drug approaches, lifestyle changes recommended include: weight reduction, a diet rich in fruits, vegetables, and low fat dairy products with a reduced content of satu-

rated and total fat, dietary sodium reduction, engagement in regular aerobic physical activity and limited alcohol consumption [2]. Extensive trials of over 100 randomized trials indicates the efficacy of behavioral treatments for hypertension [5]. Behavioral changes should also include anti-stress activities [6].

The Medical Subject Headings (MeSH) defines stress as a pathological process resulting from body response to external forces and abnormal states that tend to affect its homeostasis. It comprises daily events that increase physiological activities and consequently cause psychological wear and tear to some extent [7]. When emotional stressors are prevailing, this condition is known as psychological stress. Modern life events such as work-related and family problems, social withdrawal, financial worries and violence are some factors that can predispose or potentate stress [8].

It has been suggested that chronic exposure to psychological stress can cause increased blood pressure and lead to hypertension development [5]. A cohort study of over 3,000 young adults [9] showed that urgency/impatience behavior, and hostility assessed during young adulthood were strongly associated with a higher risk of developing hypertension 15 years later. Other exposures such as depression and anxiety were also reported. Chronic stress due to financial strain has been reported to predict high blood pressure during three to seven years of follow-up [10]. A study with 11,119 cases and 13,648 controls from 52 countries [11] reported strong associations of myocardial infarction (cases) and more frequent periods of stress at home, more severe financial stress and more stressful life events compared with controls. In terms of myocardial infarction risk, the effect of psychosocial stress was as important in magnitude as traditional cardiovascular disease risk factors such as smoking, obesity, diabetes and hypertension. In addition, a systematic review of 23 treatment comparisons from 17 randomized trials conducted in patients with elevated blood pressure, demonstrated strong effects of transcendental meditations on reductions in blood pressure. Despite non-significant results, other anti-stress interventions such as biofeedback, progressive muscle relaxation and stress management training also reported clinically important reductions in blood pressure [12]. Therapies such as these may help patients to reduce the effects of stress by reducing physiologic arousal and restoring autonomic balance, thereby reducing blood pressure [5].

The purpose of the present meta-analysis was to assess the effect of psychological stress on blood pressure increase.

Methods

A systematic review followed by meta-analysis of prospective cohort studies was conducted.

Search strategy

The systematic search of articles was carried out based on *Undertaking Systematic Reviews of Research on Effectiveness* guidelines [13] and Cochrane Reviewers' Handbook [14]. The following databases were searched: Biological Abstracts; CAB Abstracts; Ingenta; Psycinfo; PubMed; Scopus; Web of Science; SIGLE; NTIS; NDLTD and reference lists of the selected articles. Table 1 shows searches in the different databases.

Inclusion and exclusion criteria

Inclusion criteria were: published between January 1970 and December 2006, with this starting date chosen because studies investigating the effect of psychological stress on the development of morbid conditions were first published in that decade [15,16]; prospective cohort design, this study design being one of the most appropriate for assessing causality [17] while taking into consideration the major issue of temporality, i.e., exposure prior to disease; 18 to 64 year-old normotensive adults; main exposure measured through reactivity or recovery, reactivity is the difference between blood pressure during the stressor task and baseline [18] and recovery is blood pressure measured after a stressful task [19]; dichotomous outcome as arterial hypertension or increase in systolic and/or diastolic blood pressure ≥ 3.5mmHg; and reporting relative risks, hazard ratios or odds ratios (OR).

Articles were excluded if they were based on hypertensive men and/or women at enrollment; reported other types of stress or if outcome was measured on a continuous scale.

Study quality

The quality of studies selected for inclusion in the meta-analysis was assessed. Assessments were based on the National Institute for Health and Clinical Excellence criteria [20] including subject selection, refusals, losses to follow-up, exposure and outcome measurement, level of exposure and adjustments for confounders. Two independent evaluators conducted quality assessments.

Data extraction

Data were independently extracted by two researchers. The principal information obtained

Table 1

Searches, keywords and boolean operators, number of retrieved and selected articles according to the databases.

Date of search	Database	Keywords	Retrieved articles	Selected articles
03/Jan/2007	Biological Abstracts, CAB Abstracts and Psycinfo	(stress OR psychological stress OR emotional stress OR life stress) AND (blood pressure OR hypertension) AND (cohort studies OR prospective studies OR follow-up studies)	34	0
04/Jan/2007	Ingenta	(stress OR psychological stress OR emotional stress OR life stress) AND (blood pressure OR hypertension) AND (cohort studies OR prospective studies OR follow-up studies)	62	0
08/Jan/2007	PubMed	(stress [MeSH] OR psychological stress [mh] OR emotional stress [mh] OR life stress [mh]) AND (hypertension [MeSH] OR blood pressure [MeSH]) AND ((cohort studies [MeSH] OR risk [MeSH] OR (odds [WORD] AND ratio* [WORD]) OR (relative [WORD] AND risk [WORD])). Limits: Adolescent: 13-18 years, Adult: 19-44 years, Middle Aged: 45-64 years, Publication Date from 1970/01/01 to 2006/12/31, Journal Article, Humans.years, Middle Aged: 45-64 years, Publication Date from 1970/01/01 to 2006/12/31, Journal Article, Humans	617	5
09/Jan/2007	Scopus	(stress OR psychological stress OR emotional stress OR life stress) AND (hypertension OR blood pressure) AND (cohort studies OR prospective studies OR follow-up studies)	13	0
09/Jan/2007	Web of Science	(stress OR psychological stress OR emotional stress OR life stress) AND (hypertension OR blood pressure) AND (cohort studies OR prospective studies OR follow-up studies)	1,158	5
10/Jan/2007	NITS	(stress OR psychological stress OR emotional stress OR life stress) AND (hypertension OR blood pressure) AND (cohort studies OR prospective studies OR follow-up studies)	0	0
10/Jan/2007	SIGLE	(stress OR psychological stress OR emotional stress l OR life stress) AND (hypertension OR blood pressure) AND (cohort studies OR prospective studies OR follow-up studies)	0	0
10/Jan/2007	NDLTD	(stress OR psychological stress OR emotional stress OR life stress) AND (hypertension OR blood pressure) AND (cohort studies OR prospective studies OR follow-up studies)	160	0

included: title, authors, journal, year of publication, country, number of subjects, follow-up time, losses to follow-up, age, study population, measurement of exposure, outcome and confounders in the multivariable model.

Most studies reported OR as the measure of effect. Data extracted from each article were neperian logarithm of OR and standard error (SE). One study reported the hazard ratio [21] and another reported the relative risk [22] and these measures were converted into OR [23]. When only confidence intervals were available, they were converted into SE [14].

Data analysis

Data analyses were performed using Stata software 9.2 (Stata Corp., College Station, USA). The variability of the selected studies was evaluated through a heterogeneity test using models with

fixed effects when the test was statistically non-significant ($p \geq 0.05$) and random effects when the test was statistically significant ($p < 0.05$) [14]. Begg's and Egger's tests were used to investigate the existence of publication bias [24].

To avoid potential effects of heterogeneity and assess the individual impact of each variable studied, analyses were conducted by the following subgroups: age, gender, study losses, years of follow-up, test applied, exposure measurement and multivariable analysis. Lastly, a combination of measures of effect by study was assessed as well as the impact of exclusion of each study on the combined effect.

Results

Figure 1 shows the number of studies found and reasons for exclusion at each step of the systematic search. Of 110 cohort studies, six were selected yielding 23 comparison groups. For example, Steptoe et al. [25] assessed the effect of middle and highest tercile systolic and diastolic blood pressure recovery on two outcomes: increased blood pressure ≥ 3.5mmHg and ≥ 5mmHg, thus providing 8 comparisons (2x2x2). Table 2 illustrates the study characteristics and comparisons of the meta-analysis.

Of the cohort studies included in the meta-analysis, three were from North America and three from Europe. The sum of subjects in the comparisons yielded a total of 34,556 subjects. Mean follow-up was 11.8 years ranging between 3 to 25 years. Loss to follow-up ranged from between 8.3% and 34.2%. In most studies, exposure was measured through reactivity to mental tasks. The main method of analysis was logistic regression and the main measure of effect was OR (Table 2).

The Q test showed the existence of heterogeneity among studies (Figures 2 and 3). Therefore results from random effect models showed that those subjects with higher reactivity/recovery were 21% more likely to have blood pressure increase when compared to those with lower reactivity/recovery (OR = 1.21; 95%CI: 1.14-1.28) (Figure 2). Similar results were found when only one measure of effect by study was considered (OR = 1.28; 95%CI: 1.13-1.43) (Figure 3). The exclusion of the Matthews et al. [21] or Markovitz's et al. [26] studies increased the effect of psychological stress on blood pressure by about 20% (OR = 1.51; 95%CI: 1.17-1.94).

The subgroup analysis revealed significant effects (OR > 2) in studies including subjects over 46 years of age, small losses to follow-up, long-term follow-up, those with a combination of stressful tasks and those where exposure was measured through recovery (Table 3). In addition, heterogeneity between studies was non-significant when the outcome was hypertension, exposure was measured by recovery, combinations of tests, females and studies with longer years of follow-up and lower losses.

Publication bias

Both Begg's and Egger's tests showed statistically significant results that were confirmed by the funnel plot asymmetry.

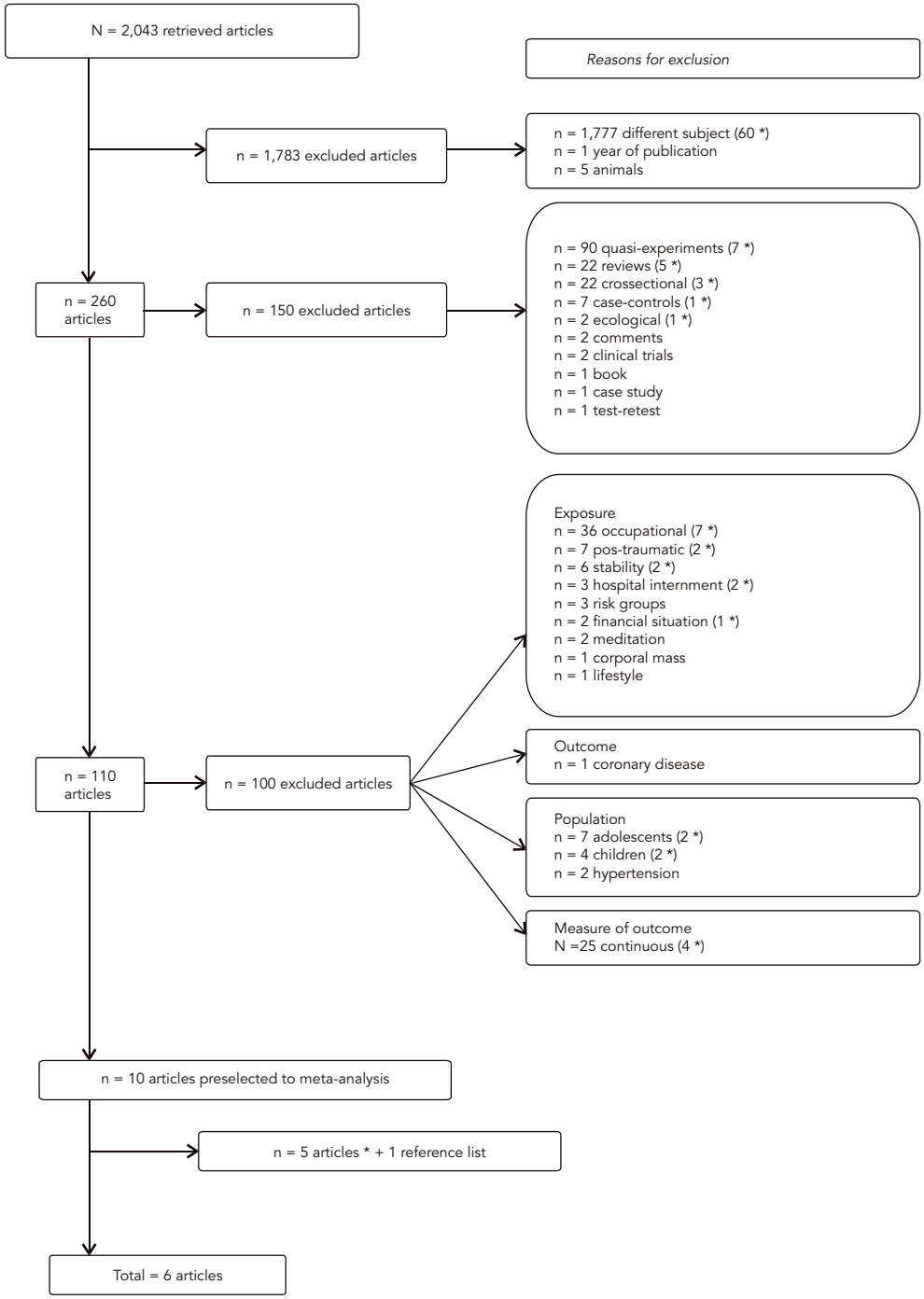
Discussion

The present meta-analysis assessed the effect of psychological stressful tasks on blood pressure increase in adults aged between 18 and 64 years. Individuals with high increases of blood pressure during stressful tasks (reactivity) and those with high blood pressure in the recovery period after the tasks (recovery) showed greater odds of developing hypertension or increased blood pressure. This finding corroborates other findings of studies on the association between psychological stress and blood pressure increase [27,28,29].

It has been suggested that repeated episodes of heightened cardiovascular reactivity could contribute to hypertension development by promoting vascular remodeling [30]. These pathophysiological changes, in turn, could alter the long-term regulation of blood pressure by the kidneys, resulting in a shift in the blood pressure set point to higher levels. Poor cardiovascular recovery could contribute to hypertension development through the same mechanisms that have been proposed for heightened cardiovascular reactivity [31]. Alternatively, it has been hypothesized that both heightened cardiovascular reactivity and poor cardiovascular recovery could be markers for other pathophysiological processes involved in the etiology of hypertension, such as dysfunction in the regulation of the heart and vasculature by the autonomic nervous system [32]. More specifically, heightened cardiovascular reactivity could reflect sympathetic hyperresponsivity or enhanced vagal withdrawal during stress, whereas poorer cardiovascular recovery could be due to prolonged sympathetic activation, diminished vagal tone, or attenuated or delayed vagal rebound following the termination of stress [33]. In addition, several studies have reported associations between psychosocial variables and vascular function [34,35], inflammation [36], increased blood clotting and decreased fibrinolysis [37,38].

Figure 1

Number of selected studies and reasons for exclusion at each step of the systematic search.



* Duplicates in different databases.

Table 2

Descriptive characteristics of studies and comparisons of the meta-analysis.

Author	Country	Baseline (n)	End (n)	Loss (%)	Years of follow-up	Age	Participants
Borghi et al. [43]	Italy	89	70	21.3	15	< 45	Men/Women
Carroll et al. a [44]	England	1,003	796	20.6	10	35-55	Men
Carroll et al. b [44]	England	1,003	796	20.6	10	35-55	Men
Markovitz et al. a [26]	USA	5,115	1,557	34.2	5	45-59	Men *
Markovitz et al. b [26]	USA	5,115	1,557	34.2	5	45-59	Men *
Markovitz et al. c [26]	USA	5,115	1,763	34.2	5	45-59	Women *
Markovitz et al. d [26]	USA	5,115	1,763	34.2	5	45-59	Women *
Matthews et al. a [21]	USA	5,115	3,553	30.5	13	18-30	Men/Women
Matthews et al. b [21]	USA	5,115	4,075	20.3	13	18-30	Men/Women
Matthews et al. c [21]	USA	5,115	4,100	19.8	13	18-30	Men/Women
Matthews et al. d [21]	USA	5,115	3,463	32.3	13	18-30	Men/Women
Matthews et al. e [21]	USA	5,115	4,108	19.7	13	18-30	Men/ women
Matthews et al. f [21]	USA	5,115	4,122	19.4	13	18-30	Men/Women
Menkes et al. a [22]	USA	1,130	815	19.3	25	< 45 *	Men
Menkes et al. b [22]	USA	1,130	346	19.3	25	≥ 45 *	Men
Steptoe et al. a [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe et al. b [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe et al. c [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe et al. d [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe_et al e [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe et al. f [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe et al. g [25]	England	228	209	8.3	3	45-59	Men/Women
Steptoe et al. h [25]	England	228	209	8.3	3	45-59	Men/Women

(continues)

Table 2 (continued)

Descriptive characteristics of studies and comparisons of the meta-analysis.

Exposure	Severity of exposure	Stress task	Outcome (mm/Hg)	Analysis	Confounders
Reactivity DBP	High	Mental arithmetic	HTN (DBP > 95)	Logistic regression	Age, BMI, sex, cholesterol, family history of HTN, baseline SBP/DBP
Reactivity SBP *	High	Raven's matrices	HTN (SBP ≥ 16 DBP ≥ 90)	Logistic regression	Age, baseline SBP
Reactivity DBP *	High	Raven's matrices	HTN (SBP ≥ 160 DBP ≥ 90)	Logistic regression	Age, baseline SBP
Reactivity SBP	Moderate *	Video game	Increase ≥ 8	Logistic regression	-
Reactivity SBP	High *	Video game	Increase ≥ 8	Logistic regression	-
Reactivity SBP	Moderate *	Video game	Increase ≥ 8	Logistic regression	-
Reactivity SBP	High *	Video game	Increase ≥ 8	Logistic regression	-
Reactivity SBP *	High	Cold pressor *	HTN (SBP ≥ 140 DBP ≥ 90)	Cox Regression	Age, BMI, education, SBP/DBP
Reactivity SBP *	High	Star tracing *	HTN (SBP ≥ 140 DBP ≥ 90)	Cox regression	Age, BMI, education, SBP/DBP
Reactivity SBP *	High	Vídeo game *	HTN (SBP ≥1 40 DBP ≥ 90)	Cox regression	Age, BMI, education, SBP/DBP
Reactivity DBP *	High	Cold pressor *	HTN (SBP ≥ 140 DBP ≥ 90)	Cox regression	Age, BMI, education, SBP/DBP
Reactivity DBP *	High	Star tracing *	HTN (SBP ≥ 140 DBP ≥ 90)	Cox regression	Age, BMI, education, SBP/DBP
Reactivity DBP *	High	Vídeo game *	HTN (SBP ≥ 140 DBP ≥ 90)	Cox regression	Age, BMI, education, SBP/DBP
Reactivity SBP	High	Cold pressor	Increase ≥ 20 SBP/DBP	Cox regression	Age, BMI, smoking, HTN familial history, SBP
Reactivity SBP	High	Cold pressor	Increase ≥ 20 SBP/DBP	Cox regression	Age, BMI, smoking, HTN familial history, SBP
Recovery SBP *	Moderate *	Colour-word/ Mirror tracing	Increase ≥ 5 SBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline SBP
Recovery SBP *	High *	Colour-word/ Mirror tracing	Increase ≥ 5 SBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline SBP
Recovery DBP *	Moderate *	Colour-word/ Mirror tracing	Increase ≥ 5 SBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline SBP
Recovery DBP *	High *	Colour-word/ Mirror tracing	Increase ≥ 5 SBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline SBP
Recovery SBP *	Moderate *	Colour-word/ Mirror tracing	Increase ≥ 3.5 DBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline DBP
Recovery SBP *	High *	Colour-word/ Mirror tracing	Increase ≥ 3.5 DBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline DBP
Recovery DBP *	Moderate *	Colour-word/ Mirror tracing	Increase ≥ 3.5 DBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline DBP
Recovery DBP *	High *	Colour-word/ Mirror tracing	Increase ≥ 3.5 DBP *	Logistic regression	Age, sex, job, antihypertensive medications, BMI, smoking, baseline DBP

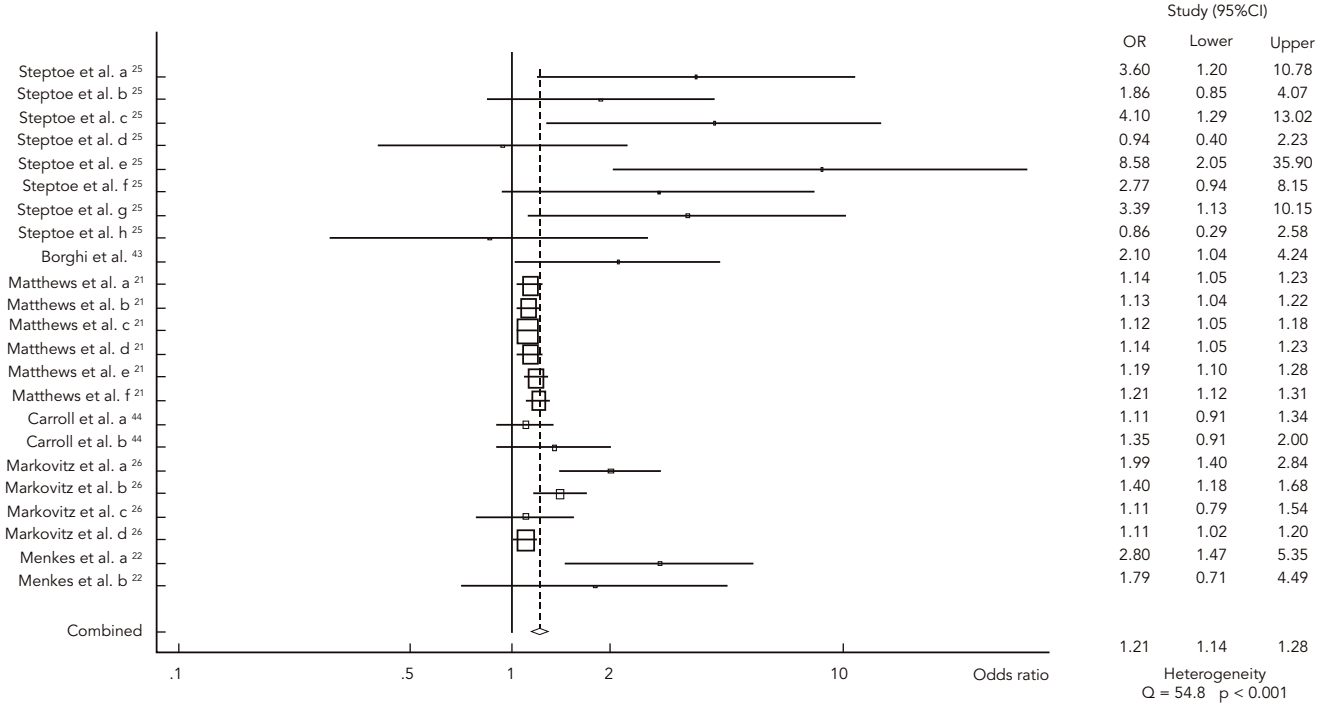
HTN: hypertension; SBP: systolic blood pressure; DBP: dyastolic blood pressure; BMI: body mass index.

Note: letters at end of authors' name shows different comparison groups within a single study.

* Indicates the location of differences between comparison groups.

Figure 2

Random effect model, odds ratio for increased blood pressure ≥ 3.5mmHg/hipertension of the effect of responses to stressor tasks in 23 comparison groups from 6 prospective cohorts.



Note: letters at end of authors' name shows different comparison groups within a single study.

Another action mechanism of stress involved in blood pressure increase could be indirect. Stress would be associated to risk factors such as obesity, smoking, alcohol abuse and physical inactivity and they would cause blood pressure increase. For example, a meta-analysis including 69 studies demonstrated that, despite the relatively small effects, physically active subjects had better cardiovascular recovery than inactive ones [39].

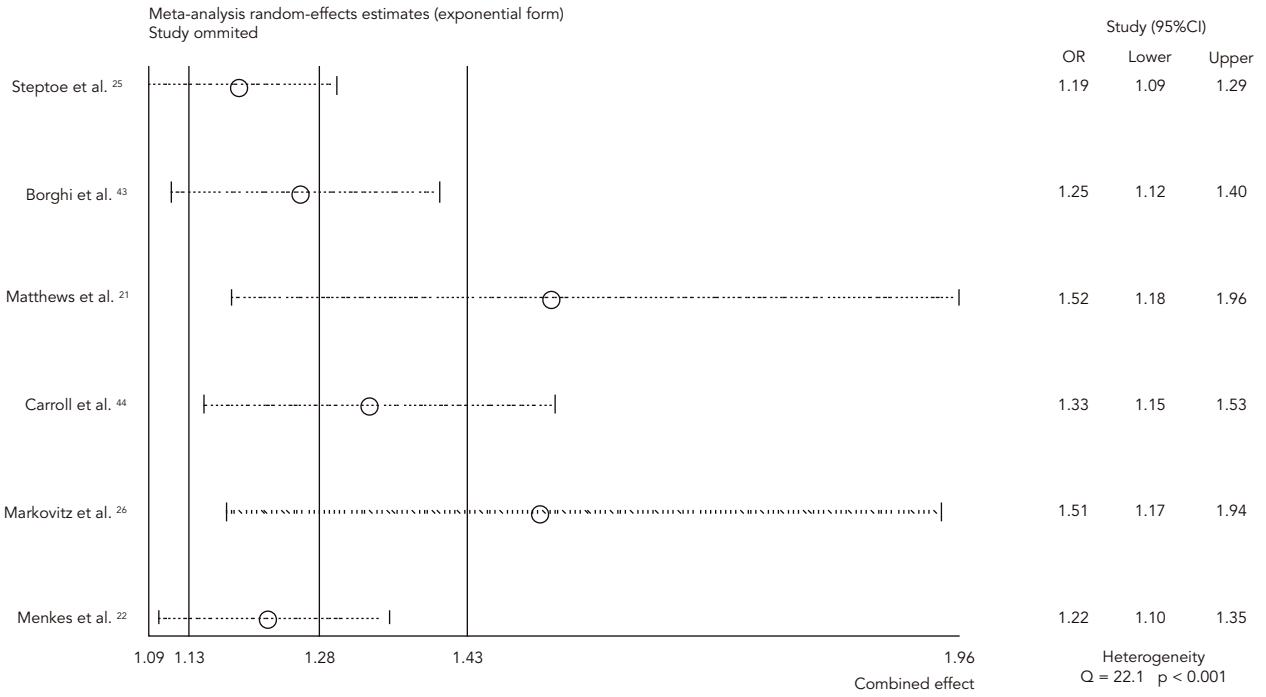
In addition, the results of subgroup analysis show that individuals with higher blood pressure in the recovery period were twice as likely to have blood pressure increase when compared to those whose exposure was measured through reactivity. This finding suggests that cardiovascular measures of recovery can provide valuable information not captured in measures of reactivity and thus help predicting longitudinal changes in blood pressure [28]. A study found that individuals undergoing a stressful task had late recovery of blood pressure, which suggests recovery might be a helpful predictor of blood pressure increase [39].

More pronounced effects were seen in studies with small losses and longer follow-up. In prospective studies there is greater concern with subject losses when they are associated to the study outcome or risk categories. The greater the loss, the greater the likelihood of bias [40]. Besides, in chronic exposures, such as stress, individuals have to be exposed for a time period long enough to set off the causal process [17]. Individuals aged between 46 and 64 years were about twice more likely to develop hypertension or blood pressure increase than young adults (Table 3).

This study has several limitations. The first is due to the heterogeneity of the studies selected. Measures of effect of highly heterogeneous studies have low validity [41]. In this study, heterogeneity was mostly due to differences in study populations, measures of exposure and outcome, losses and follow-up time. The effect of heterogeneity was partially overcome by the use of random effects models, subgroup analysis, a combination of effect by study and analysis of the impact of

Figure 3

Random effects model, odds ratio and 95% confidence interval for the combined effect by study and the impact of the exclusion of each study.



exclusion of each study on the final combined effect [41]. The second limitation is the detection of publication bias, which calls for a careful interpretation of findings. However, an evaluation comprising 48 systematic reviews of the Cochrane database demonstrated that, despite the fact that biases were seen in 50% of the studies, they significantly affected results in less than 10% of meta-analyses [42]. Third, although laboratory stress measurements potentially allow for greater control on the part of the investigator, stress tasks were applied on an acute basis and stress is assumed to occur chronically thus limiting test conclusions. The fourth limitation is related to the fact that the majority of studies included in the meta-analysis reported OR as measure of effect. When an outcome is commonly seen in a study population (as is the case of blood pressure increase), the OR might overestimate the effect of association [23]. However, further analyses showed that when OR were converted into relative risks, a relative risk of 1.17 (95%CI: 1.10-1.25) was found for the combined effect. There seems to remain an effect of stress on blood pressure increase.

In conclusion, although the magnitude of effect was relatively small, results point to the relevance of control of psychological stress for the non-therapeutic management of high blood pressure. Further research investigating the role of stress in hypertension pathogenesis should be conducted.

Table 3

Combined effect of psychological stress on the increase of the blood pressure in sub-groups of cohorts according to participants and study design.

Variables	Number of combined	Size of the sample	OR (95%CI) *	p-value	Heterogeneity
Age (years)					
18-45	12	30,946	1.18 (1.12-1.24)	< 0.001	p = 0.002
46-64	9	2,018	2.12 (1.51-2.97)	< 0.001	p = 0.113
18-64	2	1,592	1.15 (0.97-1.37)	0.118	p = 0.371
Sex					
Men	6	5,867	1.51 (1.20-1.90)	< 0.001	p = 0.015
Women	2	3,526	1.11 (1.02-1.19)	0.01	p = 1.0
Men/Women	15	25,163	1.18 (1.11-1.25)	< 0.001	p = 0.006
Loss (%)					
0-10	9	1,742	2.16 (1.56-4.66)	< 0.001	p = 0.118
11-20	7	13,891	1.17 (1.12-1.22)	< 0.001	p = 0.116
21 or more	7	18,923	1.18 (1.10-1.26)	< 0.001	p = 0.009
Years of follow-up					
0-10	14	9,904	1.44 (1.20-1.73)	< 0.001	p < 0.001
11-20	7	23,491	1.15 (1.12-1.18)	< 0.001	p = 0.393
21 or more	2	1,161	2.41 (1.42-4.10)	< 0.001	p = 0.433
Test					
Combined	8	1,672	2.17 (1.51-3.12)	< 0.001	p = 0.076
Arithmetic	3	1,662	1.19 (1.01-1.41)	0.044	p = 0.181
Videogame	6	14,862	1.21 (1.10-1.32)	< 0.001	p = 0.003
Cold pressor	4	8,177	1.19 (1.04-1.36)	0.01	p = 0.041
Star tracing	2	8,183	1.16 (1.09-1.22)	< 0.001	p = 0.377
Exposure					
Reactivity	15	32,884	1.18 (1.12-1.24)	< 0.001	p = 0.007
Recovery	8	1,672	2.17 (1.51-3.12)	< 0.001	p = 0.076
Outcome					
HTN	11	26,244	1.15 (1.12-1.19)	< 0.001	p = 0.124
SBP/DBP increase ≥ 3.5mmHg	12	8,312	1.59 (1.25-2.03)	< 0.001	p < 0.001
Multivariable analysis					
Yes	19	27,916	1.19 (1.12-1.27)	< 0.001	p = 0.002
No	4	6,640	1.32 (1.05-1.66)	< 0.001	p = 0.002

HTN: hypertension; SBP: systolic blood pressure; DBP: dyastolic blood pressure.

* Fixed effects models are used when the heterogeneity test was statistically non-significant (p ≥ 0.05) and random effects models when the test was statistically significant.

Resumo

Estudos sugerem que a exposição crônica ao estresse tenha influência no aumento dos níveis pressóricos. Foi realizada uma revisão sistemática seguida de metanálise com o objetivo de avaliar o efeito do estresse psicológico no aumento da pressão arterial. As principais bases de dados utilizadas foram Ingenta, Psycinfo, PubMed, Scopus e Web of Science. Os critérios de inclusão foram: publicado entre janeiro de 1970 e dezembro de 2006, delineamento de coorte prospectiva, adultos, estresse psicológico/emocional como exposição principal, hipertensão arterial ou aumento na pressão arterial ≥ 3,5mmHg como desfecho. A busca resultou em 2.043 artigos, sendo 110 coortes. Desses, seis eram elegíveis, os quais geraram 23 grupos de comparação e 34.556 sujeitos. A mediana do tempo de seguimento e do percentual de perdas foi 11,5 anos e 21%. Indivíduos com maior reação a tarefas estressoras possuíam 21% mais chances de apresentar aumento na pressão arterial quando comparados com aqueles com menor reação (OR = 1,21; IC95%: 1,14-1,28; p < 0,001). Embora com magnitude de efeito relativamente modesta, os resultados sugerem a importância do controle do estresse psicológico no tratamento não medicamentoso da hipertensão arterial sistêmica.

Pressão Arterial; Hipertensão; Estresse Psicológico

Contributors

D. Gasperin initiated the study, conducted the systematic review and wrote the manuscript. G. Netuveli assisted in the study design. J. S. Dias-da-Costa helped in theoretical aspects. M. P. Pattussi supervised the study and data analysis. All authors reviewed the manuscript and interpreted results.

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