Journal of Psychosomatic Research xxx (2014) xxx–xxx

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

Attributional styles and stress-related atherogenic plasma lipid reactivity in essential hypertension

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ARTICLE INFO

Article history:
Received 9 October 2013
Received in revised form 10 April 2014
Accepted 11 April 2014
Available online xxxx

Keywords:
Attributional styles
Cholesterol
Low-density-lipoprotein cholesterol
Hypertension
Plasma lipids
Psychosocial stress
Trier Social Stress Test

ABSTRACT

Objective: Hypertension and an atherogenic lipid profile are known risk factors for coronary heart disease (CHD). Hypertensives show greater changes in atherogenic plasma lipids to acute stress than normotensives. In this study, we investigated whether attribution of failure is associated with lipid stress reactivity in hypertensive compared with normotensive men.
Methods: 18 normotensive and 17 hypertensive men (mean ± SEM; 45 ± 2.2 years) underwent an acute standardized psychosocial stress task that can be viewed as a situation of experimentally induced failure. We assessed external-stable (ES), external-variable (EV), internal-stable (IS), and internal-variable (IV) attribution of failure and psychological control variables (i.e. extent of depression and neuroticism). Moreover, total cholesterol (TC), low-density-lipoprotein cholesterol (LDL-C), and norepinephrine were measured immediately before and several times after stress.
Results: ES moderated TC- and LDL-C-stress reactivity in hypertensives as compared to normotensives (interaction mean arterial pressure [MAP]-by-ES for TC: $F = 3.71$, $p = .015$; for LDL-C: $F = 3.61$, $p = .016$). TC and LDL-C levels were highest in hypertensives with low ES immediately after stress ($p \leq .039$). In contrast, hypertensives with high ES did not differ from normotensives in TC and LDL-C immediately after stress (p's $> .28$). Controlling for norepinephrine, depression, and neuroticism in addition to age and BMI did not significantly change results. There were no significant associations between lipid baseline levels or aggregated lipid secretion and IS, IV, or EV (p's $> .23$).
Conclusion: Our data suggest that ES may independently protect from elevated lipid stress reactivity in hypertensive individuals. ES thus might be a protective factor against CHD in hypertension.

Introduction

Essential hypertension ranks among the main risk factors for coronary heart disease (CHD) [1] but the underlying mechanisms are not fully understood. A well-known classic risk factor for CHD is an atherogenic lipid profile with increased total cholesterol (TC), low-density lipoproteine cholesterol (LDL-C), and triglycerides (TG) on the one hand, and decreased high-density lipoprotein cholesterol (HDL-C) on the other [2,3]. Hypertensives tend to have greater TC, LDL-C, and TG resting levels, and lower resting levels of HDL-C; moreover the prevalence of hyperlipidemia in hypertension is as high as 40% [4]. Psychological stress has increasingly been implied in CHD development [5,6]. Specifically, studies on short-term physiological responses to controlled stress induction serve as a window into complex psychological and physiological processes involved in the development of CHD [7]. Recent studies also suggest that elevated physiological stress reactivity or hyper-reactivity is an independent CHD risk indicator per se [8–11]. In particular, stress-induced hyper-activation of the sympathetic nervous system (SNS) including the cardiovascular system [8], and the hypothalamus–pituitary–adrenal (HPA) axis [9] have been implicated to increase CHD risk, either by direct effects and/or by inducing adverse changes in intermediate biological risk factors including blood lipids [10,11]. With respect to hypertension, we recently found greater TC and LDL-C changes to acute psychosocial stress in hypertensives compared to normotensives [12]. This suggests that hypertensives are particularly susceptible to stress-induced elevations in atherogenic lipids. Given the importance of elevated physiological stress reactivity for CHD, the observed heightened TC/LDL-C stress reactivity in essential hypertension may provide one mechanism by which stress might increase CHD risk in hypertension.

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http://dx.doi.org/10.1016/j.jpsychores.2014.04.004
0022-3999/

In line with such reasoning, it might be of clinical relevance to identify conditions that relate to lower TC/LDL-C stress reactivity in essential hypertension. We recently found higher social support and beneficial emotional regulation to be related to a reduced stress reactivity of stress hormones in hypertension [13], but studies on this issue are sparse. Previous research has suggested links between attributional styles and health outcomes [14–18]. Attributional styles are the habitual manner in which individuals explain positive or negative events in their lives. Seligman's theory of causal attribution distinguishes between an optimistic and a pessimistic attributional style [19]. The optimistic attributional style is characterized by the tendency to explain negative events in terms of external, unstable, and situation-specific causes and positive events in terms of internal, stable, and global causes [14,19]. The pessimistic attributional style is defined as the opposite of the optimistic one. Individuals with an optimistic attributional style are regarded as more optimistic with more favorable expectancies of the future than individuals with a pessimistic attributional style [14]. A variety of studies suggests that optimistic attributional styles or optimism are associated with clinically important benefits in cardiovascular outcomes [18,20–22]. For example, a 10-year large-scale longitudinal study demonstrated a strong association between an optimistic attributional style and lower incidence of CHD [18]. Although the underlying pathways by which an optimistic attributional style or optimism affects cardiovascular outcomes are poorly understood, lower physiological stress responses are presumed to be involved [23]. The role of attributional styles in the interface between TC/LDL-C stress reactivity and hypertension has not yet been studied.

The aim of this study was to investigate whether attributional styles might be associated with TC/LDL-C stress reactivity in otherwise healthy, middle-aged hypertensive men, compared with age-matched normotensives. To induce psychosocial stress, we used the Trier Social Stress Test (TSST), a motivated performance task disguised as a job interview [24]. This standardized stress test combines elements of uncontrollability and social-evaluative threat, and creates a context in which participants are unable to get positive feedback despite best effort. Thus, the TSST can be viewed as a situation of experimentally induced failure. We assumed that the habitual manner to attribute failure to external (e.g. misfortune or task difficulty) but not to internal (e.g. effort or ability) factors might especially be associated with stress preventive effects because subjects would not attribute failure as self-inflicted. We hypothesized that greater external-stable (ES) or external-variable (EV) attribution of failure and lower internal-stable (IS) or internal-variable (IV) attribution of failure would be associated with attenuated increases in atherogenic blood lipids (i.e. TC and LDL-C) to acute stress in hypertensives relative to normotensives.

Methods

Participants

The current study is a secondary analysis of a project assessing lipid stress reactivity in essential hypertension [12] and was formally approved by the Ethics Committee of the State of Zurich, Switzerland. Due to known gender and menstrual cycle phase related alterations in endocrine stress reactivity [25] we recruited men only. Of a total of 45 participants, 35 men completed our attribution questionnaire. All participants provided written informed consent. With the aid of the Swiss Red Cross of Zurich and through advertisements, we recruited nonsmoking hypertensive and normotensive men who, apart from hypertension, were otherwise in good physical and mental health, as confirmed by an extensive health questionnaire and telephone interview. Specific exclusion criteria, obtained by participants' self-report, were as follows: regular strenuous exercise, alcohol and illicit drug abuse, any heart disease, varicosis or thrombotic diseases, elevated blood glucose level and diabetes, elevated cholesterol level, liver and renal diseases, chronic obstructive pulmonary disease, allergies and atopic diathesis, rheumatic diseases, and current infectious diseases. In addition, participants were included only if they reported no regular or occasional intake of medications. If the personal or medication history was not conclusive, the participants' primary care physician was contacted for clarification.

Assessment of hypertension

After a 15-minute rest, three seated screening blood pressure (BP) measurements were obtained on three separate days by a fully automated sphygmomanometry device (Omron 773; Omron Healthcare Europe, Hoofddorp, The Netherlands) and the average BP was computed. Participants were categorized into hypertensive and normotensive individuals following the World Health Organization/International Society of Hypertension definition (systolic BP $\geq$ 140 mm Hg and/or diastolic BP $\geq$ 90 mm Hg) [26]. The screening procedure yielded 17 hypertensive and 18 age-matched normotensive men (all with complete attributional style data) whose characteristics are listed in Table 1. The average mean arterial pressure (MAP) across all individuals according to the formula two-thirds diastolic BP + one-third systolic BP was used for analysis.

Psychosocial stress procedure

All experimental sessions commenced between 2 pm and 4 pm and lasted for approximately 2 h. Participants abstained from food and drinks (other than water) for 2 h before the experiment, and from physical exercise, alcohol, and caffeinated beverages starting the evening before the test day. To inflict psychosocial stress, we used the Trier Social Stress Test (TSST). The TSST combines a 5-minute preparation phase followed by a 5-minute mock job-interview, and a 5-minute mental arithmetic task in front of an audience [24]. The TSST can be viewed as a situation of experimentally induced failure [27] that reliably provokes profound endocrine and cardiovascular responses [24,28]. During the 45 min before introduction to the TSST and for another 60 min after task completion, participants remained seated in a quiet room.

Blood for lipid and norepinephrine (NE) measures was obtained immediately before stress, immediately after stress, and at 20 min, and 60 min after stress. At the end of blood sampling, participants were debriefed and participation was remunerated with 80 Swiss francs.

Measurements

External and internal attribution of failure

External and internal attribution of failure was measured by the German questionnaire for the assessment of causal attributions (IE-SV-F; questionnaire for the assessment of internal/external and stable/instable attributions depending on success and failure [Fragebogen zur Erfassung der internalen/externalen und stabilen/variablen Attributionen in Abhängigkeit von Erfolg und Misserfolg] [29]). Using a 5-point rating scale ranging from 1 (not at all) to 5 (absolutely), subjects were asked to rate the extent to which the statements for the given situations of failure applied to themselves (e.g. to be more criticized at work is just a coincidence and is independent of my work performance). The subscales external-*stable* attribution of failure (consisting of 10 items) and external-*variable* attribution of failure (consisting of 9 items) assess the habitual tendency to explain negative events by external-stable causes (e.g. task difficulty) or external-variable causes (e.g. bad luck), respectively. The subscales internal-*stable* attribution of failure (consisting of 10 items) and internal-*variable* attribution (consisting of 11 items) assess the habitual tendency to explain negative events by internal-stable causes (e.g. low ability) or internal-variable causes (e.g. lack of effort), respectively. Higher scores reflect a higher tendency to attribute failure to external-stable, external-variable, internal-stable, or internal-variable causes. Cronbach's alpha ($n = 174$) was 0.68 (external-stable), 0.69

Please cite this article as: Kuebler U, et al, Attributional styles and stress-related atherogenic plasma lipid reactivity in essential hypertension, J Psychosom Res (2014), http://dx.doi.org/10.1016/j.jpsychores.2014.04.004