Personal and Interpersonal Factors Moderate the Relation Between Human-Made Trauma and Hypertension: A Path Analysis Approach

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ABSTRACT

Background: Although the link between trauma, posttraumatic stress disorder (PTSD), and hypertension is established, its underlying mechanisms remain underexplored.

Objective: This study tested a theoretical model exploring the moderating influence of psychological (emotion regulation) and interpersonal (social support) factors on the mediation between trauma and hypertension, through PTSD.

Methods: We measured these variables through self-reported questionnaire on 212 patients, recruited from internal medicine in a general hospital of Bukavu, a region affected by more than 25 years of armed conflicts. We first evaluated the PTSD mediation in the absence of moderators, before testing each moderator using moderated path analysis.

Results: Results showed that PTSD partially mediates the relationship between human-made trauma and hypertension, whereas social support and maladaptive emotion regulation moderate the relationship between human-made trauma and PTSD.

Conclusions: The relationship between human-made trauma, PTSD, and hypertension might be modulated by psychological and interpersonal factors, which paves the way for new interventions targeting emotion regulation and social support to reduce PTSD and hypertension in populations exposed to human-made violence.

Key words: hypertension, trauma, posttraumatic stress disorder, social support, regulation of emotion, Democratic Republic of Congo.

INTRODUCTION

Arterial hypertension affects 1.39 billion individuals worldwide (1) and is a major cause of morbidity/mortality (2). Arterial hypertension is associated with potential biological causes and psychological risk factors (3). Among psychological factors, there is a growing body of evidence highlighting the association between posttraumatic stress disorder (PTSD) and hypertension (4–7), but the mechanisms underlying this association are still poorly understood. Moreover, most studies focused on military populations (5) or on patients with resistant hypertension (8) in developed countries, whereas 75% (1.04 billion) of hypertensive individuals live in low- and middle-income countries (1), where important conflicts take place and could generate PTSD. Therefore, there is a need to test the importance of PTSD and to identify other psychological factors related to hypertension in such populations.

The Eastern territories of the Democratic Republic of Congo are among the most war-affected regions worldwide, with a cumulative history of 30 years of armed conflicts that resulted in millions of deaths (9). This war exposure led to a high prevalence of PTSD (41%) (10). Furthermore, hypertension is also highly prevalent, both in urban (41%) and rural (38%) areas (11). We have recently shown in this population that, compared with nonhypertensive patients, hypertensive patients presented strikingly increased rates of exposures to traumatic events, particularly human-made traumatic situations (12). The present study further explored the link between human-made trauma and hypertension, by determining the moderating role played by personal (emotion regulation) and interpersonal (social support) factors in this relation, which was not explored in our previous article. Hence, in the present work, we went beyond the inferential analyses developed in our previous work to test a moderation/mediation model including the variables considered as a determinant in the relationship between exposure to trauma and hypertension. We used a moderated path analysis to

PDS-F = Post-traumatic Diagnostic Scale—French adaptation, **PTSD** = posttraumatic stress disorder

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test the model, a general analytical framework that combines moderation and mediation integrating moderated regression analysis and path analysis (13). It indicates how the direct, indirect, or mediated paths are influenced by moderator variables, thus going far beyond our previous reports. Such approaches indeed allow a better discrimination regarding how variables interact, and might open more clinical implications.

Building on the biopsychosocial framework (14), we focused on two key potential psychosocial moderators of the relation between trauma and hypertension. At the personal level, emotion regulation has emerged as a major concept in mental health. It is defined as the extrinsic and intrinsic processes monitoring, evaluating, and modifying emotional reactions, especially their intensity and temporality, to accomplish one's goals (15). Emotion regulation can be adaptive or maladaptive (16), maladaptive emotion regulation strategies being related to psychological and physiological consequences (17), including PTSD (18). At the interpersonal level, social support is defined as the help accessible to an individual through social ties with other individuals or groups (19). As for emotion regulation, social support is associated with both physical and mental health outcomes (20) and constitutes a preventive factor regarding the emergence of PTSD after a trauma (21). Besides, hypertension is associated with both emotion regulation (22,23) and social support (24).

Here, we tested the influence of PTSD (as a mediator), emotion regulation, and social support (as moderators of the "trauma–PTSD" relation) on the relation between human-made trauma and hypertension through a moderated path analysis (13). We hypothesized that PTSD constitutes a mediator accounting for the link between human-made trauma and hypertension (Hypothesis 1), and that adaptive emotion regulation (Hypothesis 2) and social support (Hypothesis 3) moderate that relationship.

METHODS

Procedures

We recruited adult outpatients consulting at the internal medicine department of the General Hospital of Bukavu between January 2018 and December 2019 who were invited to participate. All the recruitment process is described in our previous article (12). Ethical approval for the study was obtained from the Catholic University of Bukavu Research Ethics committee under the reference UCB/FACMED/CE/149/018. Written informed consent was obtained from the participants, and privacy and confidentiality of the participants were ensured.

Participants

Among the 212 participants, 106 (50%) fulfilled the diagnosis criteria of hypertension. Most patients were women (67%). Ages ranged from 20 to 66 years (mean [standard deviation] = 42.5 [13.6] years). Most participants were married (52%). Among people who met the inclusion criteria, only 10 (6 women, and 2 with hypertension) declined to be part of the study. All patients with hypertension were on treatment (median number of drugs = 2; interquartile range = 1). More than half (61.3%) of hypertensive patients were on angiotensin-converting inhibitors, 23.5% were on angiotensin receptor blockers, 73.6% were on calcium antagonists, 9.4% were on diuretics and 30.2% were on β-blockers.

Measures

We used a locally generated questionnaire to collect sociodemographic data including age, sex, and marital status. Data were collected by five medical students using a structured questionnaire. Students were trained by the principal investigator (AMB) during a 3-day session, emphasizing the theoretical and practical aspects of the questionnaire, informed consent, and participant confidentiality.

Blood Pressure Measurement

The diagnosis of hypertension was made for patients who had already a diagnosis of hypertension according to the European Society of Cardiology/European Society of Hypertension definition (2) and/or were already on antihypertensive medication(s). This includes repeated BP measurements on different occasions. Hypertension was defined as office systolic blood pressure values at least 140 mm Hg and/or diastolic blood pressure values at least 90 mm Hg at repeated BP measurements on different occasions (2). We measured blood pressure at the internal medicine consultation using a validated Omron HEM-907 digital monitor (25). We performed analyses on the mean of three consecutive measurements.

Psychological Assessment

The Post-traumatic Diagnostic Scale—French adaptation (PDS-F), and the Stressful Events Scale measured PTSD and past traumatic events. These scales assess the types and magnitude of a wide variety of traumatic events, and PTSD (26). PTSD items are based on the validated *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (27) criteria and include exposure to traumatic events, and symptoms of reexperiencing, of avoiding, and of hyperarousal signs. The PDS-F is interpreted with a severity score ranging from 0 to 51, obtained by adding up items' responses. For the PTSD diagnosis to be made, we considered the cutoff for moderate to severe symptoms, rating > 20 as reported by McCarthy (28). The PDS included the instruction of identifying an index trauma and rating each problem according to the traumatic event indexed. The traumatic events in this study have been grouped in human-made traumatic events (e.g., physical assault, sexual assault, experience of combat, victim of hostage-taking or kidnapping) and non–human-made traumatic events (e.g., accident, environmental disaster, animal aggression, serious health problem, death of a relative; Table S2, Supplemental Digital Content, http://links.lww.com/PSYMED/A947). For each event, participants had to report its frequency (on a scale from 0 to 3) and its related stress (on scale from 0 to 7). Event intensity was then calculated as the mean of product of frequency by stress. In our previous work, we found that only human-made traumatic event was associated with hypertension (12).

The Cognitive Emotional Regulation Questionnaire assessed strategies of emotion regulation. It is a 36-item validated questionnaire capturing five adaptive (acceptance, positive refocusing, refocus on planning, positive reappraisal, putting into perspective) and four nonadaptive (self-blame, rumination, catastrophizing, blaming others) stable-dispositional strategies to regulate emotions (29).

The Questionnaire of the Frequency of and Satisfaction with Social Support assessed frequency and degree of satisfaction regarding perceived emotional, instrumental, and informational support received from four different sources (partners, family, friends, community) (30).

Statistical Analysis

We first tested Hypothesis 1 by assessing the mediation without moderation. We first assessed whether human-made trauma (the predictor variable) was associated with hypertension. Then, we assessed if this association was mediated by PTSD. The mediating effect is expressed in structural equation modeling as an indirect effect and is significant on the basis of 95% confidence of unstandardized coefficient estimates through generating 1000 bootstrap coefficient estimates (31). We performed path analysis with AMOS 29. We assessed Hypotheses 2 and 3 by using moderated path analysis. We analyzed the data separately for each moderator including precursor and moderator in each analysis. We analyzed the total effect moderation model as described by Edwards and Lambert (13). We centered all measures before all analyses (32). We used IBM SPSS version 26 to estimate all the coefficients, including those from bootstrap samples. We analyzed data for the simple effects using man-made trauma as a predictor. Then we followed the procedure described by Edwards and Lambert (13) to compute simple effects for direct, indirect, and total effects at low and high values of each of the mean-centered moderator variables (1 standard deviation below and above the mean). We computed the difference in simple effects by subtracting the effects for high values of each moderator from the effect for low values. We used 95% confidence intervals of unstandardized coefficient estimates to test differences for direct effects. We used bias-corrected 95% confidence intervals derived from 1000 bootstrap coefficient estimates to test differences for the indirect and total effects.

RESULTS

Descriptive Statistics

We included 212 participants, among which 143 (67%) were women, 110 (52%) were married, and 129 (61%) were Catholics (Table 1). Hypertensive and normotensive patients did not differ in terms of demographic factors. All patients with hypertension were under treatment (median number of drugs taken = 2; interquartile range = 1). Besides, none of the patients of the control

TABLE 1. Demographic and Clinical Parameters in HPs Versus NCs (N = 212)

Variables	Total	HP	NC	p
Age, y	42.5 (13.6)	42.4 (13.5)	42.6 (14.6)	.42
Women, *n* (%)	143 (67)	60 (57)	83 (78)	.23
Married, *n* (%)	110 (52)	48 (45)	62 (58)	.43
Systolic blood pressure, mm Hg	131 (15)	141 (12)	121 (10)	<.001
Diastolic blood pressure, mm Hg	79 (8)	82 (7)	75 (8)	<.001
Traumatic event frequency, *n* (%)	9.76 (3.30)	11.15 (3.39)	8.38 (2.64)	<.001
Traumatic event intensity, *n* (%)	37.76 (14.87)	46.00 (14.56)	29.53 (10.25)	<.001
Human-made trauma, *n* (%)	78 (37)	65 (61)	13 (12)	<.001
Non–human-made trauma, *n* (%)	66 (31)	39 (36)	27 (25)	.121
Adaptive emotion regulation, *n* (%)				
Acceptance	6.53 (4.15)	6.13 (4.21)	6.93 (3.97)	.082
Positive refocusing	9.72 (4.92)	8.46 (4.88)	10.98 (4.94)	.132
Refocus on planning	8.40 (4.11)	8.59 (4.12)	8.21 (4.11)	.153
Positive reappraisal	7.73 (3.67)	5.02 (3.69)	10.44 (3.67)	.043
Putting into perspective	8.71 (3.78)	7.92 (3.80)	9.5 (3.77)	.158
Global Adaptive emotion regulation	41.09 (5.28)	36.12 (6.21)	46.06 (5.29)	.052
Maladaptive emotion regulation, *n* (%)				
Self-blame	8.26 (3.23)	10.42 (3.25)	6.10 (3.22)	<.001
Rumination	9.73 (4.50)	11.25 (4.52)	8.21 (4.48)	<.001
Catastrophizing	10.57 (3.43)	12.87 (3.45)	8.27 (3.46)	.034
Blaming others	8.37 (4.34)	8.21 (4.37)	8.53 (4.36)	.119
Global maladaptive emotion regulation	36.93 (5.24)	42.75 (6.22)	31.11 (5.07)	.021
Social support, *n* (%)				
Emotional support	32.85 (15.05)	24.85 (14.12)	40.85 (15.26)	<.001
Instrumental support	20.81 (13.90)	18.24 (12.54)	23.38 (14.02)	.082
Informational support	19.83 (11.41)	14.87 (11.87)	24.79 (11.26)	<.001
Global social support	73.49 (23.24)	57.96 (22.59)	89.02 (25.14)	<.001
PDS score	15.76 (4.52)	18.02 (5.14)	13.50 (4.08)	<.001
PTSD, *n* (%)	42 (19)	36 (33)	6 (7)	<.001

HPs = hypertensive patients; NCs = normotensive controls; PDS = Post-traumatic Diagnostic Scale.

Values are expressed as mean (standard deviation), unless otherwise indicated.

group had taken antihypertensive drugs. When compared with normotensive subjects, patients with hypertension scored higher to both frequency of traumatic events, particularly human-made traumatic events, and intensity of stress related to traumatic events. They were more likely to suffer from PTSD. Regarding emotion regulation, patients with hypertension were more likely to use maladaptive (self-blame, rumination, catastrophizing) strategies and less likely to use adaptive ones (positive reappraisal). For descriptive statistics and Pearson's correlation for all study variables, see Table S1, Supplemental Digital Content, http://links.lww.com/PSYMED/A947.

Hypothesis 1: Mediation by PTSD

In the linear regression, human-made trauma was associated with PTSD (= 0.63, $p = .015$). In the logistic regression, hypertension was associated with human-made trauma (2.74 [1.98–9.06]) and PTSD (4.74 [1.54–14.06]). We tested mediation using a structural equation model, with PTSD mediating the relationship between human-made trauma and hypertension. This model showed a good fit ($\chi^2 = 12.05$; $df = 4$; root mean square error of approximation = 0.05; Tucker-Lewis index = 0.94; standardized root mean square residual = 0.03), supporting Hypothesis 1 (Figure 1). The PTSD mediation was, however, partial, as the relationship between human-made trauma and hypertension remained significant (= 0.19, $p < .01$).

Hypotheses 2–3: Moderation by emotion regulation and social support (see Tables S3 and S4, Figure S1, Supplemental Digital Content, http://links.lww.com/PSYMED/A947)

Adaptive emotion regulation did not moderate either the indirect path (from human-made trauma to PTSD and from PTSD to hypertension) or the direct effect (from human-made trauma to hypertension), contradicting Hypothesis 2. However, maladaptive emotion regulation only moderated the indirect path. As hypothesized, maladaptive regulation of emotion increased the slopes of the relationship between human-made trauma and PTSD. Social support moderated only the indirect path (from human-made trauma to PTSD). As hypothesized, social support reduced the slopes of the relationship between human-made trauma and PTSD.

DISCUSSION

Data fully supported the proposal that PTSD partially mediated the relationship between human-made trauma and hypertension (Hypothesis 1) and that social support moderated the relationship between human-made trauma and PTSD (Hypothesis 3). Regarding the role of emotion regulation, maladaptive emotion regulation (but not adaptive one) moderated the relationship between human-made trauma and PTSD (Hypothesis 2). A first main result is the confirmation, in a nonmilitary sub-Saharan African population, that PTSD is strongly related to human-made trauma and hypertension, but also mediates their relationship. We thus extended previous studies conducted in Western military populations (4–7) and discriminated for the first time two paths through which a traumatic event can be related to hypertension: a direct (from human-made trauma to hypertension (4)) and an indirect one (from human-made trauma to hypertension through PTSD). Both paths should thus be considered when conceptualizing how human-made trauma can be related to hypertension and developing preventive interventions of hypertension in populations exposed to violence.

The second key result is the moderating role played by intrapersonal (emotion regulation) and interpersonal (social support) factors in the relation between human-made trauma and PTSD. First, our data showed that PTSD was strongly and negatively related to maladaptive emotion regulation strategies, whereas it was only weakly and positively related to adaptive ones, in line with previous results (18,33,34). We were, however, the first to explore the moderating role of such emotion regulation strategies on the relation between human-made trauma and PTSD. We observed that maladaptive emotion regulation moderated this relation. Second, although there is a growing body of evidence assessing the link between social support and mental or physical outcomes (20,35–37), only few studies assessed the association between social support and PTSD (21,38). Our findings extend these results by suggesting that social support might be a powerful prevention factor to avoid PTSD, when the traumatogenic experience has

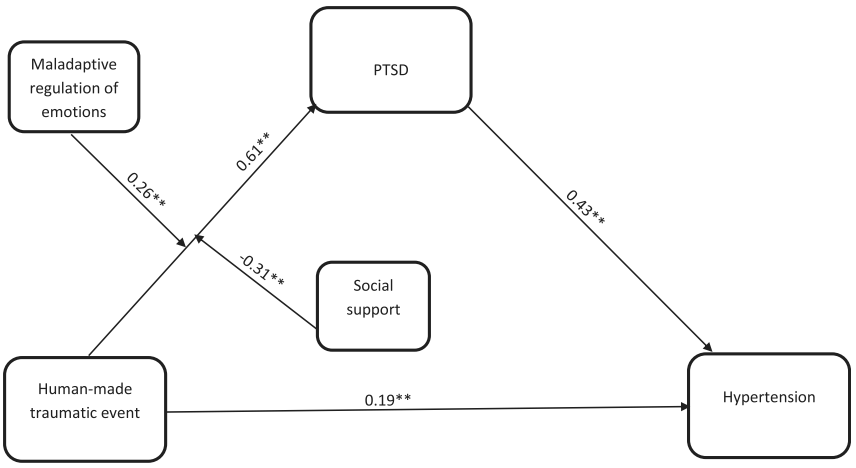

FIGURE 1. Results related to the theoretical model. All coefficients are standardized. **$p < .01$. PTSD = posttraumatic stress disorder

been elicited by humans. This leads to the proposal that the quality of the human support may somehow moderate the brutality of the human-elicited aggression and prevent it from inducing PTSD.

The present study has some limitations. Being a cross-sectional study, we could not establish a causal relationship between explored variables. Particularly, because we did not assess onsets of hypertension and trauma exposure, it is not clear whether hypertension began before trauma exposure or conversely. Even more, the fact that some participants might have been currently exposed to trauma at testing time might have an impact on blood pressure. Besides, the fact that we used self-reported questionnaire might have involved some biases such as false negatives (with participants finding other cultural explanations of their symptoms, other than the traumatic event) (39). However, we minimized the possibility of having false positives of PTSD using the PDS, as our assistant researchers were trained on theoretical and practical aspect of the diagnosis and insisted on the instruction of linking the symptoms to the traumatic events. Moreover, in the region of our study, most of the inhabitants have in fact experienced one or more traumatic events and tend, for cultural reasons, to underestimate rather than overestimate the presence of PTSD (40). Finally, we cannot exclude that unmeasured somatic or psychiatric comorbidities might be present in our sample and thus constitute confounding factors through their effect on hypertension. The fact that other somatic and psychiatric diagnoses were not systematically assessed in both hypertensive patients and normotensive control group is certainly a limitation in our study. Nevertheless, our study suggests that the association between human-made trauma and hypertension in population exposed to trauma may involve the mediation of PTSD and the moderation of both emotion regulation and social support. At the theoretical level, this study sheds light on potential mechanism explaining the transition between human-made trauma and hypertension. At the clinical level, it suggests that the nature of the intervention proposed to patients suffering from hypertension in war zones should be renewed, by adding psychosocial interventions to antihypertensive medications. However, in view of the limitations of the present study; our hypothesis should be assessed in larger sample with longitudinal design.

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Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

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