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Acute Experiences of Negative Interpersonal Interactions: Examining the Dynamics of Negative Mood and Ambulatory Blood Pressure Responses Among Black and Hispanic Urban Adults

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Transparency Statements In terms of transparency statements, neither this study nor the analysis plan were formally pre-registered. De-identified data from this study are not available in an a public archive. De-identified data from this study will be made available (as allowable according to institutional IRB standards) by emailing the corresponding author. Analytic code used to conduct the analyses presented in this study, as well as study materials related to the ecological momentary assessment questions, are available in a public archive: https://osf.io/d4xzk/?view_only=f3c196eff1034db98b30f9a8af98788d.

Abstract

Background Negative interpersonal interactions are associated with acute increases in ambulatory blood pressure (ABP). Yet, the mechanisms underlying this relationship are unclear.

Purpose This study tested whether negative interpersonal interactions predict higher ABP both in the moment and during subsequent observations, and whether increases in negative mood mediate these relations. These associations were tested among Black and Hispanic urban adults who may be at higher risk for negative interpersonal interactions as a function of discrimination. Race/ethnicity and lifetime discrimination were tested as moderators.

Methods Using a 24-hr ecological momentary assessment (EMA) design, 565 Black and Hispanic participants (aged 23–65, M = 39.06, SD = 9.35; 51.68% men) had their ABP assessed every 20 min during daytime accompanied by an assessment of negative interpersonal interactions and mood. This produced 12,171 paired assessments of ABP and self-reports of participants' interpersonal interactions, including how much the interaction made them feel left out, harassed, and treated unfairly, as well as how angry, nervous, and sad they felt.

Results Multilevel models revealed that more intense negative interpersonal interactions predicted higher momentary ABP. Mediation analyses revealed that increased negative mood explained the relationship between negative interpersonal interactions and ABP in concurrent and lagged analyses. Discrimination was associated with more negative interpersonal interactions, but neither race/ethnicity nor lifetime discrimination moderated findings.

Conclusions Results provide a clearer understanding of the psychobiological mechanisms through which interpersonal interactions influence cardiovascular health and may contribute to health disparities. Implications include the potential for just-in-time interventions to provide mood restoring resources after negative interactions.

Lay summary

Being mistreated by others has been shown to have negative impacts on cardiovascular health, including higher blood pressure (BP) levels. Yet, it is not clear why this mistreatment leads to increased and sustained influences on BP. In this paper, among a sample of Black and Hispanic urban adults, we studied whether changes in negative mood after being treated unfairly, excluded, or harassed explained the reason for higher BP levels. Participants completed reports of how they were treated in recent social interactions, and their levels of negative mood they were feeling at the current moment, every 20 min for 1 day. A BP measurement also occurred at each measurement. We found that negative mood was higher when a person reported being treated unfairly, excluded, and/or harassed, and that the negative mood that followed these negative interpersonal interactions accounted for increases in BP. These results have implications for how mistreatment can lead to chronic illness over time, and provides the potential for providing resources to restore mood and improve BP after mistreatment.

Keywords Ambulatory blood pressure · Negative interpersonal interaction · Social interaction · Ecological momentary assessment · Negative mood · Stress

Negative interpersonal interactions involve mistreatment in which one person withdraws positive actions and/or initiates and maintains negative actions towards another [1]. These negative interpersonal interactions can take different forms, including acts of exclusion, unfair treatment, or harassment [2]. Cross-sectional research suggests that people who report more chronic exposure to interpersonal mistreatment have worse cardiovascular profiles, including higher blood pressure (BP) [3, 4]. Laboratory studies demonstrate acute experiences of mistreatment causally relate to increases in negative mood and BP [5–7]. Yet, there are gaps in knowledge about the relationship between mistreatment and changes in BP, including limited understanding of the psychobiological correlates and consequences of daily experiences of maltreatment in everyday life. Further, despite theoretical and empirical linkages suggesting that negative mood occurs after negative interpersonal interactions and can predict BP levels, it is unclear whether changes in mood during and/or soon after negative interpersonal interactions mediate the association between such interactions and ambulatory blood pressure (ABP). Understanding the potential mediating role of mood states has clinical utility as mood states are potentially modifiable and could be a target for intervention. Thus, this paper examines the extent to which momentary negative interpersonal interactions in daily life are associated with momentary increases in ABP, and whether increases in momentary negative mood mediate such associations. We examine these associations in a sample of Black and Hispanic urban adults, people who may face higher rates of negative interpersonal interactions as a function of racial and ethnic discrimination [8, 9]. Further, Black adults are also at higher risk for hypertension and other forms of cardiovascular disease, increasing the public health significance of any such observed associations [10].

Negative Interpersonal Interactions in Everyday Life

In prior research, researchers found that higher average levels of daily negative interpersonal interactions were associated with higher average levels of daily ABP [11]. Although a strength of this past work was its use of a daily diary design, data were aggregated and analyzed at the between-person level. Such between-person research is critical to demonstrate who is at risk for future cardiovascular disease. However, between-person analyses do not tell the researcher why or when this association exists [12].

To advance a mechanistic understanding of these associations, within-person methodological approaches, such as ecological momentary assessment (EMA), are needed to determine how ABP changes when a given individual does and does not experience a negative interpersonal interaction. There is limited existing research using within-person approaches to examine the relations of negative interactions to cardiovascular reactivity and recovery in everyday life. For example, both Black and White adolescents who reported unfair treatment (e.g., being treated with less respect) throughout the day had elevated ABP in those moments when the unfair treatment occurred [3]. Other work indicates that perceived discrimination among Black and White adults predicts momentary increases in daytime ABP in response to interpersonal stress [13], suggesting that chronic exposure to unfair treatment sensitizes people to future mistreatment. With both studies, however, the trajectory of ABP responses following unfair treatment is unclear.

Is Negative Mood a Mediator?

Data from a variety of studies suggest that intrapersonal mechanisms, including negative mood, may mediate a connection between negative interpersonal interactions and elevated ABP. First, negative interpersonal interactions predict mood states. A meta-analysis found that social exclusion was followed by a consistent shift to more negative mood states [14]. Additionally, an EMA study of working adults revealed social interactions that made participants feel excluded or treated unfairly (versus other forms of interactions) were followed by reports of increased sadness [15]. Second, higher levels of negative mood are positively associated with higher levels of ABP in everyday life. For example, in a study of community adults, momentary mood that was characterized as having a more negative valence was associated with greater systolic ABP than mood in moments with less negative valence [16]. Other work has shown that more negative emotional states like anxiety [17], or a composite of negative affect [18], are associated with higher ABP levels than less intense states. However, no work to our knowledge has examined whether negative mood mediates associations between negative interpersonal interactions and ABP.

When examining mediation, it is important to consider the potential that relationships unfold over time. Negative interpersonal interactions might relate to mood when the interaction first occurs, but also influence mood for a sustained period following the interaction. In turn, both acute and sustained mood states may be linked to ABP. Theoretical explanations for the relations between negative interpersonal interactions and disease often suggest the possibility that negative interpersonal interactions evoke prolonged changes in mood and/or BP. Yet, this temporal approach to the study of the effects of negative interpersonal interactions has largely been ignored in empirical studies [19]. The use of EMA paradigms can permit us to investigate not only if negative interactions have concurrent relationships with mood and ABP, but also if these interactions result in more sustained changes

in mood that are associated with elevations in ABP. These data can potentially provide novel evidence for direct and indirect pathways by which negative interpersonal interactions can lead to other downstream effects on health.

Race/Ethnicity and Discrimination as Potential Moderators

In the present research, all participants identified as either Black or Hispanic and reported on lifetime discrimination at the intake session. Past work has found that those with higher levels of lifetime discrimination reported higher levels of diastolic ABP [20, 21]. Expanding beyond this initial finding, it is possible that those who have experienced more lifetime discrimination may be sensitized to negative interpersonal interactions resulting in greater increases in BP responses to interpersonal maltreatment [22, 23]. Over time, chronically elevated BP may be, in part, a function of repeated occurrences of acute BP reactions and delayed recovery.

The Present Study

The present study utilized an EMA design in which participants reported on negative interpersonal interactions and mood every 20 min during daytime hours over a 24-hr period while ABP was concurrently assessed. The use of EMA to repeatedly assess negative social interactions and mood has the benefit of reducing recall bias and responses influenced by social desirability [24]. This is particularly important with negative interpersonal interactions because an individual's perceptions of the social interactions, which are often complex social stressors, could change over time as they cope with these events or as the situation evolves [25]. We focused on ABP as it is a unique and often superior predictor of cardiovascular disease and mortality in comparison to lab-based and clinic BP [26]. Moreover, moment-to-moment elevations in ABP are also predictive of cardiovascular disease over time [27]. We implemented 20-min time periods based on field and laboratory research demonstrating that mood [28] and BP [29] effects can last for at least this time period. Moreover, although ABP can be quick to recover in response to negative stimuli [30], the reactivity of ABP to negative stimuli makes it a strong candidate to study in relation to negative mood [16]. The proposed mediational models thus have the potential to show both the immediate and prolonged effects of negative interpersonal interactions, which may act through negative mood, to put extended burden on the cardiovascular system. As such, Research Question (RQ) 1 tested the hypothesis that there would be direct effects of negative interpersonal interactions on momentary levels of ABP. RQ2 tested the hypothesis that negative mood would mediate the relationship between negative interpersonal interactions and ABP, both in concurrent assessment models and in lagged analyses testing a cascading effect over time. RQ3 tested whether greater lifetime discrimination would be associated with a stronger connection between negative interpersonal interactions and ABP.

Methods

Participants

Participants were recruited for a study of racism, coping, and BP [31]. An initial sample of 670 participants were recruited, of whom 644 had at least some data for ABP and EMA. Additionally, participants needed at least six ABP observations and EMAs to ensure stability of the measures and to provide a reliable person-level estimate [32]. The final sample (n = 565) consisted of participants who identified as either Black (302; 53.45%) or Hispanic (263; 46.55%) and were all U.S.-born English-speaking adults between the ages of 23 and 65 (M = 39.06, SD = 9.35); about half were men (292, 51.68%). Participants average annual income was $20,114 ($SD$ = $29,860). The sample had the following levels of education: only grade school (18; 3.19%), some high school education (149; 26.37%), obtained a high school degree (142, 25.13%), some college education (119; 21.06%), technical school training (29; 5.13%), obtained a college degree (70; 12.39%), or at least some graduate school training (38; 6.73%). Finally, participants had an average body mass index (BMI) of 28.05 (SD = 5.35).

Recruitment took place in the New York City metropolitan area. Participants were recruited by Clinical Directors Network (CDN), a practice-based research network. CDN enabled us to collaborate with local community health centers and to recruit participants through their sites (e.g., visits to the waiting rooms and posting flyers). Further, a portable lab was established on an urban campus of the university of one of the authors, and flyers were posted in neighboring venues (e.g., stores, coffee shops). Participants could refer others to join the study.

Inclusion criteria included identifying as American-born Black/African American or American-born Hispanic/Latino(a), being between the ages of 25–65, and being able to read and write at an 8th grade level in English or Spanish. Exclusion criteria included taking medication that affected BP, having a major medical condition that could affect ABP, or having an arm circumference greater than 44 cm (which would have precluded the use of available equipment).

Procedure and Materials

Overview and training

All study materials were approved by the Institutional Review Boards of St. John's University, CDN, Jamaica Hospital Medical Center, and the City University of New York. Participants provided written informed consent prior to participating in any study activities.

At a baseline session, participants completed a brief survey to assess demographics and had height and weight measurements taken. About two weeks after this visit, participants were fitted with a BP cuff and then had eight initial sitting and standing readings taken to train participants on the ABP measurement process and to establish baseline BP. As part of training, participants were instructed on how to terminate or initiate an ABP reading. They were also trained on EMA completion. ABP readings were then taken for the ensuing 24 hrs, every 20 min, with EMA completion occurring with each ABP reading during daytime hours (based on self-reported times when participants reported they would likely be going to sleep). They returned the equipment the next day, after which they were paid $165 and debriefed. Additional measurements not relevant to the present study were collected at all visits. All participants were English-speaking and completed study materials in English (but had the option to complete them in Spanish). All EMA and ABP data were collected on a weekday. Data were collected between October 2003 and March 2007.

Baseline assessment

At baseline, participants provided basic demographic information, including age in years, sex (0 = woman, 1 = man), identification as Latina/o/x or Hispanic, and race identity. The ethnicity and race variables were recoded into a single category (0 = Hispanic, 1 = Black), given that only participants identifying as exclusively Black or Hispanic were included in the study. Lifetime discrimination was measured with the 34-item Perceived Ethnic Discrimination Questionnaire—Community Version [33]. This scale measures lifetime experiences of racial and ethnic discrimination within an interpersonal context. A sample item asks, "Because of your race or ethnicity, how often has someone said something disrespectful, either to your face or behind your back?." Participants responded on a scale from 1 (*never*) to 5 (*very often*). Items were averaged together such that higher numbers indicated more lifetime experiences of perceived discrimination (Cronbach's α = .95). Finally, height and weight were measured to calculate BMI.

Ambulatory blood pressure measurement

Ambulatory blood pressure was collected via the Suntech Accutracker II (Suntech Medical Instruments, Raleigh, NC), which has shown to be a reliable and valid device [34]. To ensure the validity of readings, all data from a particular measurement were deleted if any of the following conditions were met [31, 35]: (a) the difference between systolic ABP and diastolic ABP readings was either less than 20 mm Hg or greater than 90 mm Hg, (b) a reading was accompanied by an error code from the device, and/or (c) if systolic ABP was less than 85 mm Hg or greater than 196 mm Hg, if diastolic ABP was less than 41 mm Hg or greater than 130 mm Hg, and/or if heart rate was less than 46, or greater than 130, beats per minute. There was an average of 30.89 (*SD* = 9.16, range = 6–58) readings per participant.

Ecological momentary assessment

After each daytime ABP measure, participants were instructed to complete the EMA. EMAs were administered using a personal device assistant (SONY CLIE) with the Quest Admin Program [36]. Assessments were date- and time-stamped. Picture icons accompanied the written questions to facilitate interpretation and response.

For negative interpersonal interactions, participants reported if they were talking with anyone when the timer went off (0 = no, 1 = yes). When participants indicated yes, they then responded on a 0–100 scale to three items assessing the degree of their perceptions of the interactions: "Did you feel [left out/ignored, harassed, treated unfairly]." These three items were correlated at moderate levels at the within-person momentary level (.53 to .59). As such, to examine an overall effect of negative interpersonal interactions, we created an average at each moment, with higher numbers indicating the interaction was perceived as more intensely negative. This scale demonstrated moderate reliability to detect within-subject differences in change over time, R_C = .53 [37]. In the same moments, mood was measured on a 0–100 scale asking, "When the timer went off did you feel [angry, nervous, sad]." For each assessment, the negative mood items were averaged together, with higher numbers indicating more negative mood; this scale demonstrated moderate reliability to detect change over time, R_C = .52.

To control for potential momentary influences on ABP, participants reported on their current posture from the following options: reclining (0), sitting (1), standing (2), walking (3), or running (4). Additionally, participants reported if they had eaten, smoked, drunk caffeine, and/or consumed alcohol since the last observation (i.e., 20 min earlier), with each item coded as no (0) or yes (1). There was an average of 21.65 (*SD* = 9.13, range = 6–42) EMA readings per participant. In the data processing stage, EMAs were merged with the accompanying ABP readings if the assessment occurred within 5 min of the reading, resulting in 12,171 paired ABP and EMA moments.

Analytic Plan

To test RQ1, we conducted multilevel models to account for the nested nature of observations within people. Models examined whether negative interpersonal interactions predicted systolic and/or diastolic ABP, testing both between-person and within-person effects. At the between-person level, negative interpersonal interactions were entered as an averaged value across all moments to test whether those who experienced more intense negative interpersonal interactions were also those with worse mood/ABP. At the within-person level, negative interpersonal interactions were entered as a person-mean centered value to test whether experiencing more intense negative interpersonal interactions in a moment than was typical for that person resulted in higher levels of negative mood/ABP in that moment compared to other moments with less intense negative interpersonal interactions for that person.

All models controlled for the person-level factors of age, sex, race/ethnicity, and BMI. At the momentary level, models controlled for time of day, the posture of the participant at time of measurement, and whether the participant had eaten, smoked, consumed caffeine, and/or drunk alcohol since the last measurement. A random intercept was included to allow the likelihood that participants had differing initial levels of mood and/or ABP. A random slope was specified for the within-person level of negative interpersonal interactions to allow for the possibility that the size of the relationship between negative interpersonal interactions and outcomes differed across participants. An autoregressive covariance structure was included to control for the possibility that observations closer in time to each other were more strongly related to those further apart. Finally, to estimate effect size, pseudo R^2 was calculated by estimating a predicted value for each moment that was then correlated with the actual value [38].

To test RQ2, we adopted two recommended approaches given the lack of consensus for how to perform mediation models for multilevel data. First, in line with a more traditional approach [39, 40], we conducted a series of models that tested (a) whether person-mean centered negative interpersonal interactions predicted negative mood, and (b) whether person-mean centered negative mood predicted systolic ABP and/or diastolic ABP. If significant relationships were observed, (c) we then examined a model in which both person-mean centered negative interpersonal interactions and negative mood were tested in the same model, to assess if negative interpersonal interactions had a direct relationship with ABP. All models used the same control variables as described in RQ1, including both a random intercept and random slope for the predictor of interest, and an

autoregressive covariance structure. Second, we followed procedures to detect indirect effects in which the within-person predictor and mediator were simultaneously modeled as predictors of the within-person outcome—a 1–1–1 mediation model [41]. With this approach, the data were restructured to split the negative interpersonal interactions and mood scores from the same observation to separate rows, thus creating a new data level. A multilevel model was then run with the negative interpersonal interactions and mood predicting ABP, along with a series of dummy coded terms and interaction effects that accounted for the new nested data structure. A macro was then utilized to produce indirect and total effects [42].

To test these processes over time, we ran a similar set of models but now tested negative interpersonal interactions, mood, and ABP at different time points. Specifically, we tested whether ABP at time *t* was predicted by mood at time *t*–1 that was predicted by negative interpersonal interactions at time *t*–2. For these lagged models, we chose not to include lagged ABP in our models in line with perspectives that this variable is very likely correlated with the random components of the multilevel model and thus would violate model assumptions [43, 44]. We continued to model a random intercept and random slope for the predictor of interest, and included the same controls as the models in RQ1.

To test RQ3, we ran two sets of models extending those tested in RQ1. First, to test race/ethnicity as a moderator, we added an interaction term of negative interpersonal interactions at the within-person level with race/ethnicity. Second, to test lifetime discrimination as a moderator, we added both the measure of lifetime discrimination (grand mean centered to aid in interpretation of any observed interaction effects), and an interaction term of negative interpersonal interactions at the within-person level and lifetime discrimination.

Results

Descriptive Statistics

Participants reported talking with someone in about half of all observations (n = 5,680, 46.44%). During these moments, participants reported low average intensity of negative interpersonal interactions but with large variation in these responses (M = 4.68, SD = 13.10). Interactions were perceived as at least minimally negative (i.e., a non-zero report on exclusion, harassment, or unfair treatment) during 25.26% (n = 1,435) of observations with a social interaction. As is typically observed, participants reported relatively low levels of negative mood (M = 6.83, SD = 14.63). Finally, on average participants had pre-hypertensive levels of systolic ABP on average (M = 133.09, SD = 20.91), but normotensive levels of diastolic ABP (M = 79.50, SD = 14.22).

RQ1: Negative Interpersonal Interactions and ABP

RQ1 examined the overall effect of negative interpersonal interactions on ABP. At the between-person level, experiencing more negative interpersonal interactions on average was not significantly related to systolic (t = 0.07, p = .94) nor diastolic ABP (t = 0.45, p = .65) (see Table 1). In contrast, at the within-person level, the intensity of negative interpersonal interactions in the moment was positively associated with the level of systolic ABP (t = 2.02, p = .044) and diastolic ABP (t = 2.31, p = .022).

RQ2: Negative Mood as a Mediator

RQ2 examined whether negative mood functioned as a mediator between negative interpersonal interactions and ABP. All models controlled for the same variables as in RQ1, but only the main pathways of interest are presented in Figs. 1 and 2. Reports of the indirect and total effect are provided in Table 2. For the concurrent assessments, as shown in Fig. 1a and b, more intense levels of negative interpersonal interactions were associated with higher levels of momentary negative mood (t = 10.54, p < .001), and higher levels of momentary negative mood were associated with higher levels of systolic (t = 2.36, p = .019) and diastolic ABP (t = 3.67, p < .001). When both negative interpersonal interactions and negative mood were included in the same model, negative interpersonal interactions were no longer significantly associated with systolic (t = 1.56, p = .12) nor diastolic ABP (t = 1.70, p = .09). A test of indirect effects, displayed in Table 2, indicated negative interpersonal interactions had a significant indirect effect on diastolic ABP through negative mood (t = 3.01, p = .003), but this indirect effect was not significant for systolic ABP (t = 1.01, p = .31). This pattern of results suggests that in concurrent analyses, negative mood mediates the relationship of negative interpersonal interactions on diastolic ABP.

For the lagged assessments, as shown in Fig. 2a and b, greater negative interpersonal interactions at time *t*–2 predicted increased negative mood at time *t*–1 (t = 6.48, p < .001). In turn, higher levels of negative mood at time *t*–1 predicted higher time *t* systolic (t = 2.71, p = .007) and time *t* diastolic ABP (t = 2.82, p = .005). Furthermore, as reported in Table 2, the indirect effect of negative mood was significant in analyses of systolic ABP (t = 2.31, p = .021), but not diastolic ABP (t = 0.67, p = .50). This pattern of results suggest that negative mood mediates the relationship of negative interpersonal interactions on systolic ABP over time.

RQ3: Race/Ethnicity and Lifetime Discrimination as Moderators

We then explored whether lifetime discrimination predicted the level of negative interpersonal interactions at each moment. We used the models as described in RQ1, except with grand mean centered lifetime discrimination predicting levels of negative interpersonal interactions. Those who reported higher levels of lifetime discrimination also had higher levels of negative interpersonal interactions in their daily life, b = 2.06, SE = 0.64, t = 3.23, CI = 0.81–3.32, p = .001.

RQ3 tested whether race/ethnicity or lifetime discrimination moderated associations tested in RQ1. All models were the same as reported earlier except an interaction term of negative interpersonal interactions by race/ethnicity (RQ3a) or lifetime discrimination and an interaction between negative interpersonal interactions by lifetime discrimination (RQ3b) was added. For race/ethnicity, the interaction term was not significant for neither systolic ABP, b = –0.02, SE = 0.06, t = –0.27, CI = –0.14 to 0.11, p = .79, nor diastolic ABP, b = –0.04, SE = 0.05, t = –0.79, CI = –0.14 to 0.06, p = .43, suggesting similar effects for Black and Hispanic participants in the study. Likewise, for lifetime discrimination, the interaction term was not significant for neither systolic ABP, b = 0.004, SE = 0.04, t = 0.09, CI = –0.08 to 0.09, p = .92, nor diastolic ABP, b = 0.02, SE = 0.03, t = 0.54, CI = –0.05 to 0.09, p = .59, suggesting similar effects for those reporting higher versus lower levels of lifetime discrimination. Given

Table 1 Effect of Negative Interpersonal Interactions (NIIs) on Ambulatory Blood Pressure (ABP)

	Systolic ABP			Diastolic ABP		
	b	*SE*	95% CI	*b*	*SE*	95% CI
Random effects						
Intercept	**152.98**	**14.46**	–	**77.72**	**5.94**	–
Intercept * NII	−0.14	0.44	–	−0.09	0.20	–
NII	**0.03**	**0.02**	–	**0.02**	**0.01**	–
Variance	**112.74**	**8.51**	–	**105.13**	**2.28**	–
Autoregression	**0.68**	**0.05**	–	**0.15**	**0.02**	–
Residual	**110.31**	**8.31**	–	**1.02**	**0.00**	–
Fixed effects						
Intercept	**95.66**	**4.36**	**87.10–104.23**	**53.70**	**2.92**	**47.97–59.44**
Age	0.12	0.07	−0.01 to 0.26	**0.19**	**0.05**	**0.10–0.28**
Male	**9.13**	**1.26**	**6.65–11.61**	**4.20**	**0.85**	**2.54–5.86**
Black Race	**3.43**	**1.27**	**0.93–5.93**	**3.18**	**0.85**	**1.51–4.85**
BMI	**0.68**	**0.12**	**0.45–0.91**	**0.38**	**0.08**	**0.22–0.53**
Time	**0.003**	**0.001**	**0.001–0.01**	0.001	0.001	−0.0004 to 0.002
Posture	**2.22**	**0.23**	**1.76–2.68**	**1.91**	**0.17**	**1.57–2.26**
Eating	**1.17**	**0.49**	**0.21–2.14**	**0.76**	**0.37**	**0.04–1.49**
Smoking	**1.44**	**0.59**	**0.28–2.60**	**1.67**	**0.44**	**0.80–2.53**
Caffeine	0.36	0.65	−0.92 to 1.63	−0.40	0.48	−1.36 to 0.54
Alcohol	1.65	1.14	−0.58 to 3.88	**2.69**	**0.82**	**1.09–4.29**
NII (Between-Person)	0.004	0.06	−0.11 to 0.12	0.02	0.04	−0.05 to 0.10
NII (Within-Person)	**0.06**	**0.03**	**0.002–0.12**	**0.05**	**0.02**	**0.01–0.10**
Model statistics						
Pseudo R^2	.104			.100		

Coefficients in bold significant at $p < .05$.

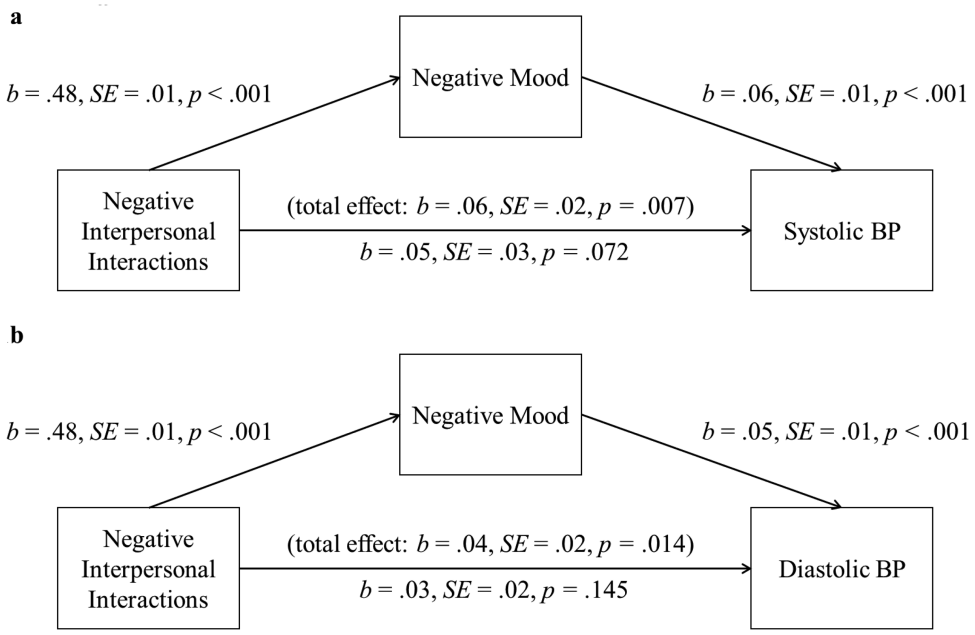

Fig. 1. Negative mood as a mediator of negative interpersonal interactions and ambulatory blood pressure (ABP).

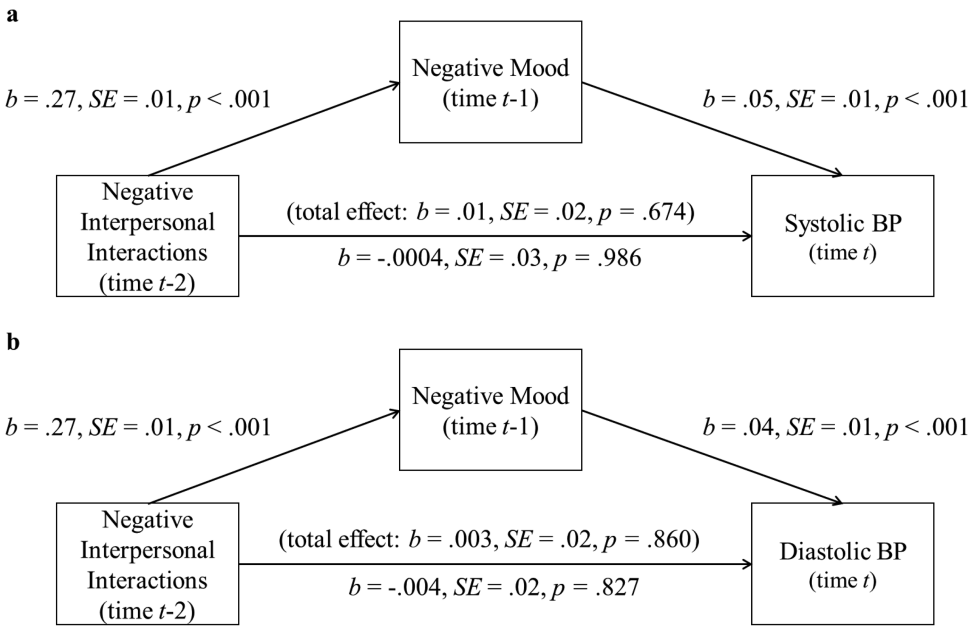

Fig. 2. Time T-1 negative mood as a mediator of time T-2 negative interpersonal interactions and time T ABP.

Table 2 Indirect and Total Effects of Negative Interpersonal Interactions of Systolic and Diastolic ABP through Negative Mood

	Indirect effect			Total effect		
	b	*SE*	CI	*b*	*SE*	CI
Concurrent models						
Negative Interpersonal Interaction → Negative Mood → Systolic ABP	0.02	0.02	−0.02 to 0.15	**0.08**	**0.04**	**0.01–0.15**
Negative Interpersonal Interaction → Negative Mood → Diastolic ABP	**0.04**	**0.01**	**0.01–0.06**	**0.06**	**0.03**	**0.01–0.11**
Lagged models						
Negative Interpersonal Interaction$_{t-2}$ → Negative Mood$_{t-1}$ → Systolic ABP$_t$	**0.02**	**0.01**	**0.004–0.04**	0.03	0.04	−0.04 to 0.10
Negative Interpersonal Interaction$_{t-2}$ → Negative Mood$_{t-1}$ → Diastolic ABP$_t$	0.01	0.01	−0.01 to 0.03	0.004	0.03	−0.04 to 0.05

Coefficients in bold significant at $p < .05$.

the lack of moderation for RQ1, we did not consider additional moderation effects for RQ2.

Discussion

Although prior research has demonstrated that certain types of negative interpersonal interactions can affect ABP and negative mood, we extended this work to examine both ABP reactivity and recovery from negative interpersonal interactions. This research is particularly novel in examining whether changes in mood helped explain connections between negative interpersonal interactions and ABP. We examined these associations in a sample of Black and Hispanic participants, who historically are at high risk for hypertension [45] and increased risk of experiencing negative interpersonal interactions as a function of racial and ethnic discrimination [8, 9], which we also observed in the present study.

We first tested direct relationships between negative interpersonal interactions and ABP. As expected, higher levels of momentary negative interpersonal interactions were associated with higher ABP. These findings support and extend prior work demonstrating the activating effects of negative interpersonal interactions [3, 15, 46]. These findings also help demonstrate how common these experiences are, with negative interpersonal interactions occurring around 12% of all moments, or a bit more than three experiences per each 24-hr assessment period. The relatively high frequency of negative interpersonal interactions in the present research may be in part a function of our use of EMA to capture nuanced and in-the-moment episodes in daily life rather than relying on retrospective reports that may focus on more memorable, recent, or unresolved events, for example, [47]. Other research using EMA to examine experiences of sexist experiences also reported a relatively high frequency of discrimination [48]. The frequency of negative interpersonal interactions in daily life is likely important, as consequences may accrue to predict risk. For example, the frequency of negative interactions has been found to mediate the effects of perceived discrimination on health outcomes [49].

A second goal of the present research was to test the hypothesis that emotional responses help explain how negative interpersonal interactions are related to ABP, both at a given moment in a day and later in the same day. Although past work suggests that negative interpersonal interactions can function as a stressor [13], causing emotional and cardiovascular reactivity [14], to our knowledge no past research has examined changes in negative mood as a mediator of negative interpersonal interactions on ABP in daily life. In our concurrent mediation analyses, negative mood mediated the relationship between negative interpersonal interactions and diastolic (but not systolic) ABP. In the lagged mediation analyses, although there were no direct associations between negative interpersonal interactions and ABP assessed later in the day (i.e., up to 40 min later), there was an indirect relationship through negative mood assessed up to 20 min after the negative interpersonal interaction on systolic (but not diastolic) BP. This pattern of results indicates that the effects of negative interpersonal interactions on BP are sustained due to the lack of affective recovery following the interaction, and suggests complex patterns of emotion regulation that evolve over time, influencing the relations of social interactions, mood, and ABP. As such, there may be other possible mechanisms that buffer BP reactivity and/or aid in BP recovery, including positive mood, that have demonstrated a protective role for cardiovascular health [50, 51].

Lastly, we tested whether participants' race/ethnicity or reported lifetime discrimination moderated associations but did not find any significant moderation effects. Given prior work suggesting that past discrimination may sensitize people to experiences of mistreatment and predict greater reactivity [22, 23], we hypothesized that individuals who reported greater lifetime discrimination in the current study might show greater reactivity to negative interpersonal interactions. In prior research, researchers were able to connect attributions for the negative interpersonal mistreatment to one's race or ethnicity. In everyday life, it is possible that higher lifetime exposure to discrimination makes one more likely to anticipate or expect to be treated in a discriminatory manner [52], but not necessarily to have greater physiological reactivity to all negative interactions. That said, we found that higher levels of lifetime discrimination predicted higher levels of negative interpersonal mistreatment. This suggests that, contrary to some extant work [53], the experience of discrimination in the past does not desensitize an individual to the ongoing experience of discrimination (and perhaps may even sensitize one to noticing negative experiences [8], or that ongoing discriminatory experiences are occurring). Future research is warranted to examine nuances in the extent to which lifetime discrimination influences affective and cardiovascular responses to negative interpersonal interactions, including testing the potential for an indirect effect of lifetime discrimination through everyday interactions.

Overall, the present findings likely have clinical implications given the strong link between ABP and health risk [26, 27]. Our analyses highlight the need to better understand the impact of negative interpersonal interactions on both mood and ABP and to examine in-the-moment coping mechanisms. To this end, an important next step would be to uncover additional modifiable psychological, cognitive, or behavioral mechanisms that lead to sustained negative effects of interpersonal maltreatment. For example, experiencing mistreatment may evoke negative mood and trigger ruminative or vigilant states that maintain negative mood and make a future negative interpersonal interaction more likely [54]. Another complementary possibility is that individuals experiencing negative interpersonal interactions engage in social withdrawal, preventing additional conflict but limiting the ability to acquire skills in emotion regulation. Moreover, coping strategies that engage more social support may improve cardiovascular outcomes [55]. Social and behavioral strategies at a dyadic or societal level are needed to prevent the occurrence of, or minimize the effects of, negative interpersonal interactions that have downstream effects on cardiovascular risk.

Limitations and Future Directions

As is common for this type of EMA research, ABP in this study was assessed frequently (every 20 min) with a self-report at each assessment. This approach allowed for a tight window between any reported negative interpersonal interaction and the subsequent assessment of mood and ABP. However, this frequency of assessment could have changed the way people interacted with others. Due to the frequency of assessments, the EMA measures were brief to reduce participant burden, including the three-item negative mood scale, which may have been less reliable as a measure than a scale with additional items [56]. Our models had relatively small effect sizes in predicting ABP, although negative interpersonal interactions had independent effects similar to a range of known and strong influences on BP. This suggests that additional variables are needed to further consider when and for whom negative interpersonal interactions relate to cardiovascular outcomes. For example, researchers could capture the extent to which a person attributed the reason for their unfair treatment to a social identity, including their race and ethnicity. This momentary measure of attributions would allow for the comparison of whether discriminatory experiences have more negative psychobiological consequences than other types of negative interpersonal interactions. Future research could also measure and examine a wider range of contextual factors that may influence the cardiovascular consequences of negative interactions, including the social context in which the interactions occur [57], and individual differences factors, including personality, which may interact to affect responses to interpersonal interactions [58]. To this end, although we had a diverse sample of participants who identified as Black or Hispanic, they all came from a large Northeastern city in the USA. Researchers should use caution when generalizing these findings to other samples.

Conclusion

These results extend a growing literature on the deleterious effects of negative interpersonal interactions. In keeping with the Science of Behavior Change approach of identifying mechanisms of action [59], negative mood was examined as potential psychological mechanisms contributing to health outcomes. Higher levels of negative mood during these negative interpersonal interactions appeared to explain associations between interactions and ABP, both concurrently and over time. Although there is more work to be done to further uncover the nature of these relationships, these findings point to possible intervention targets, including reducing negative interactions and enhancing stress regulation.

Compliance with Ethical Standards

Conflicts of Interest No authors have conflicts of interest to declare.

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