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Discrimination and anger control as pathways linking socioeconomic disadvantage to allostatic load in midlife

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ABSTRACT

Objective: Recent evidence suggests that experiences of discrimination contribute to socioeconomic status health disparities. The current study examined if the experience and regulation of anger—an expected emotional response to discrimination—serves as an explanatory factor for the previously documented links between socioeconomic disadvantage (SED), discrimination, and allostatic load.

Methods: Data were drawn from the second wave of the Midlife in the United States (MIDUS) study and included 909 adults who participated in the biomarkers subproject.

Results: Results revealed that perceived discrimination was associated with higher levels of allostatic load. Furthermore, we found evidence that perceived discrimination and anger control sequentially explained the relationship between SED and allostatic load, such that greater discrimination was associated with lower levels of anger control, which, in turn accounted for the effects of discrimination on allostatic load. These results remained significant after controlling for negative affect, positive affect, other forms of anger expression, as well as demographic covariates.

Conclusions: Our findings suggest that low anger control may be an important psychological pathway through which experiences of discrimination influence health.

Our experiences as members of particular social groups can shape many aspects of our health and well-being. These effects can be particularly detrimental if the groups to which we belong to are marginalized or otherwise disparaged by the larger society. For example, being part of socioeconomically disadvantaged groups can compromise both mental and physical health, contributing to greater depression and anxiety [1,2], increased risk for chronic diseases [3,4], and even greater risk for mortality [5]. Because members of socioeconomically disadvantaged groups are targets of many negative stereotypes, recent evidence suggests that the experience of discrimination also contributes to socioeconomic status health disparities [6]. The current study expands on this perspective by examining the experience and regulation of anger—an expected emotional response to discrimination—as an explanatory factor for the previously documented links between socioeconomic disadvantage (SED), discrimination, and biological indicators of health. Our analyses focus on allostatic load, a biological index that summarizes dysregulation across several physiological systems [7], because of its established relationship with many clinical endpoints (e.g., mortality), as well as both SED and discrimination [6,8].

1. SED, discrimination, and allostatic load

Discrimination refers to the negative treatment of an individual based on the social group(s) of which she or he is a member. A person can be discriminated based on his/her membership in multiple social groups (e.g., sexual orientation, age, religion, social class, race, ethnicity). Further, experiences of discrimination can be major discrete life events (e.g., being fired because of one's ethnicity) or daily chronic hassles (e.g., being verbally harassed because of one's social status). For these reasons, discrimination is a multidimensional construct, similar to social status.

For many members of disadvantaged groups, experiences of

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discrimination constitute a source of chronic stress, with detrimental consequences for physiological functioning, such as elevated blood pressure or increased levels of inflammation [9]. Although the discrimination-health link has been studied primarily in ethnic minorities, experiences of discrimination also extend to members of other underprivileged groups, such as those from low socioeconomic backgrounds [10–14]. Numerous studies show that individuals from low social status groups are often stereotyped as lazy or incompetent [13], beliefs that are particularly salient in countries that endorse meritocracy. For example, in a qualitative study conducted in two Canadian cities, Reutter and colleagues found that low-income individuals reported being perceived as lazy, irresponsible, and a burden to society [12]. Interestingly, other studies have shown that the threat of these attributions remains with low status individuals even as they try to integrate into more privileged environments [15,16]. Further, although it is recognized that both societal and individualistic factors cause poverty, discrimination is linked to considering the latter to be more important than the former [10,17]. These stereotypes and prejudices against low social status individuals foster distancing and discrimination toward this social group from other members of the society [11].

In addition to this social psychological perspective, the link between SED, discrimination, and health can also be understood in terms of the theory of fundamental causes of health inequalities [18,19]. According to this theory, socioeconomic status inequalities in health can be attributed to differential access to individual and contextual key resources (i.e., knowledge, money, power, prestige, and social support). These resources shape individual experiences, such as perceived discrimination, which in turn act as more proximal risks and causes of health outcomes.

Recent research has provided support for these theoretical accounts by showing that perceptions of discrimination among low-SES individuals [6,14,20,21] can lead to negative emotional responses (e.g., anger) [13] and risky behaviors (e.g., substance abuse) associated with poor health [22]. For example, Fuller-Rowell and colleagues [6] found that perceived discrimination partially mediated the association between poverty and allostatic load in a sample of predominantly White rural youth. Their findings are noteworthy for at least two reasons: they are among the first to demonstrate a link between low socioeconomic status and detrimental biological responses as a result of perceived unfair treatment, and they focus on allostatic load, an important measure of cumulative biological risk that foreshadows the onset of many chronic diseases [7].

Allostatic load refers to the physiological burden experienced by the body as a result of the chronic or repeated activation of the cardiovascular, autonomic, neuroendocrine, immune, and metabolic systems [7]. It is hypothesized that chronic stressors can cause dysregulation of interrelated physiological systems, which if prolonged, may ultimately lead to greater risk of chronic disease, cognitive decline, and mortality [23,24].

Although many studies have investigated the relationship between reported experiences of unfair treatment and health (for a review, see [25–27]), few studies have related discrimination to multisystem functioning. Rather, most studies have focused on individual physiological indicators or preclinical endpoints of poor health. For example, several studies have found that unfair treatment and discrimination are associated with elevated nocturnal blood pressure [28,29], excess adiposity [30], coronary artery calcification [31,32], and inflammation [33]. Given that the effects of chronic stress are typically nonspecific [34], single system studies do not adequately capture the cumulative impact of discrimination. In comparison, a multi-systems approach is consistent with evidence that many people, particularly older adults, suffer from multiple, co-occurring chronic conditions, which contribute to increased risks for morbidity and mortality [35]. Interestingly, analyses from the MacArthur Studies of Successful Aging have shown that, although the overall summary measure of allostatic load predicts risk for major health outcomes, none of the individual components of allostatic load is a significant independent risk factor [36,37].

Research has shown that socioeconomic disadvantage predicts allostatic load in different cultures [38] and among different age groups [39,40]. Direct evidence also supports the association between discrimination and allostatic load [6], including studies showing that experiences of discrimination predict health conditions characterized by increased allostatic load, such as diabetes and cardiovascular disease [9]. Moreover, greater perceptions of unfair treatment are associated with coronary artery calcification among African American women [32] as well as coronary events and metabolic syndrome among civil employees [41]. Yet, only a few studies have tested whether daily discrimination mediates the relationship between SED and allostatic load in middle aged and older adults [21,42]. Midlife may be an important point in the lifespan to examine these processes, because it ushers in a period of rapidly rising risk for acute and chronic illness. Further, to our knowledge, no studies have tested the more proximal underlying affective mechanisms through which chronic discrimination might lead to elevated allostatic load. In this study we try to address these gaps by focusing on anger, an affective response commonly associated with detrimental health outcomes [43,44].

2. The mediating role of anger control

Anger is an approach-oriented emotion that typically stems from experiences of violation, injustice, or obstacles to desired goals [45]. As such, it is not surprising that anger shares a strong association with both SED and perceived discrimination given that, in both situations, individuals face unjust challenges related to their social status, race, or ethnicity [22]. For example, those who have lower education or face economic hardship report more frequent experiences of anger and are more likely to show poor anger control (i.e., the ability to restrain arousal and calm down; [46,47]). Similarly, those who are exposed to discrimination, either as targets or bystanders, respond with greater anger to and take longer to recover physiologically from discriminatory experiences compared to those who do not encounter such stressors [48,49]. Notably, both experimental and field studies indicate that anger is the most common affective reaction to discrimination, regardless of its underlying cause (i.e. racial vs. non-racial) or the race of the target [50,51].

These converging links between SED, discrimination, and anger are particularly compelling in light of complementary evidence showing that experiences of anger are associated with several health conditions and their underlying biological mechanisms. For example, high levels of trait anger, as well as certain aspects of anger expression, such as the tendency to express anger outwardly (anger out) or the tendency to suppress anger expression (anger in), have been associated with adverse cardiovascular outcomes including greater risk of hypertension and cardiovascular disease morbidity and mortality over time [43,52,53]. Greater anger control, on the other hand, is considered to be beneficial for health given that it allows individuals to restrain arousal while engaging in activities that help to dissipate the experience of negative affect [54]. Indeed, research has shown that anger control is inversely related to pro-inflammatory and coagulation markers such as interleukin-6 (IL-6) and fibrinogen [47], but positively associated to adaptive immune processes (i.e., faster wound healing) and lower cortisol reactivity to a physical stressor [55]. Furthermore, anger control is prospectively associated with lower risk of cardiovascular disease incidence, above and beyond the influences of anger in and anger out, suggesting that anger control may be a stronger predictor of health outcomes than other forms of anger expression [53]. The role of anger control as a unique predictor for allostatic load, however, remains to be clarified.

Although there is no direct evidence demonstrating that exposure to discrimination mediates the link between SED and biological responses through its effect on anger control, results from several separate but related lines of work suggest that this sequence is plausible. Broadly

speaking, experiences of discrimination have been related to self-regulatory deficits, which may also extend to the ability to control negative emotional experiences such as anger [15,56]. Laboratory studies show that, in comparison to rejection by in-group members, being discriminated/rejected by out-group members is followed by greater nonverbal expressions of anger and slower physiological recovery from detrimental cardiovascular responses, which suggests that discrimination may undermine anger control [49,51]. In correlational research, trait anger and unfair treatment significantly predicted blood pressure outcomes among African American adolescents living in poor neighborhoods [28]. Furthermore, longitudinal studies in another sample of African American youth have shown that greater discrimination predicted increased anger experiences after 22 months, which in turn, were related to greater substance abuse, suggesting that cumulative experiences of discrimination may diminish the ability to control anger in the long run [22].

Together, these findings provide a strong premise for the hypothesis that anger control serves as one of the psychosocial mechanisms through which experiences of discrimination link socioeconomic disadvantage to biological indicators of health such as allostatic load. In the current study we tested this hypothesis in a large sample of U.S. adults from the Midlife in the United States (MIDUS) project. First, we sought to replicate the previously identified link between SED, discrimination, and allostatic load, extending these findings to an older population. Next, we tested the hypothesis that anger control is a psychological mechanism underlying the effect of discrimination on allostatic load. Given that previous research has suggested that anger control may constitute a stronger predictor for health-related outcomes relative to other forms of anger expression, we were also interested in investigating whether anger control would remain a significant predictor of allostatic load even after accounting for the influence of anger in and anger out. We restricted our analyses to White individuals because of the low number of non-white participants in the sample and to reduce confounding of discrimination between SED and ethnicity or race.

3. Method

3.1. Participants

Participants were part of the Midlife in the United States (MIDUS) survey, a national longitudinal study focused on understanding factors that contribute to healthy aging. The first wave of data collection took place from 1995 to 1996 and targeted non-institutionalized adults between the ages of 20 and 75. Consenting participants completed a phone interview and a self-administered questionnaire at home. The same assessments took place again during 2004–2006, as part of MIDUS II. The current study analyzed data from a subset of MIDUS II respondents (N = 1054) who provided biological samples during an overnight visit at one of three regional medical centers (for details on sampling procedures see Radler & Ryff, 2010). Clinical staff obtained participants' complete medical history, conducted a physical examination, and collected cardiovascular and heart rate variability measurements along with blood, urine, and saliva samples. Fasting blood was collected at 07:00 h, and urine was collected between 19:00 h and 7:00 h. As mentioned in the Introduction, because of the low number of non-white participants in this sample (n = 86) and the confounding between ethnicity/race and perceived discrimination, only White participants were retained for analyses. Of these, only individuals with complete data on all study variables were selected, reducing the final sample to 909 individuals (M age = 55.37; SD = 11.85, 54.13% female). Psychological scales were created following instructions from the MIDUS authors, and associated Cronbach's α-s are the ones obtained from the overall sample. Descriptive statistics are reported in Table 1. Data collection for all phases of the MIDUS project was approved by institutional review boards at each participating site, and all participants provided informed consent.

Table 1
Descriptive statistics.

Descriptive variables	%	Mean or median	SD
Female	54.13%	–	–
Age (years)	–	55.37	11.85
Any chronic condition	78.11%	–	–
Current financial situation			
Worst possible	27.39%	–	–
Average	36.19%	–	–
Best possible	36.41%	–	–
Money for basic needs			
Not enough	15.73%	–	–
Just enough	51.93%	–	–
More than enough	32.34%	–	–
Difficulty paying bills			
Very/somewhat difficult	23.87%	–	–
Not very difficult	36.74%	–	–
Not at all difficult	39.39%	–	–
Education			
High school/GED or less	23.87%	–	–
Some college/associate degree	28.05%	–	–
Bachelor's degree or higher	48.07%	–	–
Family-size adjusted income to poverty ratio			
< 300%	26.95%	–	–
Between 300% and 599%	37.18%	–	–
> 600%	35.86%	–	–
Negative affect	–	1.48	0.53
Positive affect	–	3.44	0.69
Trait anger	–	23.78	5.24
Anger in	–	14.60	4.05
Anger out	–	12.76	3.18
Anger control	–	10.15	2.16
Perceived discrimination	–	12.51	4.14

Note: GED = General Educational Development.

3.2. Measures

3.2.1. Socioeconomic disadvantage (SED)

Following Gruenewald et al. [40], we created this variable by summing values from five indicators: education level (2 = high school/GED or less, 1 = some college/associate arts degree, 0 = bachelor's degree or higher), family-size adjusted income to poverty ratio (2 = < 300%, 1 = 300%/599%, 0 = 600% or more), current financial situation (2 = rating from 0-worst possible to 5, 1 = rating from 6 to 7, 0 = rating from 8 to 10-best possible), availability of money to meet basic needs (2 = not enough, 1 = just enough, 0 = more than enough), and difficulty of paying bills (2 = very or somewhat difficult, 1 = not very difficult, 0 = not at all difficult). SED was computed for all cases that had at least four indicators with valid values. Scores ranged from 0 to 10 (M = 4.26, SD = 2.65).

3.2.2. Perceived discrimination

Everyday experiences of discrimination were assessed with nine items designed to capture perceptions of unfair treatment [57]. Using a 4-point scale (1 = often, 4 = never), participants rated how often they believed they were the target of discriminatory acts in daily life because of their background (e.g., gender, age, or other characteristics). Example items are “You are treated with less courtesy than other people”, “You receive poorer service than other people at restaurant or stores”, and “People act as if they think you are not as good as they are” (α = 0.92). For cases with at least five valid items, one total score was calculated by summing the values of the items. Mean imputation was used for items with a missing value. Higher scores indicated higher perceived discrimination. Scores ranged from 9 to 30. After completion of the nine items, participants were asked to report the main reason/s (i.e., age, gender, race, ethnicity, religion, weight/height, other aspect of physical appearance, physical disability, sexual orientation, other

reasons) for their discrimination experiences. The three most common reasons (above 10%) were gender (19.80%), age (15.73%), and other reason (10.56%). In our analyses, age and gender were treated as covariates in all models.

3.2.3. Anger

Anger facets were assessed with the State-Trait Anger Expression Inventory [58]. Participants provided responses on a 4-point scale (1 = almost never, 4 = almost always) with higher scores indicating higher standing on each construct. For each scale a total score was calculated by summing the ratings of the items for cases that had no or only one missing value. Mean imputation was used in cases with one missing value. Anger control was assessed with four items of the anger control subscale (i.e., "I keep my cool", "I calm down faster", "I control my temper", and "I make threats"-reversed). Scores ranged from 4 to 14 ($\alpha = 0.69$). Trait anger was assessed by 15 items (e.g., "I have a fiery temper"). Scores ranged from 15 to 47 ($\alpha = 0.84$). Anger in was assessed with eight items (e.g., "I am angrier than I'm willing to admit" and "I am irritated more than others are aware"). Scores ranged from 8 to 31 ($\alpha = 0.82$). Lastly, anger out was assessed by eight items (e.g., "If someone annoys me I tell them how I feel"). Scores ranged from 8 to 28 ($\alpha = 0.77$).

3.2.4. Allostatic load

We used an allostatic load index used in previous work on this sample [40]. The index was calculated by averaging the number of physiological indicators for which participants were categorized into the highest quartile of risk. Indicators from the following seven systems were selected for the current analyses: cardiovascular, metabolic-lipids, glucose metabolism, inflammation, sympathetic nervous system, parasympathetic nervous system, and hypothalamic-pituitary-adrenal axis. First, risk scores were defined as the upper or lower quartile depending on whether high or low values of the biomarker typically confer greater risk for poor health outcomes. System risk indices were computed for individuals with values on at least half of the system biomarkers and were expressed as the percentage (0–1) of system biomarkers in high-risk range. Allostatic load scores were calculated for participants with data on at least six of the seven systems (possible range: 0–7, observed range: 0–4.8). Table 2 presents descriptive statistics for each allostatic load component.

3.2.5. Psychological covariates and alternative psychological mediators

Increased negative affect and reduced positive affect have been suggested as two broad pathways linking SED to poor health [59]; thus, we controlled for these two emotional dimensions. For positive and negative affect [60] participants were asked to rate six adjectives on a 5-point scale (1 = all of the time, 5 = none of the time) to indicate to what extent they felt a specific positive (e.g., cheerful, satisfied) and negative (e.g., hopeless, nervous) emotional state during the last 30 days. Each scale was computed by calculating the mean of the item responses for cases that had valid values for at least one item. Scales were recoded such that higher scores indicate higher positive ($\alpha = 0.90$) or negative ($\alpha = 0.85$) affect.

3.2.6. Physical health

During MIDUS II, participants reported whether they had any chronic condition in the previous 12 months (0 = no, 1 = yes). This variable was used as an index of participants' physical health.

3.3. Statistical analyses

Multiple regression analyses were conducted to test whether SED separately predicted perceived discrimination and allostatic load, and whether perceived discrimination was associated with allostatic load while controlling for SED. Next, we used PROCESS [61] for SPSS to conduct our mediation analyses. Path analyses (e.g., single and serial mediation in the present study) can be conducted with PROCESS, which is based on ordinary least squares analyses. PROCESS was used to perform bootstrap analyses (20,000 repetitions) to derive a 95% confidence interval (CI) for the indirect effect linking SED to allostatic load via perceived discrimination. CIs not including 0 indicate statistically significant indirect effects. These analyses were conducted first controlling only for demographics (age, gender; Model 1), then controlling for demographics and physical health (Model 2) and then controlling for demographics, physical health, and psychological covariates (anger in, anger out, trait anger, positive affect, and negative affect; Model 3). To facilitate interpretation, all continuous variables were standardized, and dichotomous variables were coded as 0 and 1 (i.e., 0 = male, 1 = female).

In order to test the hypothesis that low anger control predicts allostatic load and that perceived discrimination and low anger control explain the link between SED and allostatic load, we ran two-step mediation analyses (for a graphical representation, see Fig. 1). Using PROCESS, we tested three indirect pathways: 1) one from SED to allostatic load via perceived discrimination (a1a2); 2) one from SED to allostatic load via anger control (b1b2 in Fig. 1); and, 3) one from SED to allostatic load via both perceived discrimination (first) and anger control (second) (a1a3b2 in Fig. 1). Bootstrapping (20,000 repetitions) was used to derive 95% CI for all indirect effects. As above, these analyses were conducted controlling only for demographics (age, gender; Model 1), then controlling for demographics and physical health (Model 2) and then controlling for demographics, physical health, and psychological covariates (anger in, anger out, trait anger, positive affect, and negative affect; Model 3). Lastly, because anger in,

Table 2
Descriptive statistics and high-risk cutpoints for biomarkers used to compute total allostatic load.

Descriptive variables	N	M	SD	High-risk cutpoint (≥)
Cardiovascular				
Resting SBP (mmHg)	909	130.86	17.49	143.00
Resting DBP (mmHg)	909	74.90	10.31	82.00
Resting hear rate (bpm)	908	70.63	11.06	77.00
Metabolic – lipids				
BMI	909	29.03	5.84	32.31
WHR	908	0.89	0.10	0.97
Triglycerides (mg/dL)	907	130.83	80.15	160.00
HDL cholesterol (mg/dL)	907	54.60	17.67	41.37
LDL cholesterol (mg/mL)	907	106.33	34.72	128.00
Metabolic – glucose metabolism				
Glycosylated hemoglobin (HbA1c)	905	5.95	0.83	6.10
Fasting glucose (mg/dL)	904	99.49	20.48	105.00
Insulin resistance (HOMA-IR)	903	3.17	2.92	4.05
Inflammation				
IL-6 (pg/ml)	909	2.75	2.69	3.18
CRP (mg/L)	904	2.66	4.01	3.18
Fibrinogen (mg/dL)	905	338.57	82.94	390.00
sE-selectin (ng/Mi)	909	41.02	20.56	50.58
sICAM-1 (ng/MI)	909	289.31	99.56	329.65
Sympathetic nervous system				
Urine epinephrine (μg/g creatine)	894	2.03	1.26	2.54
Urine norepinephrine (μg/g creatine)	898	27.54	13.00	33.33
Hypothalamic pituitary adrenal axis				
Urine cortisol (μg/g creatine)	906	16.62	16.78	21.00
Blood DHEA-s (μg/dL)	905	105.61	76.32	51.00
Parasympathetic nervous system				
SDRR (ms)	836	35.23	17.25	23.54
RMSSD	836	21.43	16.30	11.83
Low frequency spectral power	836	432.32	652.91	113.96
High frequency spectral power	836	270.44	686.96	54.16
Allostatic load	909	1.70	1.02	

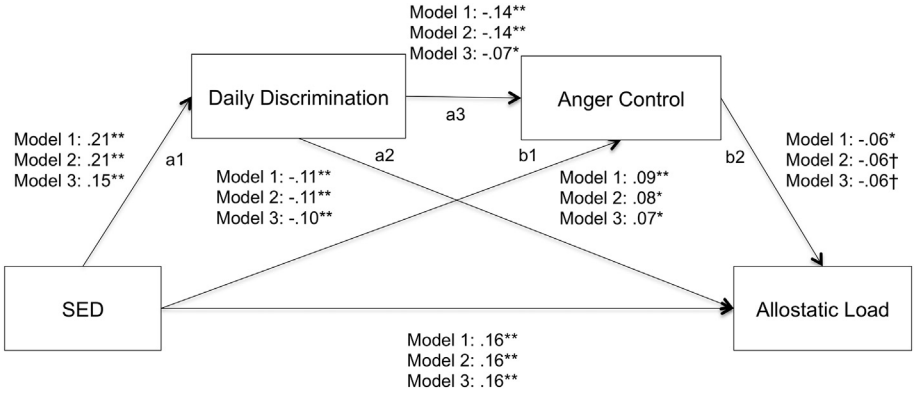

Fig. 1. Multiple indirect effect models linking socioeconomic disadvantage to allostatic load via perceived discrimination and anger control. †$p < 0.10$, *$p < 0.05$, **$p < 0.01$.

anger out, and affect could also act as pathways of the perceived discrimination/allostatic load link, we tested indirect effects linking perceived discrimination to allostatic load via anger in, anger out, and affect.

4. Results

4.1. SED, perceived discrimination, and allostatic load

Bivariate correlations among study variables are presented in Table 3, while bivariate correlations between the SED, perceived discrimination, anger control, and each allostatic load component are reported in Table 4. Regression analyses controlling for demographics revealed a significant effect of SED on perceived discrimination [β = 0.215, 95% CI: 0.1515: 0.2780, $p < 0.001$, Model 1; β = 0.211, 95% CI: 0.1480: 0.2738, $p < 0.001$, Model 2; β = 0.146, 95% CI: 0.0823: 0.2089, $p < 0.001$, Model 3]. As expected, greater SED was associated with greater discrimination. Further, greater perceived discrimination predicted higher levels of allostatic load [β = 0.090, 95% CI: 0.0277: 0.1519, $p = 0.005$, Model 1; β = 0.076, 95% CI: 0.0136: 0.1376, $p = 0.017$, Model 2; β = 0.071, 95% CI: 0.0067: 0.1357, $p = 0.031$, Model 3]. Bootstrapping analyses revealed a significant indirect effect of SED on allostatic load via perceived discrimination [a1a2 = 0.019, 95% CI: 0.0056: 0.0379, Model 1; a1a2 = 0.016, 95% CI: 0.0027: 0.0335, Model 2; a1a2 = 0.010, 95% CI: 0.0013: 0.0244, Model 3].

4.2. SED, perceived discrimination, anger control, and allostatic load

Regression analyses controlling for demographics revealed a significant effect of anger control on allostatic load [β = −0.062, 95% CI: −0.1235: −0.0015, $p = 0.045$, Model 1], such that low levels of anger control were associated with higher levels of allostatic load. The effect of anger control on allostatic load was of similar magnitude, but failed to reach statistical significance after controlling for demographics and physical health [β = −0.059, 95% CI: −0.1199: 0.0012, $p = 0.055$, Model 2] and after controlling for demographics, physical health, and psychological covariates [β = −0.060, 95% CI: −0.1249: 0.0041, $p = 0.066$, Model 3] (Fig. 1). Further, we found evidence for the hypothesis that perceived discrimination and anger control sequentially explained the relationship between SED and allostatic load (i.e., SED ➔ perceived discrimination ➔ anger control ➔ allostatic load) [a1a3b2 = 0.0019, 95% CI: 0.0003: 0.0048, Model 1; a1a3b2 = 0.0017, 95% CI: 0.0002: 0.0045, Model 2; a1a3b2 = 0.0007, 95% CI: 0.00004: 0.0024, Model 3]. Interestingly, anger control alone (i.e., SED ➔ anger control ➔ allostatic load) also explained the effect of SED on allostatic load [b1b2 = 0.0069, 95% CI: 0.0008: 0.0183, Model 1; b1b2 = 0.0065, 95% CI: 0.0006: 0.0171, Model 2; b1b2 = 0.0061, 95% CI: 0.0003: 0.0166, Model 3].

4.3. Testing alternative indirect effects models

To test whether the indirect effect linking SED and perceived discrimination to allostatic load was specific to anger control, we ran the same two-step indirect effect model (i.e., SED ➔ perceived discrimination ➔ psychological pathway ➔ allostatic load) using four alternative psychological constructs: anger in, anger out, positive affect, and negative affect. These models were tested controlling for demographics and psychological covariates, including anger control. No evidence for a significant indirect effect was found for any of these alternative psychological processes. Specifically, the estimated indirect effect for anger in was −0.0003 (95% CI: −0.0018: 0.0001), the

Table 3
Bivariate correlations among study variables.

Descriptive variables	1	2	3	4	5	6	7	8	9	10	11	12
1. Female	1	−0.052	0.127**	0.063†	−0.003	0.027	−0.066*	−0.007	−0.071*	0.063†	0.111**	−0.017
2. Age		1	0.094**	−0.189**	0.196**	−0.096**	−0.248**	−0.220**	0.054	−0.164**	−0.068*	0.349**
3. Chronic condition			1	0.212**	−0.166**	0.171**	0.067*	0.052	−0.049	0.109**	0.041	0.164**
3. Negative affect				1	−0.632**	0.356**	0.337**	0.181**	−0.155**	0.317**	0.267**	0.020
4. Positive affect					1	−0.332**	−0.365**	−0.167**	0.180**	−0.227**	−0.215**	0.001
5. Trait anger						1	0.495**	0.524**	−0.296**	0.209**	0.096**	0.039
6. Anger in							1	0.191**	−0.147**	0.188**	0.091**	−0.080*
7. Anger out								1	−0.321**	0.181**	0.026	−0.041
8. Anger control									1	−0.173**	−0.149**	−0.078*
9. Perceived discrimination										1	0.228**	0.065*
10. SED											1	0.158**
11. Allostatic load												1

Note: SED = socioeconomic disadvantage.
† $p < 0.10$.
* $p < 0.05$.
** $p < 0.01$.

Table 4
Bivariate correlations between the SED, perceived discrimination, anger control, and each allostatic load component.

Descriptive variables	1	2	3	4	5	6	7	8	9	10	11
1. Cardiovascular	1	0.229**	0.124**	0.133**	0.140**	0.014	0.171**	0.498**	0.051	− 0.012	− 0.020
2. Metabolic – lipids		1	0.350**	0.244**	− 0.071*	− 0.137**	0.090**	0.443**	0.117**	0.124**	0.023
3. Metabolic – glucose metabolism			1	0.243**	0.071*	− 0.004	0.171**	0.571**	0.119**	0.040	− 0.057†
4. Inflammation				1	0.115**	− 0.006	0.189**	0.510**	0.210**	0.121**	− 0.063†
5. Sympathesic nervous system					1	0.131**	0.114**	0.482**	0.028	− 0.004	− 0.024
6. Hypothalamic pituitary adrean axis						1	0.052	0.325**	0.060†	− 0.020	− 0.030
7. Parasympathetic nervous system							1	0.582**	0.002	0.001	− 0.082*
8. Allostatic load								1	0.158**	0.065*	− 0.078*
9. SED									1	0.228**	− 0.149**
10. Perceived discrimination										1	− 0.173**
11. Anger control											1

Note: SED = socioeconomic disadvantage.
† $p < 0.10$.
* $p < 0.05$.
** $p < 0.01$.

estimated indirect effect for anger out was − 0.0002 (95% CI: − 0.0013: 0.0004), the estimated indirect effect for negative affect was 0.00003 (95% CI: − 0.0023: 0.0021), the estimated indirect effect for positive affect was 0.000004 (95% CI: − 0.0004: 0.0005). These results support the hypothesis that anger control might serve as a unique mediator of the link between SED, perceived discrimination, and allostatic load.

5. Discussion

In the current study, we extended previous research on the link between socioeconomic disadvantage, perceived discrimination, and allostatic load in two important aspects. First, whereas Fuller-Rowell and colleagues [6] showed that perceived discrimination explained a significant portion of the effect of SED on allostatic load in a sample of predominately White rural youth, we found that this association is also present among White adults in their midlife, a time when chronic diseases become more prevalent. Second, and most importantly, our findings revealed that low levels of anger control, but not trait anger or other forms of anger expression or affect, might be one of the underlying psychological mechanisms accounting for this association.

These findings contribute to several lines of research focused on understanding the mechanisms through which membership in socially and economically disadvantaged groups increases vulnerability to negative health outcomes. First, many theoretical accounts argue that emotional reactions to the challenges presented by socioeconomic disadvantage may serve as proximal mechanisms through which SED influences health outcomes [1,62]. Our findings provide further empirical support for this argument by showing that confrontation with discrimination may impact the ability of socioeconomically disadvantaged individuals to control negative emotions such as anger. Furthermore, our findings suggest that lower anger control contributes to the link between discrimination and allostatic load, providing additional support for the idea that deficits in emotional regulatory capacity increase vulnerability to the dysregulation of several physiological parameters in midlife [1].

Second, several lines of research suggest that unlike other stressors, experiences of discrimination give rise to a cascade of highly arousing and approach-oriented cognitive and affective experiences which may be detrimental for health [49]. In line with these arguments, we also found that perceptions of discrimination were negatively associated with anger control, even after accounting for constructs that capture other forms of unpleasant affective experiences such as negative affect. Our findings, therefore, add evidence to the perspective that discrimination is associated with a specific profile of emotions, such as anger, which may contribute to detrimental health-outcomes above and beyond the influence of other affective experiences. Our results also support the view that discrimination has particularly detrimental effects on emotion regulation [22], which, in turn, can impact health directly through physiological pathways, or indirectly through behavioral pathways (i.e. increased drinking or substance abuse in an attempt to regulate arousing emotions).

Finally, our findings contribute to the literature on the influence of anger on health-related biological outcomes. Previous research has shown that not all aspects of anger are consistently related to health-relevant outcomes [43], and that these associations may differ depending on the demographic characteristics of the sample [63] as well as the outcomes in question. Furthermore, very few studies have assessed the influence of various forms of anger simultaneously to determine which aspect may contribute more strongly to health-related outcomes. Our investigation not only adds to the small number of studies following this approach, but also supports previous findings that show that anger control is associated to health-relevant outcomes above and beyond the influence of trait anger or other forms of anger expression (i.e., anger in and anger out; [44,64]).

Despite the novel contributions of our study, some features of our methods and analyses necessarily limit conclusions. First, although we conceptualized unfair treatment as a risk factor for increased allostatic load, in the absence of longitudinal data, it is possible that a reverse association exists, in which high levels of allostatic load or associated morbidities contribute to socioeconomic disadvantage, as well as increase reports of discrimination and diminished anger control. Thus, prospective studies with multiple-wave assessments of these constructs are needed to understand the directionality and time course of these relationships. Longitudinal designs may reveal, for example, whether repeated exposure to everyday mistreatment accumulates over time to influence subsequent allostatic load, in addition to the mechanisms underlying these effects. Second, our measure of daily discrimination did not explicitly include income or education as options participants could select when reporting the main reason for their discrimination experiences. Thus, a limitation that the present work shares with previous studies on the link between SED, discrimination, and biological risk [6,21] is that social status-related perceived discrimination was not directly assessed. A third caveat is that our findings pertain to White middle-aged adults. By restricting our analyses to White individuals, we tried to minimize the impact of discrimination experiences due to race and ethnicity in our sample. Although this approach could be seen as strength of our study, it also limits the generalizability of our findings to the broader U.S. population, including non-White individuals, who are likely to experience discrimination more often than White individuals. Thus, whether anger control is a proximate mechanism linking discrimination and cumulative biological risk among non-White individuals needs to be tested in future studies. Fourth, in the current study we employed a composite measure of SED, similarly to what was

previously done in this sample [40]. On the one hand, this approach allows for a more integrated view of socioeconomic status health disparities, but it does not illuminate the specific contribution of each SED dimension to the health disparities observed here. Fifth, our measures of discrimination were based on self-report and did not include comprehensive assessments of structural or institutional discrimination (e.g., residential segregation, socio-economic mobility), and research in these areas is warranted [9].

Despite the study limitations, the findings shed light on the mechanisms and biological underpinnings of socioeconomic disadvantage in midlife. To our knowledge, the present analysis is among the first to consider the cumulative effects of unfair treatment across a comprehensive measure of biological risk (i.e., allostatic load) within a large community-based sample of middle-aged adults. Additionally, our study also adds to the accumulating evidence showing that low anger control may be an important psychological pathway through which the challenges of discrimination exert a negative impact on health, contributing new insights to our understanding of the factors that influence emotional regulation and their role in health and well-being.

Conflicts of interest

The authors confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome. We further confirm that any aspect of the work covered in this manuscript that has involved human subjects has been conducted with the ethical approval of all relevant bodies.

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