

Curvilinear associations of discrimination attributions with inflammatory and antiviral gene expression

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ABSTRACT

Background: Discrimination is a social determinant of health associated with adverse health outcomes through biological mechanisms such as the Conserved Transcriptional Response to Adversity (CTRA). The biological impacts of discrimination are well-documented, but the role of attributions (i.e., individuals' beliefs about why they experienced discrimination) remains unexplored. This study examined the associations of discrimination attributions with inflammatory and antiviral gene expression in a national sample of U.S. adults.

Methods: We analyzed data from 1183 participants in the Midlife in the United States study. Discrimination attributions were measured by summing the number of different attributed reasons (range: 0–13). Gene expression was assessed using a validated 51-gene CTRA panel comprising 19 pro-inflammatory and 32 interferon/antibody genes. Multivariable regression models tested linear and quadratic associations, adjusting for assay plate, leukocyte subsets, daily discrimination, demographics, socioeconomic status, and lifestyle factors.

Results: We found no evidence of a linear association between discrimination attributions and the overall CTRA composite score. However, exploratory analyses suggested curvilinear associations between discrimination attributions and CTRA inflammatory and interferon/antibody sub-components. Expression levels were moderately elevated among those reporting no specific attributions relative to the low point observed at 2–4 attributions, and markedly elevated among those attributing discrimination to 11 or more reasons, particularly for the interferon/antibody sub-component.

Conclusion: Discrimination attributions relate to inflammatory and antiviral gene expression in a complex, nonlinear way. Findings suggest cognitive appraisals of discriminatory experiences may be an important factor influencing discrimination's biological impacts.

1. Introduction

Perceived discrimination (hereafter, discrimination) refers to unfair treatment directed at individuals because of social characteristics such as race, gender, or social class (Williams et al., 2019). A robust body of evidence demonstrates that experiences of discrimination are associated with increased emotional distress (e.g., depression and anxiety), elevated risk for cardiovascular disease, and premature mortality (Barnes et al., 2008; Chen and Mallory, 2021; Pascoe and Richman, 2009; Schmitt et al., 2014). However, the cognitive meaning individuals assign to discriminatory experiences, that is, their attributions about

why discrimination occurred, may be as consequential as the experiences themselves. Attributions involve evaluating whether a discriminatory event reflects a stable social identity (e.g., race, gender), a transient situational factor (e.g., a specific interaction, setting, or role-based expectation), or personal characteristics such as weight, age, or appearance (Major et al., 2002; Weiner, 1985).

Emerging evidence suggests that these distinctions carry independent health consequences (Crocker and Major, 1989; Schmitt et al., 2014; Vaccaro and Koob, 2019). Using data from the National Latino and Asian American Study, Gee and colleagues found that discrimination specifically attributed to race or ethnicity, as opposed to other

Abbreviations: CTRA, Conserved Transcriptional Response to Adversity.

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causes, was significantly associated with elevated depressive and anxiety disorders (Gee et al., 2007). More critically, the degree of stigma associated with a given identity appears to compound this effect, such that individuals who hold more highly stigmatized identities demonstrate correspondingly poorer health outcomes, reflecting a cumulative stigma burden (Graham, 2024; Link and Phelan, 2001; Prince et al., 2018; Schmitt et al., 2014). For example, Cobb and colleagues (2022) found that among older Black adults, attributing everyday discrimination to three or more sources, reflecting multiple stigmatized identities, was significantly associated with elevated risk of all-cause mortality, even after accounting for health, behavioral, and economic characteristics (Cobb et al., 2022). Consistent with this, a recent study found that among adults, the number of identity-based discrimination attributions, rather than discrimination frequency alone, was positively associated with allostatic load, a composite measure of multisystem physiological dysregulation (Graham, 2024). This further suggests that attributing discrimination to a greater variety of stigmatized identities may compound cumulative biological stress burden. Understanding attributions is therefore essential because the meaning individuals assign to discrimination—shaping emotional reactivity, threat appraisal, and expectations of future bias—engages sympathetic nervous system pathways that can promote long-term disease risk. (Major et al., 2002; Mendes et al., 2008; Sawyer et al., 2012; Thames et al., 2019). However, little is known about how discrimination attributional processes relate to the downstream biological processes, such as gene expression, that are believed to mediate the effects of sympathetic nervous system activity on disease development and health outcomes.

One biological framework for understanding how discrimination attribution might translate into disease risk is the Conserved Transcriptional Response to Adversity (CTRA) (Irwin and Cole, 2011; Slavich and Cole, 2013a; Cole, 2019). This gene expression profile is activated by chronic social threat processes, such as discrimination, that activate the sympathetic nervous system, triggering the release of norepinephrine (Cole, 2019). Norepinephrine binds to beta-adrenergic receptors, initiating signaling cascades that alter transcription factor activity and lead to the upregulation of pro-inflammatory transcription factors (e.g., *NF- κ B*, *AP-1*, *IL1B*, *IL6*, *IL8*, *TNF*), alongside downregulation of Type I interferon response factors and antibody synthesis genes (e.g., *IFN-*, *MX-*, *OAS-*, *IFI-*, *IRF-*, and *ISG*-family genes) (Cole, 2014, 2015, 2019). Collectively, these transcriptional alterations constitute a conserved molecular pattern through which discrimination can biologically impact immune cell function, ultimately contributing to heightened susceptibility to inflammatory-related chronic diseases, including cardiovascular disease, metabolic disorders, cancer progression, and neurodegenerative conditions (Cole, 2019; Fredrickson et al., 2013). Emerging empirical evidence supports discrimination as a trigger of this molecular profile. For example, McKenna and colleagues (2024) found that among Black trauma survivors, higher levels of lifetime discrimination were associated with significant increases in CTRA gene expression, while CTRA did not increase significantly among those with lower discrimination exposure (McKenna and Brennan, 2024). Similarly, a recent study found that daily discrimination was associated with pro-inflammatory gene expression in a national sample of midlife adults (Cuevas et al., 2023). These results are similar to initial findings from a much smaller study by Thames et al. that found perceived racial discrimination could potentially account for more than 50% of the race-related difference in pro-inflammatory transcription factor activity (Thames et al., 2019). However, none of these previous studies examined how the attributed cause of discrimination shapes this molecular signature, representing a fundamental gap in understanding how the cognitive meaning of discrimination becomes biologically embedded.

The present study addresses this critical gap by examining the relationship between discrimination attributions and CTRA-related gene expression. Specifically, we investigate whether individuals' attributions regarding the causes of discriminatory experiences (i.e., attributions to race/ethnicity, gender, weight, age, or other social categories)

are differentially associated with inflammatory and antiviral gene expression patterns characteristic of the CTRA profile. We hypothesize that higher greater attribution will be associated with elevated CTRA gene expression (increased pro-inflammatory and decreased antiviral gene expression). Because stress sensitization frameworks suggest that repeated discrimination may initially upregulate biological stress responses up to a threshold, beyond which continued exposure may lead to a blunting or downregulation of those responses, we also explored the possibility of a curvilinear association between discrimination and CTRA (Cole, 2019; McEwen, 2007; Miller et al., 2007).

2. Materials and Methods

2.1. Study Design and Setting

This study analyzed data from the Midlife in the United States (MIDUS) study, a nationally representative longitudinal investigation of English-speaking U.S. adults aged 25–74 years (Brim et al., 2004). The original MIDUS cohort was recruited in 1995–1996 through random digit dialing (MIDUS 1; $n = 7108$). Follow-up assessments were conducted in MIDUS 2 ($n = 5555$, including 592 additional African American participants from Milwaukee, Wisconsin to enhance racial and ethnic diversity; 2004–2005) and MIDUS 3 ($n = 3683$; 2013–2014). To expand the aging cohort, MIDUS Refresher 1 recruited 4085 new adults between 2011 and 2014, including 508 African Americans from Milwaukee, Wisconsin.

2.2. Biomarker Projects

Participants from MIDUS 3 and MIDUS Refresher 1 were invited to complete biomarker sub-studies involving clinical assessments and biological specimen collection (Dienberg Love et al., 2010). These individuals completed two-day clinical visits after providing informed consent and received \$200 compensation, plus reimbursement for travel and childcare expenses. The MIDUS team also arranged travel logistics and permitted older participants to bring companions. Complete study protocols are described elsewhere (Brim et al., 2004; Dienberg Love et al., 2010; Radler, 2014). The MIDUS Refresher Biomarker Study (2012–2016) enrolled 863 participants, and the MIDUS 3 Biomarker Project (2017–2022) enrolled 747 participants. Of the 1610 total biomarker participants, 1215 (75.5%) had valid gene expression data. We excluded 32 (2.6%) individuals with missing number of attributions to discrimination, yielding a final analytic sample size of 1183 participants.

The University of Wisconsin Institutional Review Board approved all MIDUS protocols. This secondary analysis was deemed exempt by the New York University Institutional Review Board because it used de-identified, publicly available data (<https://www.icpsr.umich.edu/web/ICPSR/series/203>). This study followed the Strengthening the Reporting of Observational Studies in Epidemiology guidelines (von Elm et al., 2008).

2.3. Measurement of the Conserved Transcriptional Response to Adversity

Blood specimens were obtained during participants' two-day clinical visits at one of three sites: Georgetown University, the University of Wisconsin-Madison, or the University of California, Los Angeles (Dienberg Love et al., 2010). Following an overnight fast, certified phlebotomists collected blood samples on the second day of the visit using BD Vacutainer CPT Tubes. White blood cells were isolated from whole blood and immediately cryopreserved at -60°C to -80°C . Samples were shipped on dry ice to the MIDUS Biocore Laboratory and maintained at -65°C until analysis. Genome-wide gene expression profiling was performed using whole-transcriptome RNA sequencing to quantify transcript abundance across all genes in the human genome. Sequencing was completed in two phases: MIDUS Refresher Biomarker

Study samples were processed between 2017 and 2018, while MIDUS 3 Biomarker Project samples were processed between 2018 and 2022.

The CTRA gene expression profile was assessed using a 51-gene transcript panel consisting of 19 pro-inflammatory genes and 32 genes related to interferon and antibody responses. This gene panel has been validated in prior research as capturing the conserved transcriptional response to adversity and social-environmental stress (Cole, 2013, 2014; Fredrickson et al., 2013; Irwin and Cole, 2011; Slavich and Cole, 2013b).

The primary outcome was the CTRA composite score, calculated as the mean expression of pro-inflammatory genes minus the mean expression of interferon/antibody genes. Secondary outcomes included the pro-inflammatory and interferon/antibody gene subcomponent scores analyzed separately. Gene expression levels were quantified as counts per million of total human transcriptome-aligned RNA sequencing reads. To stabilize variance across the expression range, values were log2-transformed after applying a floor of one transcript per million. This processing followed the standard MIDUS RNA sequencing pipeline, yielding centered log2-transformed gene expression values for all analyses. Gene expression profiling protocols are documented in the MIDUS Colectica Portal and previous publications (Bather et al., 2025; Cuevas et al., 2023; Rodrigues et al., 2025). The 19 pro-inflammatory genes were: *CXCL8*, *FOS*, *FOSB*, *FOSL1*, *FOSL2*, *IL1A*, *IL1B*, *IL6*, *JUN*, *JUNB*, *JUND*, *NFKB1*, *NFKB2*, *PTGS1*, *PTGS2*, *REL*, *RELA*, *RELB*, and *TNF*. The 32 interferon/antibody genes were: *GBP1*, *IFI16*, *IFI27*, *IFI27L1*, *IFI27L2*, *IFI30*, *IFI35*, *IFI44*, *IFI44L*, *IFI6*, *IFIH1*, *IFIT1*, *IFIT2*, *IFIT3*, *IFIT5*, *IFITM1*, *IFITM2*, *IFITM3*, *IFITM4P*, *IFITM5*, *IFNB1*, *IGLL1*, *IRF2*, *IRF7*, *IRF8*, *JCHAIN*, *MX1*, *MX2*, *OAS1*, *OAS2*, *OAS3*, and *OASL*.

2.4. Discrimination Attributions

Participants who reported experiencing discrimination were asked to identify the perceived reason(s) for these experiences by selecting all applicable options from a 13-item list: (1) age, (2) gender, (3) race, (4) ethnicity or nationality, (5) religion, (6) height or weight, (7) other aspect of physical appearance, (8) physical disability, (9) sexual orientation, (10) occupation, (11) financial status, (12) education, or (13) other reason. Responses were summed to create a count variable representing the number of different attributed reasons for discrimination (range: 0–13), with higher scores indicating attribution to a greater diversity of discrimination reasons. Respondents who did not report any discrimination were retained in the analytic sample with attributions to discrimination set to 0.

2.5. Covariates and Potential Mediators

Covariates included assay plate number, distinct leukocyte subsets, daily discrimination, demographic factors, socioeconomic status (SES), and lifestyle characteristics. Leukocyte subset composition was characterized using log2-transformed gene expression values for seven markers: *CD19*, *CD3*, *CD4*, *CD8A*, *CD14*, *FCGR3A*, and *NCAM1*. Daily discrimination was assessed using the 9-item Everyday Discrimination Scale (D. R. Williams et al., 1997), capturing frequency of routine unfair treatment. The scale comprised nine distinct items assessing routine interpersonal indignities: (1) being treated with less courtesy than others, (2) receiving less respect than others, (3) receiving poorer service at restaurants or stores, (4) people acting as if one is not smart, (5) people acting as if they are afraid, (6) people acting as if one is dishonest, (7) people acting as if one is inferior, (8) being called names or insulted, and (9) being threatened or harassed. Items were scored on a 4-point scale (1 = often to 4 = never), reverse-coded, and summed so that higher composite values indicated more frequent discrimination (Cronbach's $\alpha = 0.94$).

Demographic factors comprised age (measured continuously), sex (male/female), race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, non-Hispanic Other), and marital status (married, divorced/separated/widowed, never married). The non-Hispanic Other

group included Pacific Islander, Native Hawaiian, Asian, Native American, and Alaska Native. SES variables were educational attainment (high school or less, some college/associate's degree, college degree or higher) and annual household income (<\$50,000; \$50,000 to <\$100,000; \$100,000 +). Lifestyle characteristics included smoking status (never, past, current), past month alcohol consumption (never, <1 day/week, 1–2 days/week, ≥ 3 days/week), and body mass index (BMI, measured continuously). C-reactive protein ($\mu\text{g/mL}$) and interleukin-6 (pg/mL) were summarized descriptively but omitted from regression models, as they represent downstream biological pathways rather than pre-exposure confounders of the exposure-outcome associations.

2.6. Analytic Strategy

Descriptive statistics were generated for all variables. Means and SDs characterized continuous measures, and counts and proportions described categorical variables. Zero-order correlations assessed the unadjusted relationships among key study variables. Multiple imputation using fully conditional specification was employed to address missing data (Buuren and Groothuis-Oudshoorn, 2011). We specified polytomous logistic regression for nominal variables, proportional odds logistic regression for ordinal variables, and predictive mean matching for continuous variables. Results from ten imputed datasets were aggregated using Rubin's rules (Rubin, 1987).

Multivariable linear regression tested whether a quadratic relationship existed between attributions to discrimination and the CTRA composite score (primary analysis) (Hidalgo and Goodman, 2013). Model 1 adjusted for assay plate number and leukocyte subset composition (*CD19*, *CD3*, *CD4*, *CD8A*, *CD14*, *FCGR3A*, and *NCAM1*). Model 2 added daily discrimination. Model 3 further controlled for demographic characteristics (age, sex, race/ethnicity, marital status). Model 4 additionally adjusted for SES (educational attainment and annual household income). Model 5 incorporated lifestyle factors (smoking status, alcohol consumption, BMI). Secondary analyses followed the same covariate adjustment strategy for the CTRA subcomponents of pro-inflammatory and interferon/antibody gene expression. A two-sided $p < 0.05$ was considered statistically significant. We plotted adjusted predicted values for easier interpretation of findings.

2.7. Sensitivity Analyses

Sensitivity analyses were conducted to evaluate model robustness and potential component-level heterogeneity. First, models were re-estimated without adjusting for race/ethnicity. Second, to assess whether the composite CTRA score masked divergent associations across its underlying subcomponents, we implemented person-clustered generalized estimating equations with an exchangeable working correlation structure (LIANG and ZEGGER, 1986). Linear and quadratic discrimination attribution terms were interacted with indicator variables for the CTRA subcomponents.

3. Results

The analytic sample included 1183 participants with a mean age of 57.0 years ($SD = 12.2$); 52.7% were female and 73.7% were non-Hispanic White (Table 1). Zero-order correlations showed no statistically significant associations between attributions to discrimination and the CTRA composite score ($r = -0.017$, $p = 0.57$), inflammatory subcomponent ($r = 0.008$, $p = 0.78$), or interferon/antibody subcomponent ($r = 0.019$, $p = 0.51$; Table 2). Table 3 and Supplemental Tables 1–3 present adjusted associations between discrimination attributions and CTRA outcomes across five models. For the primary outcome, the CTRA composite score did not vary significantly as either a linear or quadratic function of discrimination attributions in any model. However, suggestive curvilinear (quadratic) associations emerged for

Table 1

Summary statistics on 1183 individuals from the Midlife in the United States study.

Characteristic	N = 1183
Age, Mean (SD)	57.0 (12.2)
Sex, n (%)	
Male	560 (47.3)
Female	623 (52.7)
Race/ethnicity, n (%)	
non-Hispanic White	872 (73.7)
non-Hispanic Black	189 (16.0)
Hispanic	37 (3.1)
non-Hispanic Other	78 (6.6)
Missing	7 (0.6)
Marital status, n (%)	
Married	739 (62.5)
Divorced/Separated/Widowed	272 (23.0)
Never married	171 (14.5)
Missing	1 (0.1)
Educational attainment, n (%)	
High school or less	229 (19.4)
Some college/associate's degree	369 (31.2)
College degree or higher	584 (49.4)
Missing	1 (0.1)
Annual household income, n (%)	
< \$50,000	557 (47.1)
\$50,000 to \$100,000	281 (23.8)
\$100,000 +	265 (22.4)
Missing	80 (6.8)
Smoking status, n (%)	
Never	702 (59.3)
Past	369 (31.2)
Current	112 (9.5)
Alcohol consumption, n (%)	
Never	383 (32.4)
< 1 day a week	314 (26.5)
1–2 days a week	195 (16.5)
3 + days a week	291 (24.6)
Body mass index (kg/m ²), Mean (SD)	28.8 (6.6)
Missing, n (%)	26 (2.2)
C-reactive protein (µg/mL), Mean (SD)	3.5 (5.4)
Interleukin-6 (pg/mL), Mean (SD)	3.3 (3.1)
Missing, n (%)	4 (0.3)
Daily discrimination, Mean (SD)	12.9 (4.9)
Missing, n (%)	9 (0.8)
Number of attributions to discrimination, Mean (SD)	1.0 (1.7)
CTRA composite score (centered log ₂ gene expression values), Mean (SD)	0.0 (0.6)
CTRA Inflammatory sub-component (centered log ₂ gene expression values), Mean (SD)	0.0 (0.8)
CTRA Interferon/Antibody sub-component (centered log ₂ gene expression values), Mean (SD)	0.0 (0.8)
CD19 (log ₂ gene expression values), Mean (SD)	4.2 (2.4)
CD3 (log ₂ gene expression values), Mean (SD)	7.3 (2.0)
CD4 (log ₂ gene expression values), Mean (SD)	7.2 (2.3)
CD8A (log ₂ gene expression values), Mean (SD)	5.4 (2.5)
CD14 (log ₂ gene expression values), Mean (SD)	8.8 (2.2)
FCGR3A (log ₂ gene expression values), Mean (SD)	7.2 (2.4)
NCAM1 (log ₂ gene expression values), Mean (SD)	4.4 (2.6)

both the inflammatory and antiviral/antibody sub-components of the CTRA (Table 3 and Supplemental Tables 2 and 3).

Fig. 1 displays predicted centered log₂ gene expression values across the range of discrimination attributions for both inflammatory and interferon/antibody gene pathways. Both sub-components showed a curvilinear, U-shaped pattern, with the lowest expression occurring among respondents reporting approximately 2–4 attributions to discrimination. Expression was modestly higher among those reporting no attributions than at this low point, and it increased steadily from there, climbing above the sample average among respondents reporting approximately 9–10 or more attributions. The two sub-components tracked closely together across the low-to-moderate range of attributions and met at around 10 attributions, but they separated at the high end. The interferon/antibody sub-component climbed more steeply than

the inflammatory sub-component among respondents reporting 11 or more attributions, ending with the highest predicted expression values in the sample at 13 attributions. Sensitivity analyses demonstrated that the observed associations were robust to the exclusion of race/ethnicity as a covariate (Supplemental Tables 4–6). Furthermore, evaluations of component-level heterogeneity confirmed that the summary CTRA composite score did not mask divergent or conflicting relationships among its underlying sub-components (Supplemental Table 7).

4. Discussion

This study examined the relationship between discrimination attributions and inflammatory gene expression profiles characteristic of CTRA. We did not find an association between discrimination attributions and the CTRA composite score. However, exploratory analyses suggested curvilinear associations between the number of discrimination attributions and both CTRA sub-components, pro-inflammatory and interferon/antibody gene expression. Expression levels were moderately elevated among those reporting no specific attributions relative to the low point observed at 2–4 attributions, and markedly elevated among those attributing discrimination to 11 or more reasons, particularly for the interferon/antibody sub-component. These patterns persisted after adjusting for assay plate, leukocyte subsets, daily discrimination, demographic characteristics, SES, and lifestyle factors, although the curvilinear pattern for the inflammatory sub-component was attenuated in the most fully adjusted models.

Previous studies have consistently demonstrated that greater discrimination exposure predicts worse health outcomes in a monotonic fashion (Lam et al., 2025; Williams et al., 2019). However, our findings suggest that the biological impacts of discrimination attribution may operate through more nuanced psychological and biological mechanisms among midlife and older U.S. adults. Moreover, the observed U-shaped pattern may reflect fundamental differences in how individuals cognitively integrate discriminatory experiences. For example, participants reporting a moderate number of attributions (approximately 2–4), may have developed more coherent cognitive schemas for understanding and coping with discrimination, potentially serving as a form of cognitive buffering that attenuates subsequent neural and inflammatory responses (Park et al., 2010). This interpretation aligns with research showing that attributing discrimination to specific, identifiable characteristics enhances perceived control and facilitates more effective coping strategies, which in turn mitigate inflammatory stress responses (Dasgupta et al., 2025; Dong et al., 2023; Jang et al., 2008).

Conversely, the elevated gene expression observed at both extremes may reflect distinct forms of stress exposure. Among those reporting no specific attributions, discrimination experiences may be characterized by greater ambiguity and unpredictability, known to amplify stress responses (Meyer, 2003; Moody et al., 2025). Attribution ambiguity has been shown to intensify the psychological burden of discrimination by preventing effective cognitive processing and coping strategy implementation (Folkman, 2013). At the opposite extreme, attributing discrimination to eleven or more reasons may signal exposure to discrimination across multiple marginalized identities, which research has linked to cumulative disadvantage and heightened physiological stress (Wang et al., 2025; Williams et al., 2023). These individuals may face chronic, multifaceted discrimination that broadly mobilizes both inflammatory and antiviral transcriptional patterns, despite possessing cognitive clarity about discrimination sources.

The observed curvilinear patterns can be interpreted through the lens of stress-appraisal theory (Folkman, 2013). Attributional judgments shape how individuals evaluate the meaning, controllability, and anticipated recurrence of discriminatory experiences (Major et al., 2002), processes that regulate anticipatory coping and physiological preparedness for potential threat (Sawyer et al., 2012). When discrimination lacks a clear attribution, individuals may experience heightened uncertainty and anticipatory vigilance (Major et al., 2003), both of

Table 2

Zero-order correlations among key study variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Attributions to discrimination	-											
2. C-reactive protein (μg/mL)	0.14***	-										
3. Interleukin-6 (pg/mL)	0.06*	0.38***	-									
4. CTRA composite score	-0.02	-0.11***	-0.07*	-								
5. CTRA Inflammatory sub-component	0.01	-0.03	0.01	0.26***	-							
6. CTRA Interferon/Antibody sub-component	0.02	0.04	0.06	-0.45***	0.75***	-						
7. <i>CD19</i>	0.03	0.02	0.02	0.08**	0.31***	0.23***	-					
8. <i>CD3</i>	-0.03	0.11***	0.13***	-0.02	0.18***	0.18***	0.29***	-				
9. <i>CD4</i>	-0.07*	0.10***	0.16***	-0.02	0.10***	0.11***	0.13***	0.80***	-			
10. <i>CD8A</i>	-0.01	0.06*	0.07*	-0.06*	0.28***	0.30***	0.26***	0.63***	0.50***	-		
11. <i>CD14</i>	-0.10***	0.10***	0.18***	-0.02	0.25***	0.25***	0.17***	0.68***	0.76***	0.53***	-	
12. <i>FCGR3A</i>	-0.07*	0.13***	0.15***	-0.08**	0.13***	0.18***	0.10**	0.66***	0.76***	0.46***	0.74***	-
13. <i>NCAM1</i>	-0.01	0.07**	0.08**	0.02	0.27***	0.24***	0.25***	0.57***	0.49***	0.52***	0.50***	0.49***

CTRA = Conserved Transcriptional Response to Adversity.

The following variables reflect centered log2 gene expression values: CTRA composite score, CTRA Inflammatory sub-component, CTRA Interferon/Antibody sub-component.

The following variables reflect log2 gene expression values: *CD19*, *CD3*, *CD4*, *CD8A*, *CD14*, *FCGR3A*, *NCAM1*.* $p < .05$.** $p < .01$.*** $p < .001$.

which amplify sympathetic activation and inflammatory signaling (Sawyer et al., 2012). Conversely, attributing discrimination to multiple social identities may foster expectations of persistent, identity-based discrimination, similarly sustaining anticipatory stress (Major et al., 2003; Sawyer et al., 2012; Seng et al., 2012; Weiner, 1985). These appraisal and coping dynamics provide a plausible psychological mechanism linking attributional patterns to the curvilinear gene expression profiles observed in this study.

The U-shaped associations observed for both inflammatory and

interferon/antibody sub-components, which tracked closely across the low-to-moderate range of attributions, suggest that attributional diversity may influence broad transcriptional programs rather than selectively affecting specific immune pathways. This coordinated regulation across both gene sets of the CTRA profile indicates that discrimination attributions may modulate upstream regulatory mechanisms, such as sympathetic nervous system activation or transcription factor activity (Cole, 2019). Furthermore, the two sub-components converged at approximately 10 attributions and diverged thereafter, with

Table 3

Adjusted associations of attributions to discrimination with inflammatory and antiviral gene expression, Midlife in the United States study.

Characteristic	Model 1		Model 2		Model 3		Model 4		Model 5	
	Beta (95% CI)	P-value	Beta (95% CI)	P-value	Beta (95% CI)	P-value	Beta (95% CI)	P-value	Beta (95% CI)	P-value
CTRA composite score (centered log2 gene expression values)										
Attributions (linear)	0.021 (-0.018–0.059)	0.29	0.029 (-0.015–0.072)	0.20	0.035 (-0.008–0.079)	0.11	0.035 (-0.009–0.080)	0.12	0.035 (-0.009–0.079)	0.12
Attributions (quadratic)	-0.004 (-0.009–0.001)	0.12	-0.004 (-0.009–0.001)	0.093	-0.004 (-0.009–0.001)	0.10	-0.004 (-0.009–0.001)	0.11	-0.004 (-0.009–0.001)	0.11
CTRA Inflammatory sub-component										
Attributions (linear)	-0.023 (-0.060–0.015)	0.23	-0.024 (-0.067–0.018)	0.26	-0.028 (-0.071–0.015)	0.20	-0.022 (-0.065–0.021)	0.31	-0.022 (-0.066–0.021)	0.31
Attributions (quadratic)	0.005 (0.001–0.010)	0.032	0.005 (0.001–0.010)	0.034	0.005 (0.001–0.010)	0.037	0.005 (-0.001–0.009)	0.071	0.005 (-0.001–0.010)	0.065
CTRA Interferon/Antibody sub-component										
Attributions (linear)	-0.043 (-0.089–0.002)	0.061	-0.053 (-0.105 to -0.001)	0.045	-0.063 (-0.115 to -0.011)	0.017	-0.058 (-0.110 to -0.006)	0.030	-0.057 (-0.109 to -0.005)	0.032
Attributions (quadratic)	0.009 (0.003–0.015)	0.002	0.010 (0.004–0.015)	0.002	0.009 (0.003–0.015)	0.002	0.009 (0.003–0.015)	0.004	0.009 (0.003–0.015)	0.004

Abbreviation: CI = Confidence Interval, CTRA = Conserved Transcriptional Response to Adversity.

Results were pooled across ten imputed datasets.

Bold indicates $p < 0.05$.Model 1 controlled for assay plate and log2 transformations of *CD19*, *CD3*, *CD4*, *CD8A*, *CD14*, *FCGR3A*, and *NCAM1*.

Model 2: Model 1 + daily discrimination.

Model 3: Model 2 + age, sex, race/ethnicity, and marital status.

Model 4: Model 3 + educational attainment and annual household income.

Model 5: Model 4 + smoking status, alcohol consumption, and body mass index.

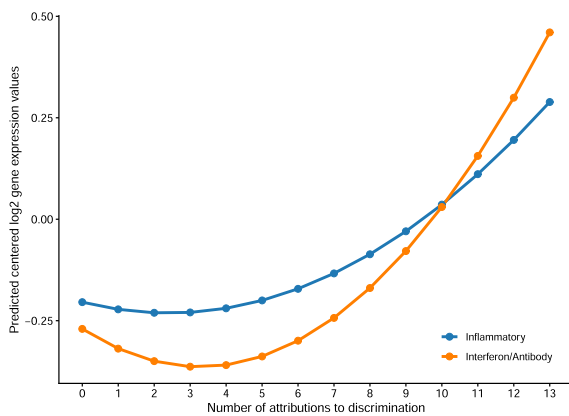


Fig. 1. Adjusted predicted values of the inflammatory and interferon/antibody sub-components as a function of discrimination attributions. These values controlled for age, sex, race/ethnicity, marital status, assay plate, daily discrimination, educational attainment, annual household income, smoking status, alcohol consumption, body mass index, and log2 gene expression values of *CD19*, *CD3*, *CD4*, *CD8A*, *CD14*, *FCGR3A*, and *NCAM1*.

interferon/antibody expression rising more steeply than inflammatory expression among respondents reporting 11 or more attributions, suggesting that attributional processing may shape the relative magnitude of these molecular responses at the upper extreme of the attribution distribution rather than their overall direction.

The absence of significant linear or curvilinear associations with the CTRA composite score, despite curvilinear patterns in both sub-components, most consistently for interferon/antibody genes, warrants careful consideration and suggests that discrimination attributions may influence overall transcriptional activity rather than producing the signature CTRA profile of reciprocal regulation between inflammatory and antiviral pathways. This discrepancy underscores the importance of examining CTRA sub-components separately rather than relying exclusively on composite scores. While the composite metric effectively captures reciprocal inflammatory-antiviral regulation in many contexts, our results demonstrate that social-cognitive processes may produce transcriptional patterns that are obscured by difference scores. Additionally, the high end of the attribution distribution fits least well with wear-and-tear theory, as a purely chronic stress explanation would predict continued antiviral gene expression decline alongside rising inflammation, yet antiviral expression reversed at extreme attribution counts. Several explanations may explain these unexpected findings. First, very high attribution counts may mark a qualitatively different exposure than moderate counts, characterized less by steady low-grade stress and more by an acute, high-vigilance state in which the immune system mobilizes broadly rather than reallocating resources between bacterial/wound-related and viral defense pathways. Second, a plausible technical explanation involves shifts in the relative proportions of circulating leukocyte subtypes, which differ in their baseline antiviral gene expression and can produce apparent transcriptional shifts without any true change in regulation within a given cell type. Third, relatively few participants fall at the extreme end of the attribution count distribution, meaning this portion of the curve is the least statistically stable and should be interpreted with caution until replicated in a larger or independent sample.

Taken together, these findings carry important implications for research on discrimination and health. First, they underscore that the number of attributed reasons for discrimination, not merely its frequency or severity, may be a meaningful predictor of biological stress responses. This suggests that discrimination attribution should be assessed routinely alongside these more commonly used measures in studies examining the biological consequences of discrimination. Second, the curvilinear nature of the associations challenges the assumption

in the literature that greater discrimination exposure produces monotonically worse biological outcomes.

Several limitations merit consideration. First, the cross-sectional design precludes causal inference and determination of temporal ordering between discrimination attributions and gene expression. Longitudinal designs tracking discrimination experiences, cognitive appraisals, and repeated gene expression measurements over time are needed to establish temporal precedence and test dynamic relationships. Second, the count-based approach treated all attributions equally, not accounting for potential differences in the salience, chronicity, or impact of discrimination based on different social identities. Future research incorporating multidimensional attribution assessments could provide more granular understanding of how specific attributional characteristics relate to gene expression. Third, we cannot rule out residual confounding from unmeasured factors that might influence both discrimination attributions and gene expression. Finally, the sample, while diverse, consisted primarily of non-Hispanic White participants, potentially limiting generalizability to other racial and ethnic groups. Future studies with larger and more racially and ethnically diverse samples should examine whether the curvilinear relationship between attributional breadth and gene expression differs across racial and ethnic groups.

5. Conclusion

This study provides evidence that discrimination attributions relate to inflammatory and antiviral gene expression in a curvilinear pattern, such that expression levels were elevated among individuals reporting both no specific attributions and, most markedly, those attributing discrimination to 11 or more reasons. This pattern was most consistent for the interferon/antibody sub-component, which remained statistically significant after adjusting for demographic, socioeconomic, and lifestyle factors, whereas the corresponding pattern for the inflammatory sub-component was attenuated in fully adjusted models. Collectively, these findings underscore the importance of social-cognitive factors in shaping how discriminatory experiences become biologically embedded. Further research is needed to better understand the range of social-cognitive factors that influence the meaning individuals assign to discrimination and, in turn, their downstream effects on gene expression.

Clinical trial number

Not applicable.

Ethics declaration

Written informed consent to take part in the study and to publish the article has been obtained from all participants or their legal representatives. The privacy rights of participants have been observed.

This study was performed in compliance with relevant laws, regulatory frameworks and guidelines where the research took place. The New York University granted an exemption. The authors provided the following explanation: De-identified participant information via secondary data.

CRediT authorship contribution statement

Adolfo G. Cuevas: Writing – review & editing, Writing – original draft, Investigation, Funding acquisition. **Jemar R. Bather:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation. **Alisha A. Crump:** Writing – review & editing, Writing – original draft, Investigation, Conceptualization. **Steven W. Cole:** Writing – review & editing, Conceptualization. **Emiko O. Kranz:** Writing – review & editing. **Ariel A. Binns:** Writing – review & editing, Writing – original draft. **Mariana Rodrigues:** Writing – review &

editing, Writing – original draft.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.psyneuen.2026.107983](https://doi.org/10.1016/j.psyneuen.2026.107983).

Data availability statement

The data that support this study's findings are publicly available through the MIDUS Colectica Portal and the Inter-university Consortium for Political and Social Research (ICPSR). Specific datasets include: ICPSR 36532 (<https://doi.org/10.3886/ICPSR36532>), ICPSR 36722 (<https://doi.org/10.3886/ICPSR36722>), ICPSR 36901 (<https://doi.org/10.3886/ICPSR36901>), ICPSR 36346 (<https://doi.org/10.3886/ICPSR36346>), ICPSR 37120 (<https://doi.org/10.3886/ICPSR37120>), and ICPSR 38837 (<https://doi.org/10.3886/ICPSR38837>).

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