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Immune response and intergroup bias: Vaccine-induced increases in cytokine activity are associated with worse evaluations of resume for Latina job applicant

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ABSTRACT

Situational factors can increase people's vulnerability to intergroup bias, including prejudicial attitudes, negative stereotyping, and discrimination. We proposed that increases in inflammatory activity that coincide with acute illness may represent a hitherto unstudied situational factor that increases intergroup bias. The current study experimentally manipulated increases in inflammatory activity by administering the seasonal influenza vaccine or a saline placebo. We quantified inflammatory activity by assessing change in salivary pro-inflammatory cytokines and assessed intergroup bias using a resume evaluation task and self-reported ethnocentrism. Primary analyses focused on a subsample of 117 participants who provided high quality data; robustness analyses included various permutations of lower quality participants. Findings revealed that changes in the cytokine interleukin-1β (IL-1β) in response to the vaccine were associated with greater intergroup bias. Among participants who received the vaccine, IL-1β change was negatively associated with evaluation of a Latina (but not a White woman) applicant's competency and recommended starting salary. Moreover, IL-1β change was positively associated with ethnocentrism. Overall, results provide support for the hypothesis that acute illness, via the mechanistic role of inflammatory cytokines, affects social cognition in ways that can increase intergroup bias.

Imagine Alex, a manager in a medium-scale company. One of Alex's main responsibilities is hiring new talent. One Tuesday, Alex wakes up feeling under the weather—but not quite "bad enough" to use sick leave. Alex gets ready and makes the usual commute into work. Upon sitting down at the office, Alex finds a few new job applications in the portal. Still under the weather, Alex begins to sort and evaluate the resumes. Could being sick lead Alex to make different candidate selection decisions than Alex would have made otherwise? The present research attempts to answer this question.

Everyone falls prey to social biases at one time or another. Numerous situational factors can increase the likelihood that biases will affect people's decision-making and social cognition (e.g., Bodenhausen, 1990; Fein & Spencer, 1997; Gilbert & Hixon, 1991; Kruglanski & Freund, 1983; Rogers & Prentice-Dunn, 1981). In the present research, we propose that another important situational factor may increase intergroup bias: illness. Specifically, we theorize that acute activation of the immune system may increase intergroup bias. Notably, we use the term intergroup bias as an overarching term that captures prejudicial attitudes, negative stereotypes, and discrimination aimed at a person or people from another racial, ethnic, or cultural group.

1. The immune systems and their behavioral influences

Increases in cytokines prompt broad changes in behavior (Dantzer &

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Kelley, 2007). Some of those behavioral changes are thought to facilitate rest and recuperation from illness by reducing energy demands of one's typical behavior and thereby balancing the necessary increased energy demands of mounting a cytokine response (Dantzer, 2001; Dantzer & Kelley, 2007). Those behavioral changes are collectively termed "sickness behaviors" and include changes in mood (e.g., anhedonia, depression), lethargy, and social withdrawal. Increases in cytokines also increase anxiety (Kullmann et al., 2013; Reichenberg et al., 2001) and appear to draw attention inward to focus on one's physiological processes (Harrison et al., 2009). Several other downstream consequences of increased cytokines include reduced self-regulation and motivation (Dantzer & Kelley, 2007; Harrison et al., 2015; Shields et al., 2017), as well as a shift toward more immediate gratification (Gassen et al., 2019b).

Increases in cytokines also affect social behavior specifically (Eisenberger et al., 2017). On the one hand, increases in cytokines have been linked to greater attunement toward close others (Inagaki et al., 2012; Jolink et al., 2022, Muscatell et al., 2016). On the other hand, increases in cytokines have also been linked to greater vigilance toward and avoidance of socially distant others (Inagaki et al., 2012; Jolink et al., 2022). These findings come from studies in which cytokine levels were experimentally increased via the administration of a substance that simulates a pathogen (e.g., endotoxin, vaccine). In response to such manipulations that simulate the state of illness, increases in cytokines are associated with greater amygdala activation in response to socially threatening images (Inagaki et al., 2012) and greater automatic avoidance of unknown social targets (Jolink et al., 2022).

Changes in social behavior in response to increases in cytokines may thus have adaptive function beyond preservation of energy resources. Illness is a time of increased vulnerability, when an individual often needs care and assistance from others and is more vulnerable to threats. Indeed, increases in cytokines appear to sensitize people to both positive and negative social information, which may function to regulate approach and avoidance behaviors (Eisenberger et al., 2017). Close others are likely to offer an individual care and support, and would thus be adaptive to approach (Muscatell & Inagaki, 2021). Strangers, however, are potential sources of threat and would be adaptive to be vigilant of and avoid (Neuberg et al., 2011).

Overall, increases in cytokines seem to facilitate greater desire for closeness with close others yet a greater desire for distance from distant others. That link is also supported by insights from computational modeling of the spread of viral infections demonstrating that selective withdrawal from others on the periphery of people's social networks is more effective for halting the spread of illness than generalized social withdrawal from everyone (Cole, 2006). Whether cytokine-induced desire for affiliation with close others coupled with withdrawal from socially distant others translates into intergroup bias, however, is currently unknown.

2. Acute inflammation and bias

In the present research, we hypothesize that acute inflammation, as quantified by increases in cytokines, will be associated with greater intergroup bias. Support for this hypothesis comes from three distinct literatures. First, increases in cytokines may lead to greater intergroup bias due to reduced cognitive effort and motivation. The links between increases in cytokines and anxiety (Kullmann et al., 2013; Reichenberg et al., 2001), as well as greater attention toward internal processes (Harrison et al., 2009), parallels social psychology research guided by the distraction hypothesis (Bodenhausen, 1993; Wilder & Simon, 2003). States characterized by heightened physiological arousal—for example, arousing emotions (Wilder, 1993), exercise (Kim & Baron, 1988; Wann & Branscombe, 1995), and stress (Keinan et al., 2000)—limit the cognitive resources used to understand the social environment and consequently lead to biased social judgments. Furthermore, the use of bias-reduction strategies requires motivation and self-regulation (Devine, 1989; Devine & Sharp, 2009; Monteith, 1993). Consequently, the reduced motivation and capacity for self-regulation stemming from acute increases in cytokines (Dantzer & Kelley, 2007; Harrison et al., 2015; Shields et al., 2017) may reduce people's typical use of bias-reduction strategies and, in turn, any automatically activated stereotypes (Devine, 1989) may result in increased intergroup bias.

Second, increases in cytokines may lead to greater intergroup bias due to greater ingroup favoritism and outgroup derogation. That is, the link between increases in cytokines and desire for closeness with close others, but desire for distance from distant others, parallels the opposing attitudes toward one's ingroup versus one's outgroup. Even in the context of novel or lab-created groups, people demonstrate an affinity toward ingroup members and antipathy for those in the outgroup (Brewer, 1979; Tajfel et al., 1971). Indeed, simply categorizing someone as an outgroup member initiates social withdrawal (Paladino & Castelli, 2008). Consequently, the avoidance of strangers (Jolink et al., 2022) and vigilance to social threats (Inagaki et al., 2012)—which are often associated with outgroups (Cottrell & Neuberg, 2005; Zárate et al., 2004)—resulting from increased cytokines may also extend to greater intergroup bias. Moreover, because people engage in less frequent contact with groups they are prejudiced against (Allport, 1954; Maunder et al., 2020; Schwab et al., 2019), those groups would be likely targets of selective withdrawal from those on the periphery of one's social network.

Finally, increases in cytokines may lead to greater intergroup bias due to heightened pathogen avoidance motives. Pathogen avoidance motives facilitate people's use of proactive and reactive strategies that function to mitigate the threat of illness (Ackerman et al., 2018; Schaller & Park, 2011).[1] Such psychological strategies are theorized to complement immune system processes by serving as a first round of defense against pathogen threat, which is a defense that may require the expenditure of far fewer energetic or immune resources than a full-fledged inflammatory response. When pathogen avoidance motives are high, people demonstrate pathogen-defensive behavior against targets who may not actually be harboring illness but are only *heuristically* associated with illness (Schaller & Park, 2011). Indeed, the link between pathogen avoidance motives and intergroup bias has been widely documented (Aarøe et al., 2017; Brown et al., 2019; Faulkner et al., 2004, Makhanova et al., 2021; Navarrete & Fessler, 2006; O'Shea et al., 2019). It has been suggested that, because both pathogen avoidance strategies and the immune system serve the same functional purpose of pathogen defense, there may be crosstalk between these processes (Ackerman et al., 2018; Clark & Fessler, 2014; Murray et al., 2019). Consequently, increases in cytokines may cue the activation of pathogen avoidance strategies, including greater intergroup bias.

3. Current research

To examine whether increases in cytokines lead to greater intergroup bias, we randomized participants to receive either a cytokine-stimulating vaccine—namely, influenza vaccination (Boyle et al., 2019; Kuhlman et al., 2018; Jolink et al., 2022)—or a placebo (saline) control injection, and subsequently assessed intergroup bias. To that end, approximately 24-hours after they received either the vaccine or placebo at the clinic, when the peak increases in cytokines were expected to occur (Radin et al., 2021), participants completed measures of intergroup bias with instructions provided over Zoom. We assayed saliva samples participants provided before the clinic appointment and before the Zoom meeting for three pro-inflammatory cytokines associated with the general response of the innate immune system: interleukin-1β (IL-

[1] The psychological strategies aimed at pathogen avoidance are frequently termed "the behavioral immune system." Because we are focused on psychoneuroimmunology and immune system activity, we opted not to use that term to increase clarity and to avoid creating a false dichotomy between the two systems (i.e., increases in cytokines also prompt behavioral changes).

1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α).

Our primary measure of intergroup bias was a resume evaluation task in which participants saw and evaluated two resumes, one from a Latina applicant and one from a White applicant. Resume evaluation tasks are a common method to examine discrimination and biased behavior (Bertrand & Mullainathan, 2004; Moss-Racusin et al., 2012). For example, research examining bias among science faculty found that the same resume for a lab manager position with a female-typical name (vs. a male-typical name) was rated as less competent and offered a lower starting salary (Moss-Racusin et al., 2012). Resume tasks have also been used to document instances of discrimination due to race and ethnicity (Bertrand & Mullainathan, 2004; Derous et al., 2009; Widner & Chicoine, 2011).

Participants also completed a general ethnocentrism scale (Neuliep & McCroskey, 1997). Ethnocentrism reflects people's beliefs that their group is superior in its customs compared to other groups. Such beliefs promote and reinforce negative attitudes toward people belonging to outgroups. Indeed, people who report greater ethnocentrism report having lower frequency of contact with people from different cultures (Neuliep & McCroskey, 1997). Moreover, ethnocentrism was negatively associated with perceived competence of and hiring recommendations for an Asian job applicant (Neuliep et al., 2005).

We tested pre-registered hypotheses that participants who received the influenza vaccine, compared to those who received the placebo, would demonstrate greater cytokine change and greater intergroup bias. Specifically, we hypothesized that participants in the vaccine condition, compared to those in the placebo condition, would rate the Latina applicant as less competent and would recommend that she receive a lower starting salary. We did not predict that condition would affect evaluations of the White applicant. Furthermore, we hypothesized that participants in the vaccine condition, compared to those in the placebo condition, would report higher ethnocentrism. Given that past research using the influenza vaccine manipulation did not use a placebo condition (Boyle et al., 2019; Kuhlman et al., 2018; Jolink et al., 2022), we hypothesized that cytokine change would be positively associated with bias specifically among participants who received the vaccine.[2]

4. Method

4.1. Participants

Participants were recruited from the community. All participants completed a phone screening to determine eligibility. Participants were eligible if they (a) were between 18–40 years old, (b) had a body-mass index (BMI) between 18.5–30, (c) had not received the annual influenza vaccine that season, (d) had never had an allergic reaction to the influenza vaccine or other vaccines, (e) were not pregnant, (f) did not have any illnesses known to affect cytokine levels (e.g., auto-immune disorders, hypothyroidism, sleep disorders), (g) were not taking medication known to affect cytokine levels (e.g., SSRIs, steroids), and (h) did not smoke or use tobacco products. See Supplementary Materials for the full list of exclusion criteria. Participants were compensated with a $40 Amazon gift card for participating in the study.

Because data collection took place during the COVID-19 pandemic, we relied on participants completing tasks and providing saliva samples remotely. Unfortunately, this led to a large number of exclusions due to technological problems and failures to follow instructions, especially regarding the timing of saliva samples. In the manuscript, we focus on the subsample of participants (n = 117) who provided saliva samples within acceptable (but not ideal) time frames. Of those participants, 5 did not complete the resume evaluation task because of technological problems and 3 different participants did not complete the ethnocentrism measure because they ran out of time during their session. Furthermore, per our preregistration, models examining bias on the resume evaluation measure additionally excluded participants (n = 6) who did not hear the audio and models examining cytokine change additionally excluded participants (n = 6) who had IL-1β levels greater than three standard deviations above the sample mean. Because the target of bias on the resume evaluation measure was Latina, this subsample excluded otherwise eligible participants who identified as Latine (n = 12). However, we conducted robustness analyses without these exclusions (see Robustness Analyses section for details).

Participants were on average 25 years old (M=25.29, SD=5.87). The majority of participants (n = 70) identified their gender as female; 34 identified as male, and 1 ran out of time during the Zoom session and did not complete the demographics questionnaire. The majority of participants identified as White (n = 85); 8 participants identified as Asian, 3 as Black, 1 as Native American, and 7 as multiracial and other. When asked to describe their political orientation on a scale of 1 (*Very Liberal*) to 10 (*Very Conservative*), participants' responses were on average slightly more liberal than the scale midpoint (M=4.24, SD=2.12, range: 1–9). This skew was more pronounced when examining frequencies: Only 30 participants identified their political orientation as more conservative than the midpoint.

4.2. Procedure and Materials

All study procedures were performed in compliance with ethical standards set forth by the American Psychological Association, relevant laws, and University of Arkansas policies. The protocol (2006269408) was approved by the University of Arkansas institutional review board on August 20, 2021. Data collection took place during two influenza vaccine administration seasons (Season 1: October 13, 2020-March 5, 2021, n = 46; Season 2: September 22, 2021- March 18, 2022, n = 59). Participants completed eligibility screening and informed consent over the phone. After providing consent, participants were scheduled for an appointment at the clinic, where they received either a placebo injection (n = 41) or the seasonal influenza vaccine (n = 64).[3] Vaccine strains are reported in the Supplementary Materials. Placebo injections were 0.5 mL of saline solution. The nurses administering the vaccines were not blind to participant condition, but participants and research assistants were blind to condition until the end of the study. After participants were unblinded, those in the placebo condition returned to the clinic to get their vaccine.

Participants provided their baseline saliva sample before getting the vaccine or placebo injection and their second saliva sample around the time of their Zoom session, which for the current subsample meant no more than 4 h before and no more than 2 h after the session.[4] On average, there was a 24-hour difference between samples (M=24.56, SD=3.56, range: 17.25–33.83), which corresponds to the anticipated peak in cytokine responses following the administration of influenza vaccine (Radin et al., 2021). Samples were assayed for IL-1β, IL-6, and

[2] We preregistered additional mediation and moderation hypotheses (i.e., whether changes in cytokines mediate the effect of condition on bias; whether certain factors attenuate the association between increases in cytokines and intergroup bias). We report the tests of these hypotheses in the Supplemental Materials because we did not have the statistical power and experimental control in our final sample to draw strong conclusions about those findings (or lack thereof).

[3] The difference in frequencies between conditions is due to the fact that random assignment was done in advance for the entire anticipated sample size using simple random assignment (i.e., without the use of a block design). However, we did not reach the anticipated sample size.

[4] Instructions participants received for providing the saliva samples are included in the Supplemental Materials.

TNF-α using commercially available multiplexing assay kits (Meso Scale Discovery [MSD], Rockville, MD, United States) per manufacturer instructions.[5] Across all samples assayed, the intra-assay CV for these assays was 3.90 % and the inter-assay CV was 4.35 %. Participants included in our analyses had detectable levels of all three cytokines for both saliva samples. As typical in PNI research, we transformed cytokine levels via natural log transformation to correct skew (e.g., Gassen et al., 2019a; Shields et al., 2016). To model cytokine change, we used residualized change between the two samples (Cronbach & Furby, 1970; Shields et al., 2019a). Because we only found consistent and robust results with IL-1β, we present these models in text. Results for IL-6 and TNF-α are included in the Supplementary Materials.

Participants completed dependent measures of intergroup bias during Zoom sessions with research assistants that lasted approximately ninety minutes.

4.2.1. Resume evaluation task

The resume evaluation task was the first task participants completed during the Zoom session. Participants saw two sets of application materials for entry level corporate jobs, one from a Latina applicant (Gabriela Hernandez) and one from a White applicant (Mary Loftis). The sets were presented in counterbalanced order. In each set, participants saw a resume and listened to six brief audio recordings of answers to interview questions purportedly answered by the applicant. The Latina applicant was voiced by a native Spanish speaker currently living in Panama for whom English was her second language. According to past research on employment discrimination, a combination of ethnic name and accent is especially likely to produce bias (Purkiss et al., 2006). Although past research has typically used a between-subject design (i.e., participants evaluated just one resume), due to sample size considerations, we opted for a within-subject design. To address the potential confound of resume equivalency, we randomly assigned half of participants to one pairing of ethnicity and resume (i.e., Latina applicant with resume and recording version 1 and White applicant with resume and recording version 2) and the other half to the opposite pairing (i.e., Latina applicant with resume and recording version 2 and White applicant with resume and recording version 1).

After viewing the resume and listening to the recordings for each applicant, participants rated the applicant on perceived competency using a measure adapted from past research (Moss-Racusin et al., 2012). Participants' answers to 7 questions (e.g., "Did the applicant strike you as competent?"; α = 0.93-0.94) were averaged into a composite of perceived competency. Participants additionally provided an open-ended recommendation for the applicant's starting salary in response to the question "If you were to hire the applicant, what salary would you suggest to HR? The average entry level salary in Arkansas is $42,000." Resume pairing did not affect perceived competency or starting salary recommendation for either the Latina applicant ($p \geq 0.381$) or the White applicant ($p \geq 0.100$).

4.2.2. Self-Reported ethnocentrism

Participants completed self-report questionnaires toward the end of the Zoom session. Ethnocentrism was assessed using the first half of the Generalized Ethnocentrism Scale (Neuliep & McCroskey, 1997); the scale was shortened due to study time constraints. Participants rated their agreement with 12 statements (e.g., "Most other cultures are backward compared to my culture;" α = 0.74) on a scale of 1 (*Strongly Disagree*) to 7 (*Strongly Agree*).

4.3. Robustness analyses

To ensure that our results were robust to idiosyncratic researcher decisions, we conducted additional ancillary analyses with different configurations of the subsamples and covariates. In addition to the main models with our pre-registered exclusions, we examined whether the findings were robust to inclusion of participants who (a) saw the resumes but did not hear the audio, (b) had IL-1β values that were higher than three standard deviations above the sample mean, (c) self-identified as Latine, and (d) those who completed a similar study through another clinic (see Supplementary Materials for a description of this sample). In the Supplementary Materials, we also provide results for both a smaller subsample of participants who had provided saliva samples within more ideal time frames and a larger subsample of participants who had provided saliva samples within less ideal time frames.

Rather than focusing solely on whether an effect was significant with the varied analysis choices, we examined the magnitude of the effects (in terms of regression coefficients) and the precision of those estimated effects using Bayesian regression models. These analyses are presented in the Supplementary Materials and report posterior distribution of the regression coefficients for condition and cytokine change from a variety of models. Specifically, we examined (a) all three cytokines, (b) all three tiers of saliva sample time frames, (c) inclusion of covariates (e.g., time difference between saliva samples, vaccine season, age, gender, political orientation, socioeconomic status, sleep quality, BMI, whether they have been sick in the last week, whether they had caffeine), and (d) inclusion of participants from the similar study through another clinic. We also report model comparisons indicating the relative posterior weight given to models including or excluding each factor (i.e., experimental condition, cytokine change) and the interaction.

4.4. Transparency and openness

Between the text and the Supplementary Materials, we report how we determined our sample size, all data exclusions, all manipulations, all measures relevant to the current hypotheses, and we follow JARS (Appelbaum et al., 2018). Hypotheses for this study were pre-registered (https://osf.io/hbwuf). Data were analyzed in SPSS and R. All materials—including the data and data analysis code—are accessible on OSF (https://osf.io/e4kgq/).

5. Results

First, we examined whether participants in the vaccine condition demonstrated greater intergroup bias than participants who received the placebo injection. As can be seen in Table 1, condition did not affect participants' ratings of either applicant's competency, either applicant's recommended starting salary, or ethnocentrism. Thus, our hypothesis was not supported.

Next, we examined whether participants in the vaccine condition demonstrated greater cytokine change than participants who received the placebo injection. Controlling for the time difference between samples, participants in the vaccine condition demonstrated a greater change in IL-1β than those in the placebo condition, $b = 0.45$, SE=2.22, $t(92) = 2.03$, $p = 0.045$, 95 % CI [0.01, 0.88], semi-partial $r = 0.20$.[6] Notably, there was substantial within-condition variability in residualized IL-1β change (vaccine: −3.00 to 4.10; placebo: −3.13 to 2.21). See Fig. 1. The heterogeneity in cytokine responses to the vaccine is

[5] Although the manufacturer instructions recommend a 1:1 dilution factor, we used a 2:1 dilution factor to enable us to capture the low inflammation levels typical of relatively healthy young adults.

[6] The effect of condition on IL-1β change was marginal when including the participants ($n = 6$) who had cytokine values greater than 3SD above the mean, $b = 0.39$, $SE = 0.23$, $t(98) = 1.68$, $p = 0.096$, 95% CI [-0.07, 0.84], semi-partial $r = 0.16$. The effect of condition on IL-1β change was significant when including participants ($n = 12$) who self-identified as Latine, $b = 0.48$, $SE = 0.22$, $t(1\ 1\ 0) = 2.18$, $p = 0.032$, 95% CI [0.04, 0.91], semi-partial $r = 0.20$.

Table 1
Difference in Social Bias Between Participants in the Placebo and Vaccine Conditions.

Dependent Measure	Placebo *M* (*SD*)	Vaccine *M* (*SD*)	*t*	*df*	*p*	*d*
Latina Applicant						
Competency	7.25 (1.24)	7.12 (1.14)	0.51	93	0.610	0.11
Salary Recommendation	$43,820.51 ($4,358.13)	$42,168.09 ($5,613.97)	1.54	93	0.126	0.33
White Applicant						
Competency	6.92 (1.34)	6.96 (1.15)	−0.16	93	0.872	0.03
Salary Recommendation	$43,025.64 ($5,269.01)	$41,469.88 ($5,697.65)	1.35	93	0.180	0.28
Ethnocentrism	2.63 (0.69)	2.49 (0.65)	1.06	100	0.290	0.21

Note. Inclusion of participants who did not hear the audio for the resume evaluation task does not change these patterns of results with the exception that participants in the vaccine condition ($M = \$42{,}100.22$, $SD = \$5{,}493.97$, $n = 59$) suggested marginally lower starting salaries for the Latina applicant compared to those in the placebo condition ($M = \$43{,}804.88$, $SD = \$4{,}261.57$, $n = 41$), $t(98) = 1.67$, $p = 0.099$, $d = 0.34$.

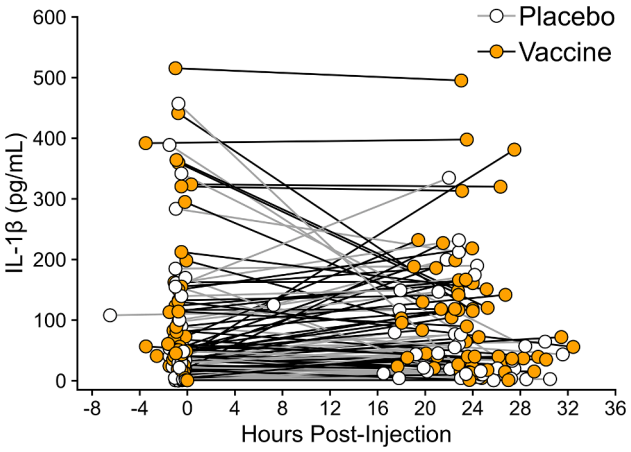

Fig. 1. Change in salivary IL-1β following the manipulation.

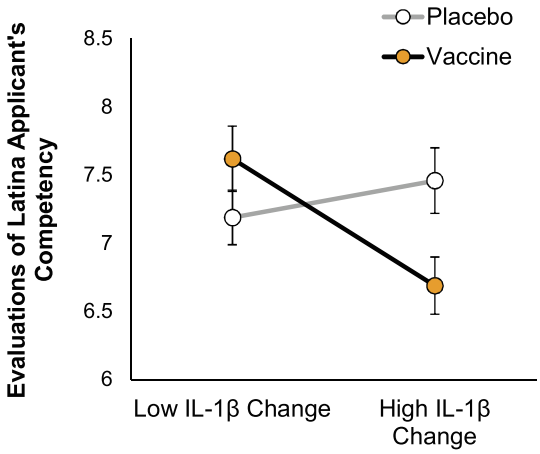

Fig. 2. Association between increases in IL-1β and evaluation of Latina applicant's competency. Note. IL-1β change is represented at ± 1SD. Error bars are SE.

consistent with results of past studies using the influenza vaccine challenge (Jolink et al., 2022; Kuhlman et al., 2018). Overall, 55 % of participants in the vaccine condition demonstrated an increase in IL-1β; the average change in IL-1β among participants in the vaccine condition was 7.24 pg/mL (*SD*=96.75) with an effect size of $d_{rm} = 0.19$.[7]

To test the critical hypothesis (i.e., that cytokine change would be associated with greater intergroup bias), we regressed evaluations of the Latina applicant's competency onto condition (−0.5 = placebo, 0.5 = vaccine), residualized IL-1β change (z-scored), and the condition × IL-1β change interaction to assess whether the association of competency evaluation with IL-β differed between participants who received the placebo or the vaccine. The models controlled for the evaluations of the White applicant's competency.[8] Although the main effect of IL-1β change was not significant, $b = -0.17$, *SE*=0.12, $t(85) = -1.45$, $p = 0.152$, 95 % CI [-0.39, 0.06], semi-partial $r = -0.13$, there was a significant IL-1β change × condition interaction, $b = -0.60$, *SE*=0.23, $t(85) = -2.64$, $p = 0.010$, 95 % CI [-1.06, −0.15], semi-partial $r = -0.23$. We probed the interaction by examining the simple effects of IL-1β change among participants who received the vaccine and among those who received the placebo (see Fig. 2). Among participants who received the influenza vaccine, IL-1β change was negatively associated with perceptions of the Latina applicant's competency, $b = -0.47$, *SE*=0.18, $t(85) = -2.66$, $p = 0.009$, 95 % CI [-0.82, −0.12], semi-partial $r = -0.23$. Conversely, among participants who received the placebo, there was no association between IL-1β change and evaluations of the Latina applicant's competency, $b = 0.14$, *SE*=0.15, $t(85) = 0.92$, $p = 0.359$. As can be seen in Table 2, this pattern of results was robust to inclusion of all additional participants. Overall, although we did not explicitly hypothesize in our preregistration that the association between cytokine change and bias would be moderated by condition, the results are consistent with our preregistered hypothesis that cytokine change would be associated with bias among participants who received the vaccine.

We next conducted a parallel set of analyses to examine participants' recommendation for the Latina applicant's starting salary. This time, the main effect of IL-1β change was significant: participants who experienced greater IL-1β change recommended lower starting salaries for the Latina applicant, $b = -836.56$, *SE*=405.55, $t(85) = -2.06$, $p = 0.042$, 95 % CI [-1642.89, −30.22], semi-partial $r = -0.14$. This main effect was, however, also qualified by a significant IL-1β change × condition interaction, $b = -1833.42$, *SE*=817.99, $t(85) = -2.24$, $p = 0.028$, 95 % CI [-3459.80, −207.03], semi-partial $r = -0.16$ (see Fig. 3). Among participants who received the influenza vaccine, IL-1β change was negatively associated with salary recommendation for the Latina applicant, $b = -1753.26$, *SE*=625.26, $t(85) = -2.80$, $p = 0.006$, 95 % CI [-2996.45, −510.08], semi-partial $r = -0.20$. Conversely, among participants who received the placebo, there was no association between IL-1β change and salary recommendation for the Latina applicant, $b = 80.15$, *SE*=522.05, $t(85) = 0.15$, $p = 0.878$. As can be seen in Table 2, this pattern of results was generally robust to inclusion of additional participants, with two exceptions. One possible explanation for the lack of an association between IL-1β change and starting salary recommendations in the model that included participants who completed a similar

[7] Following past research (Kuhlman et al., 2018), we also computed the effect size after windsorizing outliers that were 3*SD* above the mean. That effect size was d_{rm}=0.24.

[8] Inclusion of additional covariates (age, BMI, gender, socioeconomic status, quality of sleep, whether participants had caffeine, whether participants reported feeling ill within the last week, and whether participants reported having any of the conditions or medications we asked about during screening) did not change any of the reported results. See the Bayesian robustness analyses in the Supplemental materials for a more thorough investigation of covariate effects.

Table 2
Robustness Analyses for Evaluations of the Latina Applicant.

		Interaction			Simple Effect of Cytokine Change in Vaccine Condition		
Model	*df*	*t*	*p*	95 % CI	*t*	*p*	95 % CI
Competency Evaluation							
Including No Audio	89	**−2.79**	**0.007**	**[-0.99, −0.17]**	**−2.66**	**0.009**	**[-0.74, −0.11]**
Including Cytokine Outliers	95	**−2.61**	**0.010**	**[-0.90, −0.12]**	**−2.69**	**0.008**	**[-0.66, −0.10]**
Including Latine	106	**−2.27**	**0.025**	**[-0.79, −0.05]**	**−2.79**	**0.006**	**[-0.66, −0.11]**
Including Clinic B	137	–-	–-	–-	**−2.60**	**0.010**	**[-0.35, −0.05]**
Salary Recommendation							
Including No Audio	89	**−2.55**	**0.012**	**[-3505.29, −436.21]**	**−2.95**	**0.004**	**[-2933.83, −574.07]**
Including Cytokine Outliers	95	−1.94	0.055	[-2832.72, 29.53]	**−2.43**	**0.017**	**[-2288.79, −229.47]**
Including Latine	106	**−2.13**	**0.035**	**[-2765.84, 100.26]**	**−2.61**	**0.010**	**[-2254.62, −308.61]**
Including Clinic B	137	–-	–-	–-	−1.46	0.147	[-1056.44, 159.49]

Note. The sample of participants from Clinic B is described in the Supplementary Materials. Notably, all participants in that sample were students and all received the vaccine (i.e., no placebo control condition). The model for combined clinic analyses thus excluded Clinic A participants who received the placebo and only examines the main effect of IL-1β change among participants who received the vaccine at either clinic.

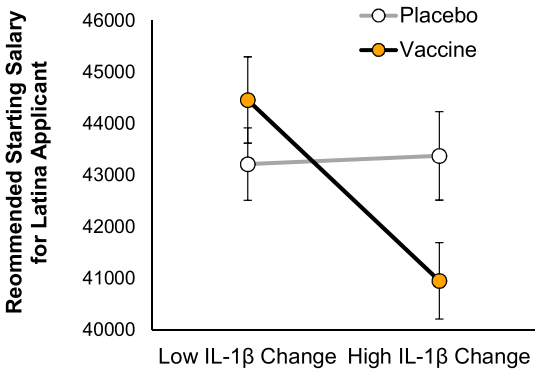

Fig. 3. Association between increases in IL-1β and starting salary recommendation for the Latina applicant. Note. IL-1β change is represented at ± 1SD. Error bars are SE.

study through Clinic B may be the fact that, although participants in our main sample were recruited from the community, participants in the Clinic B sample were all students.

We next examined the association between IL-1β change and evaluations of the White applicant. In this model, there was neither a main effect of IL-1β change, b = -0.02, SE=0.13, $t(85)$ = -0.14, p = 0.891, nor an IL-1β change × condition interaction, b = 0.23, SE=0.26, $t(85)$ = 0.90, p = 0.369. The same pattern was observed for the parallel model examining starting salary recommendations. There was neither a main effect of IL-1β change, b = 571.06, SE=459.36, $t(85)$ = 1.24, p = 0.217, nor an IL-1β change × condition interaction, b = 728.84, SE=935.59, $t(85)$ = 0.78, p = 0.438. The main effects and interactions were not significant across all robustness analyses. Overall, evaluations of the White applicant's competency and starting salary recommendations were not related to IL-1β change.

Finally, we examined whether cytokine change was associated with social bias using our second dependent measure of outgroup bias: self-reported ethnocentrism. There was a marginal, positive main effect of IL-1β change, b = 0.13, SE=0.07, $t(92)$ = 1.90, p = 0.061, 95 % CI [-0.01, 0.27], semi-partial r = 0.19, but the interaction between condition × IL-1β change was not significant, b = 0.15, SE=0.14, $t(92)$ = 1.05, p = 0.297. Notably, the main effect was significant in exploratory analyses controlling for political orientation, b = 0.16, SE=0.06, $t(91)$ = 2.48, p = 0.015, 95 % CI [0.03, 0.28], semi-partial r = 0.23. The pattern of results was the same in the subsequent models included participants who had IL-1β levels greater than three standard deviations above the sample mean. In the models that included participants who identified their ethnicity as Latine and who completed a similar study through Clinic B, the main effect was significant with and without political orientation as a covariate. Thus, the overall pattern of results suggests that participants who demonstrated greater (vs. lower) IL-1β change reported higher levels of ethnocentrism.

Additional robustness analyses are reported in detail in the Supplementary Materials. In the subsample of participants who provided saliva samples in the narrowest time frames, we found the same consistent negative association between IL-1β change and evaluation of the Latina applicant's competency and recommendations for the Latina applicant's starting salary among participants who received the vaccine. The effects were in the same direction but weakened in the subsample with the widest time frames. The positive association between IL-1β change and ethnocentrism, on the other hand, continued to become stronger with larger sample sizes. Furthermore, we also found preliminary evidence that IL-1β change statistically mediated the effect of condition (vaccine or placebo) on ethnocentrism and that the association between IL-1β change and ethnocentrism emerged primarily among participants who would typically have lower levels of prejudice (e.g., those with higher internal motivation to respond without prejudice).

We also conducted extensive Bayesian analyses that examined (a) all three cytokines, (b) all three tiers of saliva sample time frames, (c) inclusion of covariates, and (d) inclusion of participants from the similar study through another clinic. On the whole, these analyses suggested that there is an association between cytokine change and bias. Furthermore, across all analyses, to the extent that any link between cytokine change and bias behavior was detectable in the data, increased cytokine change was associated with increased bias. The results were less supportive of an interaction between receiving the flu shot and the cytokine change except for, potentially, the relationship between IL-1β change and the recommended starting salary for the Latina applicant. Overall, we predict that with a larger sample size and with better experimental control (i.e., sample timing relative to the manipulation and measure completion), the association between biased behavior and cytokine change, particularly IL-1β, would be even better supported.

6. Discussion

In this study, we tested the hypothesis that acute inflammatory activity—as measured by vaccine-induced increases in pro-inflammatory cytokines—would be associated with greater intergroup bias. Results showed that, among participants who received the vaccine, IL-1β change was negatively associated with perceptions of the Latina applicant's competency and starting salary recommendations. The findings for ethnocentrism were overall less robust, but there was a generally consistent main effect of cytokine change, such that greater cytokine change was associated with more ethnocentrism. Overall, these results provide the first direct evidence that greater immune activation following experimental inductions of immune activity may contribute to intergroup bias.

Our results add to the growing body of work in psychoneuroimmunology suggesting that immune system activity may influence social

behavior. Generally, those influences are nuanced, context dependent, and vary based on the specific relationship between the actor and target (s). For example, increases in proinflammatory cytokines have been linked to a greater desire to approach close others (Jolink et al., 2022; Muscatell et al., 2016), but the same increases have also been linked to a greater desire to avoid strangers (Jolink et al., 2022) as well as greater attention to strangers' negative emotional expressions (Inagaki et al., 2012). In support of the idea that cytokine increases may confer a bias against outgroup individuals, computational modeling of the spread of viral infections has suggested that people may selectively withdraw from those on the periphery of their social networks (Cole, 2006), and those on that periphery are likely to be outgroup members (Allport, 1954). Our results thus support and extend this work by showing that the cytokine-linked tendency to avoid distant social partners found in prior work may extend to intergroup bias and discrimination.

Our findings also extend research in social psychology by elucidating novel situational factors that affect intergroup bias—acute inflammation and illness. Past research has emphasized that situational factors affect intergroup bias, including factors such as when people are experiencing arousing emotions (Rogers & Prentice-Dunn, 1981; Wilder, 1993), feel threatened (Fein & Spencer, 1997), are under time pressure (Kruglanski & Freund, 1983), or are under cognitive load (Gilbert & Hixon, 1991), as well as during the time of day opposite of their chronotypes (Bodenhausen, 1990). The present research suggests that times when people experience illness and inflammation may also affect intergroup bias, which—due to the commonality of illnesses at every level of society, as well as the many contexts and events that also promote elevated inflammation—may have important social, economic, and political implications.

Given that our results provide preliminary support for a mechanistic role of acute increases in pro-inflammatory cytokines in intergroup bias, other situations characterized by acute increases in pro-inflammatory cytokines may also be characterized by intergroup bias. Indeed, some situational factors that have been shown to increase intergroup bias are also characterized by increases in pro-inflammatory cytokines. For example, exercise is associated with both increases in intergroup bias (Kim & Baron, 1988; Wann & Branscombe, 1995) and increases in cytokines (Cerqueira et al., 2020). Likewise, acute stress is associated with both increases in intergroup bias (Keinan et al., 2000) and increases in cytokines (Szabo et al., 2020). These associations may be fruitful to examine further, especially with an emphasis on the mechanistic role that cytokines may play in regulating social decision-making during these states. Additionally, examining inflammation-induced intergroup bias within exercise and acute stress paradigms may prove especially fruitful towards determining the sociocognitive mechanisms through which elevated cytokines result in intergroup bias, as exercise, vaccination, and acute stress each elevate cytokines, albeit through different biological pathways.

The current research examined but one process within the immune system—acute inflammatory activity associated with the general response of the innate immune sytem. If that process is linked to greater intergroup bias, there may be other processes within the immune system that are worth examining in the social domain. For example, chronic inflammation, which is characteristic of those with chronic illness or who have experienced early life adversity, may also affect social decision-making. Indeed, some research has demonstrated that people with rheumatoid arthritis, a chronic health condition that is characterized by high inflammation and increased vulnerability to other illnesses, demonstrated greater intergroup bias than people in the matched healthy control group (Oaten et al., 2017). It is likely that chronic inflammation characteristic of chronic stress also predicts elevated intergroup bias, although extant research is limited. A more thorough understanding of the biological and disease states which may result in heightened intergroup bias could help at-risk individuals to monitor their social decision making, avoiding or correcting biased decision making through cognitive attention.

Although our findings demonstrate that acute inflammation is associated with intergroup bias, we cannot speak to the sociocognitive mechanism that would explain why inflammation results in increased bias. As described above, we drew on three separate literatures when proposing our hypothesis, and each literature puts forth a unique, plausible mediator: increased reliance on social heuristics, greater ingroup favoritism and outgroup derogation, and greater pathogen avoidance motives. Future work should directly test which of these mediators, if any, explain the associations between inflammation and intergroup bias and if these differ between different inflammatory contexts (i.e., working while sick, exercise, chronic stress).

Additionally, although we did not expect to observe an association with IL-1β alone, we note that we are not the first to observe such selectivity. In particular, prior studies have found selective associations between endogenous IL-1β and measures of executive function (Serre-Miranda et al., 2020), global cognition (Jin et al., 2020), and social cognition (Baek et al., 2022; see also Turner et al., 2021). Further, IL-1β has been found to mediate vaccine-related cognitive effects in rodents (Vanderheiden et al., 2024). IL-1β is a member of a different cytokine family than either IL-6 or TNF-α (Palomo et al., 2015), and our findings, coupled with prior literature, could be taken to suggest that the IL-1 cytokine family may be particularly relevant in links between inflammatory activity and cognitive functioning.

Notably, ours is the first study to examine change in salivary (vs. plasma) cytokines in response to the influenza vaccine challenge, as well as to compare participants who received the influenza vaccine to a control condition who received a saline placebo injection. IL-1β change was greater among participants who received the vaccine compared to those who received the saline placebo injection. Compared to other studies that used the influenza vaccine challenge (Kuhlman et al., 2018; Jolink et al., 2022), fewer of our participants demonstrated an increase in cytokines (55 % in our sample vs. 80 % in past research) and the magnitude of cytokine increase was about half (our sample: $d_{rm} = 0.24$; Kuhlman et al. (2018): $d_{rm} = 0.45$). However, a direct comparison in the magnitude of response cannot be made due to the methodological differences between the studies. We focused on changes in salivary IL-1β and had to compromise on experimental control due to the pandemic necessitating remote data collection, whereas the previous studies focused on changes in plasma IL-6 and had greater experimental control due to in person sessions. It is additionally worth noting that the salivary IL-1β response to the influenza vaccine challenge observed in our study is similar in magnitude to the salivary IL-1β response to acute stress (Szabo et al., 2020).

Future research would also benefit from a more comprehensive understanding of the neural mechanisms that underpin associations between cytokine activity and intergroup bias. Cytokines are known to influence neural activity in brain regions that support self-regulatory control processes (e.g., the dorsolateral prefrontal cortex; Harrison et al., 2009) or that support dopaminergic activity (e.g., the substantia nigra; Brydon et al., 2008). Cytokines influence neural activity via multiple pathways, and they can do so directly via binding to their receptors on neurons (Friedman, 2001; Shields et al., 2017). Developing such a multi-level understanding of these dynamics may generate additional avenues for integrating psychoneuroimmunology and social psychology.

Limitations and Future Directions

This study has several strengths, including a placebo-controlled induction of immune system activity, within-study conceptual replication, and a large sample size for a study inducing inflammation using a vaccine challenge paradigm. Nonetheless, this study has limitations that should be noted. Several limitations relate to data collection during the pandemic. First, due to pandemic-related recruitment issues, we were unable to recruit our target sample size, and we further had to exclude many participants because they failed to follow instructions during the remote protocol. Nevertheless, as demonstrated in our ancillary analyses, there was a robust and consistent pattern of associations between

change in IL-1β and our intergroup bias measures.

Second, we assessed salivary cytokines, which can be viewed as another limitation; past research using the influenza vaccine to upregulate cytokines has assessed those in blood (Jolink et al., 2022; Kuhlman et al., 2018). Although other research has demonstrated that IL-6 responses to stress, for example, are similar when assessed in saliva or blood (La Fratta et al., 2018), cytokine responses in blood versus saliva have not been compared following influenza vaccination. Salivary cytokines represent the conjunction of systemic and local immune system activity (Shields et al., 2019a,b; Szabo & Slavish, 2021), and it is likely that nuisance variance related to local immune activity weakened associations between systemic immune activity and intergroup bias that could have been observed had we measured cytokines from blood. It would behoove future research to comprehensively examine cytokine responses to the influenza vaccine challenge, including the administration of a saline placebo for the control group.

Third, all PNI research must strike a balance between experimental control and generalizability of findings to populations who are not entirely free from disease. Although we restricted participation to those free from immunodeficiency-related disease (e.g., HIV, asthma), diseases associated with systemic inflammation or abnormal inflammatory marker levels (e.g., autoimmune disorders, inflammatory bowel disease), and daily use of medications which act through inflammatory pathways (e.g., SSRIs, aspirin; see Supplementary Materials for full list of exclusion criteria), we did not account for all conditions or circumstances which are associated with altered inflammatory profiles. Future research should assess how early-life stressor exposure and depression, for example, influence associations between inflammation and intergroup bias.

Fourth, our sample lacked diversity, which could affect the generalizability of the findings. Indeed, our sample was largely non-Latine White, and it is possible that the biological bases or determinants of intergroup bias differ between minority and nonminority individuals. Moreover, our Clinic B participants were all students, and students may differ from community participants in how they approach answers to salary questions. Finally, our sample was western, educated, industrialized, rich, and democratic (WEIRD), and many psychological processes differ between WEIRD and non-WEIRD societies (Henrich et al., 2010). Future work should attempt to determine whether the relations that we observed between immune system activity and intergroup bias replicate across cultures.

7. Conclusion

Remember Alex, the hiring manager who sorted through applicants' resumes while sick? The present findings suggest that Alex would have been likely to evaluate those applicants differently than usual because of illness-induced inflammation—especially if any of the applicants happened to be from racial or ethnic minoritized groups. In the present research, we found vaccine-induced inflammation to lead to increased intergroup bias, which beyond extending to social decision-making during illness-induced inflammation, could also extend to social decision-making during other times of elevated inflammation, such as chronic illness, acute stress, or even after intense exercise. Given the lack of federal sick leave policies in the United States, it is important to continue investigating the effects working while sick may have on hiring and promotion decisions; the lack of such sick leave policies may be hampering diversity, equity, and inclusion.

CRediT authorship contribution statement

Anastasia Makhanova: Writing – original draft, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Mikayla D.M. Tolliver:** Writing – review & editing, Project administration. **Zach Buckner:** Writing – review & editing, Project administration. **Grant S. Shields:** Writing – review & editing, Project administration, Data curation. **Colton L. Hunter:** Writing – review & editing, Project administration. **Summer Mengelkoch:** Writing – review & editing, Methodology, Data curation. **Joseph W. Houpt:** Writing – review & editing, Formal analysis. **Alex E. Belote:** Writing – review & editing, Methodology. **Dalton V. Hoose:** Writing – review & editing, Methodology. **Thomas K. Schulz:** Writing – review & editing, Supervision, Methodology.

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.

Data availability

Data and materials are available on OSF (https://osf.io/e4kgq/).

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbi.2024.08.039.

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