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# Emodiversity and Biomarkers of Inflammation

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There is growing evidence that inflammatory responses may help to explain how emotions get "under the skin" to influence disease susceptibility. Moving beyond examination of individuals' average level of emotion, this study examined how the breadth and relative abundance of emotions that individuals experience— *emodiversity*—is related to systemic inflammation. Using diary data from 175 adults aged 40 to 65 who provided end-of-day reports of their positive and negative emotions over 30 days, we found that greater diversity in day-to-day positive emotions was associated with lower circulating levels of inflammation (indicated by IL-6, CRP, fibrinogen), independent of mean levels of positive and negative emotions, body mass index, anti-inflammatory medications, medical conditions, personality, and demographics. No significant associations were observed between global or negative emodiversity and inflammation. These findings highlight the unique role daily positive emotions play in biological health.

*Keywords:* emodiversity, intraindividual variability, positive emotions, entropy, health



> We differ in nothing more than in our capacity to feel . . . upon that degree the dignity and significance of each life depend (Hamilton, 1942, pp. 145–146).[1]

There is tremendous variety in the emotional states that constitute everyday life. Some people have emotional experiences that are differentiated, while others experience emotions in a global manner. In their influential work on mood variability, Wessman and Ricks (1966) coined the term "affective complexity" to characterize differences in the richness of emotional life. While conceptualizations and operationalizations of emotional complexity have differed across studies, an emerging literature suggests that indices of complexity may be broadly categorized according to the extent of *covariation* or *differentiation* in self-reported experiences of emotion (Grühn, Lumley, Diehl, & Labouvie-Vief, 2013; Hay & Diehl, 2011; Lindquist & Barrett, 2008).

Measures of *emotional covariation* typically assess individual differences in the extent of co-occurrence (i.e., mixed emotions) or correlation (i.e., emotional dialecticism) of positive and negative affect over time (Grossmann, Huynh, & Ellsworth, 2016; Larsen & McGraw, 2014; Ready, Carvalho, & Weinberger, 2008). Both greater dialectical and more mixed emotional experience are associated with higher well-being and greater resilience (Adler & Hershfield, 2012; Coifman, Bonanno, & Rafaeli, 2007; Hershfield, Scheibe, Sims, & Carstensen, 2013), particularly among East Asians (Miyamoto & Ryff, 2011; Miyamoto, Uchida, & Ellsworth, 2010) and older adults (Carstensen, Pasupathi, Mayr, & Nesselroade, 2000; Carstensen et al., 2011; Ong & Bergeman, 2004), with some evidence that these associations may be moderated by differences in ideal affect and interdependent self-construals (Grossmann et al., 2016; Sims et al., 2015), the amount of intraindividual variability in positive and negative emotional states (Brose, de Roover, Ceulemans, & Kuppens, 2015; Grühn et al., 2013), and cognitive ability (Hülür, Hoppmann, Ram, & Gerstorf, 2015).

Measures of *emotional differentiation* (also referred to as emotional granularity; Barrett, 2006; Lindquist & Barrett, 2008) assess individual differences in the propensity to categorize and label emotional experiences in discrete terms. Theoretically, individuals with more differentiated emotional experiences have greater ability to make subtle distinctions among the emotional states they experience (e.g., fear, sadness, anger; Barrett, Gross, Christensen,

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[1] As cited in Wessman and Ricks (1966, p. 251).

& Benvenuto, 2001). Between-person differences in emotional differentiation generated from diary and ecological momentary assessment data show that undifferentiated emotion (particularly of negative emotions) is associated with a range of psychopathologies, including borderline personality, social anxiety, and major depressive disorder (Demiralp et al., 2012; Kashdan & Farmer, 2014; Tomko et al., 2015). Other research has similarly established an association between greater differentiation in positive emotions and adaptive coping and adjustment (e.g., Tugade, Fredrickson, & Feldman-Barrett, 2004). To date, however, little is known about how—that is, through what the biological processes—complex emotional experiences influence health outcomes. The current study examines the association between emodiversity—the breadth and relative abundance of different emotions that individuals experience—and biological inflammation.

## Emotion and Inflammatory Processes

Inflammation is a key risk factor for early morbidity and mortality, and growing evidence links emotional processes with systemic inflammation. Across clinical and population-based samples, heightened systemic inflammation has been shown to contribute to poor health (e.g., atherosclerosis, Type II diabetes, rheumatoid disease, osteoporosis) and to elicit a number of pathogenic processes (e.g., oxidative stress, insulin resistance, plaque rupture, endothelial pathology) that play a major role in the risk of premature mortality (Cesari et al., 2003; Epel & Lithgow, 2014; Miller, Chen, & Parker, 2011; Schneiderman, Ironson, & Siegel, 2005). Evidence from human laboratory research suggests that negative emotional states stimulate inflammatory responses (Duivis et al., 2011; Howren, Lamkin, & Suls, 2009; Miller & Blackwell, 2006). For example, avoidance-oriented negative emotions, such as fear and shame, have been linked to greater inflammatory activity (Dickerson, Kemeny, Aziz, Kim, & Fahey, 2004; Moons, Eisenberger, & Taylor, 2010). Similarly, the onset and progression of particular negative moods and traits (e.g., depression, hostility, and anxiety) are often followed by elevated levels of inflammatory proteins, including the proinflammatory cytokine interleukin-6 (IL-6), the acute phase C-reactive protein (CRP), and the clotting factor fibrinogen (Ai, Kronfol, Seymour, & Bolling, 2005; Duivis et al., 2011; Miller, Rohleder, Stetler, & Kirschbaum, 2005; Moons & Shields, 2015; Pitsavos et al., 2006; Suarez, 2003).

Although the bulk of studies on affect and inflammation have focused on negative affect, there is growing evidence that positive affect has independent associations with inflammatory markers. In naturalistic studies of healthy adults, trait positive affect, but not negative affect, has been linked to lower levels of CRP and IL-6 (Deverts et al., 2010; Stellar et al., 2015; Steptoe, O'Donnell, Badrick, Kumari, & Marmot, 2008). Similarly, evidence from laboratory viral challenge studies suggests that higher levels of trait positive affect are associated with lower production of proinflammatory cytokines (Cohen, Alper, Doyle, Treanor, & Turner, 2006; Janicki-Deverts, Cohen, Doyle, Turner, & Treanor, 2007; Prather, Marsland, Muldoon, & Manuck, 2007; Robles, Brooks, & Pressman, 2009). Finally, there is evidence from clinical populations that positive affect influences immune processes. For example, data from cancer patients undergoing radiation therapy suggests that positive affect enhances acute inflammatory responses to treatment (Blomberg et al., 2009; Sepah & Bower, 2009) and prospectively predicts lower levels of CRP at treatment completion and through 6- and 12-month follow-ups (Moreno, Moskowitz, Ganz, & Bower, 2016). Taken together, experiences of negative and positive emotion in both trait and state form appear to influence the adaptive regulation of core biological systems that maintain health.

## Emodiversity and Health

Expanding beyond differences in level of negative and positive emotion, we consider how emodiversity—the relative breadth and abundance of different emotions (Benson, Ram, Almeida, Zautra, & Ong, in press; Quoidbach et al., 2014)—may influence inflammation. Drawing on analytic approaches used to quantify the biodiversity of ecosystems (Morin, 1999), measures of diversity have been used to assess a variety of social and psychological phenomena, including racial/ethnic diversity (Budescu & Budescu, 2012), behavioral flexibility (Ram, Conroy, Pincus, Hyde, & Molloy, 2012), population genetics (Sherwin, 2010), community social networks (Li, Zhang, Feng, & Wu, 2015), daily stressor diversity (Koffer, Ram, Conroy, Pincus, & Almeida, 2016), and activity diversity (Lee et al., 2016).

Although to date no studies have directly investigated the link between emodiversity and inflammation, there are reasons to suspect having a rich and diverse emotional life may be beneficial to health. First, emotional experiences that are broad in range and differentiated may guide adaptation by prioritizing, organizing, and regulating behavior in ways that optimize an individual's adjustment to situational demands (Barrett & Campos, 1987; Keltner & Gross, 1999). Additionally, representing emotions in discrete terms may have greater "informational value" than global affective states (Barrett, 1998; Barrett, Mesquita, Ochsner, & Gross, 2007). That is, the ability to characterize affective information with precision (i.e., in terms of qualitatively distinct events) may reduce the potential for individuals to make misattributions about their own affective reactions (Schwarz, 1990; Schwarz & Clore, 1983). Finally, experiencing a diversity of emotional states might reduce vulnerability to affective psychopathology by preventing an overabundance or prolonging of any one emotion from dominating an individuals' emotional life (Benson et al., in press; Gruber & Bekoff, 2017). Supporting this logic, Quoidbach et al. (2014) found that greater emodiversity was associated with better mental and physical health.

Individual differences in emodiversity are illustrated in Figure 1. The figure depicts two individuals who have identical mean levels of positive and negative emotion but differ in diversity of day-to-day emotional experiences. For conceptual display, emotions are ordered along the *x*-axis in accordance with a circumplex perspective (Russell, 1980), wherein emotions range from high arousal positive to low arousal positive (e.g., enthusiastic to calm), and from high arousal negative to low arousal negative (e.g., nervous to sad). Positive valence emotions are depicted in pink and negative emotions in green, with the darker hues corresponding to higher arousal emotions and lighter hues to lower arousal emotions. The height of each bar indicates the number of occasions on which each emotion was experienced. Person A's (left panel) emotions are relatively low in diversity in that they are concentrated in a few emotion categories. In contrast, Person B's (right panel) emotions are relatively high in diversity in that they are

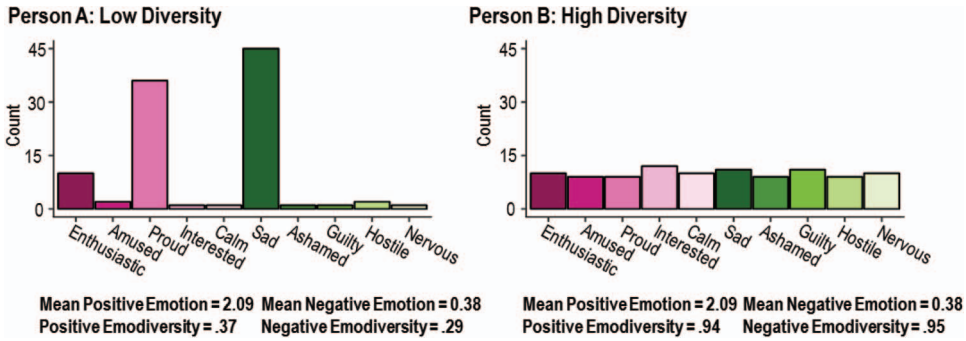


*Figure 1.* Individual differences in *emodiversity*—the breadth (the number of discrete emotions experienced) and evenness (the distribution of experiences across discrete emotions) of emotional experience. Left panel: Person A has low emodiversity, with emotion experiences that are relatively homogenous and concentrated in a few emotion categories. Right panel: Person B has high emodiversity, with emotion experiences that are relatively diverse and distributed more evenly across categories. See the online article for the color version of this figure.



distributed more evenly across categories. Importantly, these differences are distinct from mean levels of emotion. Our interest here is whether individual differences in diversity of emotion (emodiversity) may be associated with systemic inflammation.

## The Present Investigation

The current investigation sought to examine the associations between emodiversity and systemic inflammation in a community-based sample of middle-aged adults. Given prior work suggesting that greater diversity in positive and negative emotions is associated with better health (Quoidbach et al., 2014), we hypothesized that greater global emodiversity would be associated with decreased circulating levels of inflammatory markers (IL-6, CRP, fibrinogen). Furthermore, given previously documented associations between differentiated positive and negative emotions and adjustment (Barrett et al., 2001; Tugade et al., 2004), and evidence that positive and negative affect independently predict inflammation (Stellar et al., 2015; Steptoe et al., 2008), we tested the hypothesis that positive and negative emodiversity contribute uniquely to inflammation.

Our analyses were designed to extend conceptual understanding of emodiversity in four important ways. First, we consider within-person variation in emotions using time-intensive study designs that minimize retrospection bias and allow researchers to simultaneously account for within- and between-person sources of variation in data (cf. Ram & Gerstorf, 2009; West & Hepworth, 1991). This approach is in line with recent demonstrations that the intensive study of individuals over time enables researchers to move from static to more dynamic conceptual and methodological frameworks that observe peoples' emotional lives as they unfold day to day (Ram et al., 2012; Zautra, Affleck, Tennen, Reich, & Davis, 2005). Second, building on prior cross-sectional work examining links between emodiversity and mental and physical health (Quoidbach et al., 2014), the present study investigated how diversity in day-to-day emotions is related to inflammation. Third, to account for overlap in the putative measures of inflammation (Friedman & Herd, 2010), we fit structural equation models in which IL-6, CRP, and fibrinogen scores were used as indicators of a latent inflammation construct. Fourth we tested whether emodiversity was associated with inflammation above and beyond mean levels of emotion (Gruber, Kogan, Quoidbach, & Mauss, 2013). Finally, drawing on functionalist and core affect theories of emotion (Keltner & Gross, 1999; Russell, 1980; Shiota et al., in press; Shiota et al., 2014) and following prior research on emodiversity (Quoidbach et al., 2014), we explored differential effects of positive and negative emodiversity, as well as global emodiversity across positive and negative emotions.

## Method

### Participants

Data were drawn from a larger study of community-dwelling adults (40–65 years, $N = 688$) conducted in the Phoenix, Arizona metropolitan area between 2007 and 2012 (Sturgeon et al., 2016). The analytic sample for the current study consisted of 175 participants (46% male), age 40 to 65 ($M = 53.42$, $SD = 7.57$), who provided a minimum of six of 30 daily diary records and completed a 6-month follow-up interview. The median household income in the current study was between $50,000 and $65,000 per year. Participants self-identified as White (67%), Hispanic/Latino (8%), African American (3%), Asian (2%), and Native American or American Indian (1%), with 19% identifying with more than one ethnic group.

### Procedure

After providing informed consent, participants completed a demographic questionnaire and training session where they were introduced to the study procedures and instructed on how to use a study-provided tablet computer. Participants used the tablet computer to complete daily diaries each night for 30 days. Participants underwent a blood draw to assess levels of IL-6 (pg/ml), CRP (mg/L), and fibrinogen (mg/dL). Blood samples were drawn by a research phlebotomist during a 6-month follow-up visit to participants' homes (samples obtained between 7:30 a.m. and 8:00 p.m., with participants asked to fast for at least 8 hr). All procedures

were approved by the Institutional Review Board at Arizona State University.

## Measures and Materials

**Daily emotion reports.** Daily emotions were assessed as part of the daily tablet computer-based questionnaires using 32 items from the Positive Affect-Negative Affect Schedule (PANAS and PANAS-X; Watson, Clark, & Tellegen, 1988). At the end of each day, participants rated the extent to which they had experienced 16 positive valence emotions (enthusiastic, interested, determined, excited, amused, inspired, alert, active, strong, proud, attentive, happy, relaxed, cheerful, at ease, calm) and 16 negative valence emotions (scared, afraid, upset, distressed, jittery, nervous, ashamed, guilty, irritable, hostile, tired, sluggish, sleepy, blue, sad, drowsy) on a 1 = *very slightly or not at all* to 5 = *extremely* Likert-type rating scale. Daily emotion reports were summarized with respect to mean level and diversity of emotion.

**Mean emotion.** Mean positive and negative emotion scores were calculated using the continuous Likert scale ratings (0–4). Within each occasion (i.e., day), positive and negative emotion items were averaged separately, and then used to calculate an across-day average for each individual.

**Emodiversity.** Individual differences in the diversity of emotions were quantified in terms of *emodiversity*. Specifically, after recoding into a binary variable that indicated the absence or presence of each emotion on a given day, scores for *global emodiversity* ($m = 32$ items), *positive emodiversity* ($m = 16$), and *negative emodiversity* ($m = 16$) were each indexed using the Gini (1921) coefficient,

$$GiniDiversity_i = G_i = 1 - \left( \left( \frac{2\sum_{j=1}^{m} jc_{ij}}{m\sum_{j=1}^{m} c_{ij}} \right) - \frac{m+1}{m} \right)$$

where $c_{ij}$ is the count of individual $i$'s emotion experiences within $j = 1$ to $m$ emotion types, indexed in ascending order ($c_{ij} \leq c_{ij+1}$) for each participant. Using this index, scores for global, positive, and negative emodiversity can each range from 0 to 1, with higher values indicating more diversity, and in particular, evenness across the $j$ emotion types. To illustrate the calculation, the vector of observed counts for Person A's positive emotions in Figure 1 is (10, 2, 36, 1, 1). Ordered (proud, enthusiastic, amused, interest, calm) and weighted by relative order (1*1, 2*1, 3*2, 4*10, 5*36), the Gini coefficient for this individual $G_A = 1 - \left( \left( \frac{2\sum_{j=1}^{m} jc_{ij}}{5\sum_{j=1}^{m} c_{ij}} \right) - \frac{5+1}{5} \right) = 1 - .63 = .37$ . In contrast, the vector of observed counts for Person B is (10, 9, 9, 12, 10). Ordered (interest, enthusiastic, calm, amused, proud) and weighted by relative order (1*9, 2*9, 3*10, 4*10, 5*12), the Gini coefficient for this individual $G_B = 1 - \left( \left( \frac{2\sum_{j=1}^{m} jc_{ij}}{5\sum_{j=1}^{m} c_{ij}} \right) - \frac{5+1}{5} \right) = 1 - .06 = .94$ . The differences in Gini diversity thus quantify the relative unevenness/evenness of the heights of the bars evident in the visual representations. This emphasis on differences in evenness is useful in study designs like the current one, where a fixed-length list of emotion items are presented at all occasions (for a discussion, see Benson et al., in press; Brown & Coyne, in press). Of note, Gini diversity can also be calculated using counts weighted by the original 1 to 5 Likert scale by recoding values to be on a 0 to 4 Likert scale so that a true zero point is present. In these data, the pattern of results reported below for the binary counts is substantively the same as results obtained with Likert-weighted counts.

**Inflammation.** To quantify levels of IL-6 and CRP, 10 ml of blood was collected into EDTA tubes (Becton-Dickinson, Franklin Lakes, NJ), held on ice, and centrifuged within 2 hr of collection for 15 min at 1,500 g. Plasma was then aspirated, aliquoted, and frozen at −80°C until assay. Plasma levels of IL-6 were quantified using Quantikine High Sensitivity human IL-6 kits (R&D Systems, Inc, Minneapolis, MN), an enzyme-linked immunosorbent assay (33) with an intraassay coefficient of variation of 4% and interassay coefficient of variation of 10%. The minimal detectable level of IL-6 was 0.156 pg/ml. CRP was measured using the Dade Behring *N* High Sensitivity CRP turbidimetric immunoassay (Dade Behring Diagnostics, Marburg, Germany) on the BN ProSpec. Fibrinogen levels (mg/dL) were determined by a commercial laboratory (Quest Diagnostics, Los Angeles, CA) through use of a clotting assay. Data from eight participants with IL-6 values greater than 10 pg/ml and CRP values greater than 10 mg/L, suggesting the presence of acute illness, were excluded (McCaffery, Marsland, Strohacker, Muldoon, & Manuck, 2012).

**Covariates.** Body mass index (BMI), anti-inflammatory and steroid medication use, medical conditions, personality facets of neuroticism and extraversion, and demographics including age and gender were used as covariates. Medication use was coded using separate binary variables representing use of at least one anti-inflammatory medication or at least one steroid medication versus those who did not use any of these medications. A full list of the anti-inflammatory and steroid medications assessed in the current study can be found in an online appendix (Supplemental Digital Content 1, http://links.lww.com/PSYMED/A259). History of medical conditions included a series of yes/no questions pertaining to any occurrence of hypertension or high blood pressure, angina pectoris or coronary artery disease, congestive heart failure, myocardial infarction or heart attack, other heart conditions, stroke, emphysema or asthma or chronic obstructive pulmonary disease (COPD), arthritis of the hip or knee, arthritis of the hand or wrist, sciatica, diabetes or high blood sugar or sugar in the urine, and cancer (other than skin cancer). An overall medical conditions score was calculated for each participant as none, one, two, or three or more of the above (Petrov, Davis, Belyea, & Zautra, 2016). Personality facets of neuroticism and extraversion were assessed using 16 items from the Big 5 Inventory (John & Srivastava, 1990), with composite scores calculated as the sum of eight items for each facet.

## Data Analysis

A series of structural equation models were used to examine relations between emodiversity (global, positive, negative) and inflammation (latent variable indicated by IL-6, CRP, and fibrinogen). Four models were fit to the data. In Model 1, the latent inflammation factor was constructed and regressed on global emodiversity. In Model 2, age, gender, anti-inflammatory medications, BMI, medical conditions, and personality were added as covariates. In Model 3, the global diversity predictor was replaced by the positive emodiversity and negative emodiversity variables. In Model 4, mean positive emotion and mean negative emotion variables were added as covariates. Models were estimated using

Table 1
*Descriptives and Correlations Among Emotion, Inflammation, and Demographic Variables*

| Construct | Min | Max | Mean | *SD* | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Global emodiversity | .27 | .93 | .61 | .10 | — | | | | | | | | | | | | | |
| 2. Positive emodiversity | .39 | 1.00 | .92 | .11 | **.36** | — | | | | | | | | | | | | |
| 3. Negative emodiversity | .12 | .95 | .49 | .18 | **.79** | .02 | — | | | | | | | | | | | |
| 4. Mean positive emotion | .20 | 3.96 | 2.09 | .77 | −.03 | **.72** | **−.23** | — | | | | | | | | | | |
| 5. Mean negative emotion | .01 | 2.51 | .38 | .38 | **.66** | **−.23** | **.64** | **−.36** | — | | | | | | | | | |
| 6. CRP* | .10 | 75.00 | 3.59 | 7.82 | −.09 | **−.30** | .02 | −.13 | .01 | — | | | | | | | | |
| 7. IL-6* | .23 | 72.64 | 2.56 | 6.08 | −.10 | −.11 | −.12 | −.03 | −.01 | **.51** | — | | | | | | | |
| 8. Fibrinogen* | 195.00 | 712.00 | 333.05 | 75.52 | **−.15** | **−.19** | −.10 | −.10 | −.01 | **.52** | **.33** | — | | | | | | |
| 9. BMI | 16.38 | 65.16 | 27.76 | 6.52 | .07 | **−.16** | .14 | −.09 | **.15** | **.42** | **.29** | **.25** | — | | | | | |
| 10. Anti-inflammatory medication | No = 0 | Yes = 1 | 29%Y | — | .04 | .01 | .03 | −.04 | .10 | .05 | −.03 | .02 | **.18** | — | | | | |
| 11. Medical conditions | 0 | 3+ | 1.39 | 1.12 | .03 | −.08 | .00 | −.09 | **.17** | **.16** | .19 | .12 | **.17** | .33 | — | | | |
| 12. Neuroticism | 7 | 38 | 21.72 | 6.21 | **.31** | **−.17** | **.31** | **−.26** | **.29** | .02 | .01 | −.05 | **.15** | .06 | **.20** | — | | |
| 13. Extraversion | 8 | 39 | 25.97 | 6.78 | .04 | **.32** | .03 | **.31** | −.15 | .03 | .03 | −.10 | **.06** | −.08 | −.05 | **−.23** | — | |
| 14. Age | 40.00 | 65.00 | 53.42 | 7.57 | **−.16** | .12 | **−.19** | **.19** | −.06 | .13 | **.26** | **.16** | .03 | **.18** | **.27** | −.07 | −.05 | — |
| 15. Gender (% male) | F = 0 | M = 1 | 46%M | — | .06 | .11 | .03 | .12 | .03 | −.01 | .04 | −.07 | .10 | .01 | −.02 | **−.22** | .07 | .07 |

*Note.* $N = 175$; *SD* = standard deviation; CRP = C-reactive protein; IL-6 = interleukin-6; Summary statistics for the biomarkers of inflammation denoted with a * are given in raw units, whereas log transformed versions were used for the correlations and structural equation model analyses. BMI = body mass index. Bolded coefficients are significant at $p < .05$.

the *lavaan* package in R (Rosseel, 2012) with all predictor variables centered at sample means, and incomplete data was accommodated using full information maximum likelihood (Enders, 2010).

As a general framework, structural equation modeling (SEM) has been successfully used to examine associations between psychological predictors and inflammation (characterized as a latent construct with multiple indicators; e.g., Hostinar, Ross, Chen, & Miller, 2015; Petrov et al., 2016). While this approach conceptualizes each measure of inflammation as driven by a common factor (and thereby reduces measure-specific measurement error), the individual measures may also provide unique information about

Table 2
*Results from Structural Equation Models Examining Associations Between Global Emodiversity and Latent Inflammation*

| | Model 1 | | | | Model 2 | | | |
|---|---|---|---|---|---|---|---|---|
| Predictor | *Unstd* | *SE* | *Std* | *p* | *Unstd* | *SE* | *Std* | *p* |
| Common factor | | | | | | | | |
| Inflammation → CRP | = 1.00 | — | .88 | — | = 1.00 | — | .84 | — |
| Inflammation → IL-6 | .39 | .08 | .58 | <.001 | .43 | .07 | .62 | <.001 |
| Inflammation → Fibrinogen | .11 | .02 | .60 | <.001 | .12 | .02 | .61 | <.001 |
| Regression(s) | | | | | | | | |
| Global Emodiversity → Inflammation | −1.47 | .96 | −.14 | .126 | −1.24 | .88 | −.12 | .160 |
| BMI → Inflammation | — | — | — | — | .09 | .01 | .50 | <.001 |
| Anti-Inflammatory Medication → Inflammation | — | — | — | — | −.30 | .21 | −.13 | .149 |
| Medical Conditions → Inflammation | — | — | — | — | .14 | .08 | .15 | .093 |
| Neuroticism → Inflammation | — | — | — | — | −.01 | .02 | −.07 | .423 |
| Extraversion → Inflammation | — | — | — | — | −.002 | .01 | −.01 | .902 |
| Age → Inflammation | — | — | — | — | .03 | .01 | .18 | .027 |
| Gender → Inflammation | — | — | — | — | −.17 | .18 | −.08 | .324 |
| Intercepts | | | | | | | | |
| CRP | .37 | .10 | .28 | <.001 | .38 | .09 | .29 | <.001 |
| IL6 | .46 | .06 | .61 | <.001 | .47 | .06 | .61 | <.001 |
| Fibrinogen | 5.79 | .02 | 28.10 | <.001 | 5.79 | .02 | 28.06 | <.001 |
| Inflammation | .00 | — | .00 | — | .00 | — | .00 | — |
| Variances | | | | | | | | |
| CRP | .39 | .22 | .23 | .08 | .51 | .15 | .30 | .001 |
| IL6 | .38 | .05 | .66 | <.001 | .36 | .05 | .62 | <.001 |
| Fibrinogen | .03 | .004 | .65 | <.001 | .03 | .004 | .63 | <.001 |
| Inflammation | 1.28 | .28 | .98 | <.001 | .79 | .18 | .67 | <.001 |
| Model fit statistics | | | | | | | | |
| $\chi^2$ | ($df = 2$) = 1.920, $p = .383$ | | | | ($df = 10$) = 17.22, $p = .372$ | | | |
| CFI | 1.00 | | | | .991 | | | |
| RMSEA | .000 [.000, .148] | | | | .021 [.000, .074] | | | |
| SRMR | .022 | | | | .027 | | | |

*Note.* $N = 175$; *Unstd* = unstandardized coefficient; *SE* = standard error; *Std* = standardized path coefficient; *p* = *p*-value; CRP = c-reactive protein; IL-6 = interleukin-6; BMI = body mass index; CFI = comparative fit index; RMSEA = root-mean-square error of approximation; SRMR = standardized root-mean-square residual. Bracketed values represent 95% confidence intervals.

more specific inflammation processes. Making use of both of these perspectives, we also conducted follow-up regression analyses wherein each indicator was examined separately in three single-outcome regression models. Adequacy of fit of the SEM was determined using standard measures of fit as the discrepancy between the observed means and variance-covariance matrix (i.e., observed data) and the means and variance-covariance implied by the model (Bentler, 1990; Hu & Bentler, 1999). Specifically, we examined the $\chi^2$(overall measure of misfit), and a variety of metrics derived from that misfit, including the comparative fit index (comparison to a saturated model, good fit evaluated as CFI $>$ .96), the root mean square error of approximation (penalizes for model complexity, good fit as RMSEA $<$ .05), and the standardized root-mean-square residual (no penalty for complexity, good fit as SRMR $<$ .05).

## Results

### Descriptives

Descriptive statistics and intercorrelations among the study variables are provided in Table 1. As expected, IL-6 was positively correlated with both CRP ($r$ = .51) and fibrinogen ($r$ = .33), which were also positively correlated with each other ($r$ = .52). CRP levels ranged from 0.10 to 75.00 ($M$ = 3.59, $SD$ = 7.82) with 94% of sample within normal range of 0 to 10 mg/L; IL-6 levels ranged from 0.23 to 72.64 ($M$ = 2.56, $SD$ = 6.08) with 95% of sample within normal range of 0 to 7 pg/ml; and fibrinogen levels ranged from 195.00 to 712.00 ($M$ = 333.05, $SD$ = 75.52) with 87% of sample within normal range of 150 to 400 mg/dL. Distributions for IL-6, CRP, and fibrinogen values were positively skewed and therefore log-transformed for statistical analyses. Follow-up analyses with outlier cases (e.g., more than ±3 standard deviations away from the sample mean) removed provided the same pattern of results.

Consistent with previous work (Quoidbach et al., 2014), global emodiversity scores were correlated with negative emodiversity ($r$ = .79) and positive emodiversity ($r$ = .36), while positive emodiversity and negative emodiversity scores were uncorrelated ($r$ = .02).

### Associations Between Emodiversity and Inflammation

**Global emodiversity.** Models 1 and 2 examined the relation between global emodiversity and inflammation. Results are shown

Table 3
*Results from Structural Equation Models Examining Associations Between Positive and Negative Emodiversity and Latent Inflammation*

| Predictor | Model 3 | | | | Model 4 | | | |
|---|---|---|---|---|---|---|---|---|
| | *Unstd* | *SE* | *Std* | *p* | *Unstd* | *SE* | *Std* | *p* |
| Measurement model | | | | | | | | |
| Inflammation → CRP | = 1.00 | — | .86 | — | = 1.00 | — | .87 | — |
| Inflammation → IL-6 | .40 | .07 | .60 | <.001 | .40 | .07 | .59 | <.001 |
| Inflammation → Fibrinogen | .11 | .02 | .60 | <.001 | .11 | .02 | .59 | <.001 |
| Structural model | | | | | | | | |
| Positive Emodiversity → Inflammation | −2.90 | .81 | −.29 | <.001 | −3.88 | 1.20 | −.39 | .001 |
| Negative Emodiversity → Inflammation | .02 | .54 | .003 | .976 | .02 | .71 | .002 | .982 |
| Mean Positive Emotion → Inflammation | — | — | — | — | .23 | .18 | .16 | .186 |
| Mean Negative Emotion → Inflammation | — | — | — | — | .20 | .33 | .07 | .546 |
| BMI → Inflammation | .08 | .01 | .45 | <.001 | .08 | .01 | .43 | <.001 |
| Anti-Inflammatory Medication → Inflammation | −.24 | .21 | −.10 | .244 | −.23 | .21 | −.09 | .279 |
| Medical Conditions → Inflammation | .12 | .08 | .11 | .169 | .11 | .08 | .11 | .179 |
| Neuroticism → Inflammation | −.02 | .02 | −.11 | .188 | −.02 | .02 | −.11 | .202 |
| Extraversion → Inflammation | .01 | .01 | .08 | .325 | .01 | .01 | .07 | .368 |
| Age → Inflammation | .03 | .01 | .23 | .004 | .03 | .01 | .21 | .009 |
| Gender → Inflammation | −.15 | .17 | −.07 | .394 | −.15 | .17 | −.07 | .393 |
| Intercepts | | | | | | | | |
| CRP | .39 | .09 | .30 | <.001 | .39 | .09 | .30 | <.001 |
| IL6 | .47 | .06 | .62 | <.001 | .47 | .06 | .61 | <.001 |
| Fibrinogen | 5.79 | .02 | 28.09 | <.001 | 5.79 | .02 | 28.08 | <.001 |
| Inflammation | .00 | — | .00 | — | .00 | — | .00 | — |
| Residual variances | | | | | | | | |
| CRP | .43 | .16 | .25 | .006 | .40 | .16 | .24 | .013 |
| IL6 | .37 | .05 | .64 | <.001 | .38 | .05 | .65 | <.001 |
| Fibrinogen | .03 | .004 | .65 | <.001 | .03 | .004 | .65 | <.001 |
| Inflammation | .79 | .18 | .62 | <.001 | .80 | .18 | .62 | <.001 |
| Model Fit Statistics | | | | | | | | |
| $\chi^2$ | ($df$ = 18) = 22.07, $p$ = .229 | | | | ($df$ = 22) = 27.740, $p$ = .184 | | | |
| CFI | .973 | | | | .962 | | | |
| RMSEA | .036 [.000, .080] | | | | .039 [.000, .078] | | | |
| SRMR | .030 | | | | .027 | | | |

*Note.* $N$ = 175; *Unstd* = unstandardized coefficient; *SE* = standard error; *Std* = standardized coefficient; $p$ = $p$-value; CRP = c-reactive protein; IL-6 = interleukin-6; BMI = body mass index. CFI = comparative fit index; RMSEA = root-mean-square error of approximation; SRMR = standardized root-mean-square residual. Bracketed values represent 95% confidence intervals.

in Table 2. Although the overall fit of Model 1 to the data was good (e.g., RMSEA $<$ .05) and the inflammation factor was well defined (standardized factor loadings .88, .58, and .60), global emodiversity was, contrary to predictions, not significantly related to latent inflammation ($B = -.14$, $p = .13$). Although Model 2 also fit the data well (e.g., RMSEA $<$ .05), inclusion of age, gender, BMI, medication, medical conditions, and personality as covariates did not reveal any association between global emodiversity and inflammation ($B = -.14$, $p = .09$).

**Positive and negative emodiversity.** We next examined the extent to which positive and negative emodiversity were uniquely associated with inflammation. As seen in Table 3, Model 3 fit the data well (e.g., RMSEA $<$ .05). In accordance with hypotheses, positive emodiversity was related to inflammation ($B = -.26$, $p = .001$). In particular, greater positive emodiversity was associated with lower inflammation, independent of age, gender, anti-inflammatory medications, BMI, medical conditions, and personality. Contrary to hypotheses, negative emodiversity was not significantly related to latent inflammation ($B = -.03$, $p = .71$). We then explored whether the association between positive emodiversity and inflammation held, over and above mean levels of positive and negative affect. Results from this comprehensive model are shown in Figure 2. As seen in the figure and in Table 3, positive emodiversity was associated with inflammation, even after controlling for mean levels of positive and negative emotion ($B = -.38$, $p = .002$), while negative emodiversity was not associated with inflammation ($B = -.02$, $p = .86$).

## Supplemental Analyses

To assess the unique associations among the predictors and individual markers of inflammation, we supplemented the four structural equation models with 12 regression models wherein the three markers of inflammation were examined as unique outcome variables. The associations were generally consistent with the common factor approach, although some of the associations did not reach statistical significance. Parallel to the structural equation models, regression models indicated that global emodiversity was negatively, but nonsignificantly associated with CRP, IL6, or fibrinogen in base models (see Table 4), or after accounting for covariates (see Table 5). Regression models indicated that higher positive emodiversity was significantly associated with lower CRP and lower fibrinogen after adjusting for covariates (see Table 6). Additionally, the inverse relation between positive emodiversity and CRP remained significant after mean levels of positive and negative emotions had been included as additional covariates (see Table 7). Regression models indicated that positive emodiversity was not significantly associated with IL6, but the sign of the coefficient was in the hypothesized direction. Finally, in none of the regression models was negative emodiversity significantly associated with individual markers of inflammation.

## Discussion

This study had two principal goals. The first was to test the hypothesis that diversity in day-to-day positive and negative emotions would be associated with lower inflammatory activity. In SEM analyses adjusting for demographic and health covariates, we did not find an association between global emodiversity and latent inflammation (characterized by IL-6, CRP, and fibrinogen). These results differ from those of a prior study documenting better mental and physical health among adults reporting greater global emodiversity (Quoidbach et al., 2014). The discrepancies may reflect the different measurement approaches and populations sampled. For example, Quoidbach et al. (2014) used a single-occasion measure to derive emodiversity scores, whereas the current study used repeated measures of daily emotional experience obtained over 30 days. Thus, it may be that the associations between global emodiversity and health are limited to across-person (nomothetic) responses that are not captured by our within-person (idiographic)

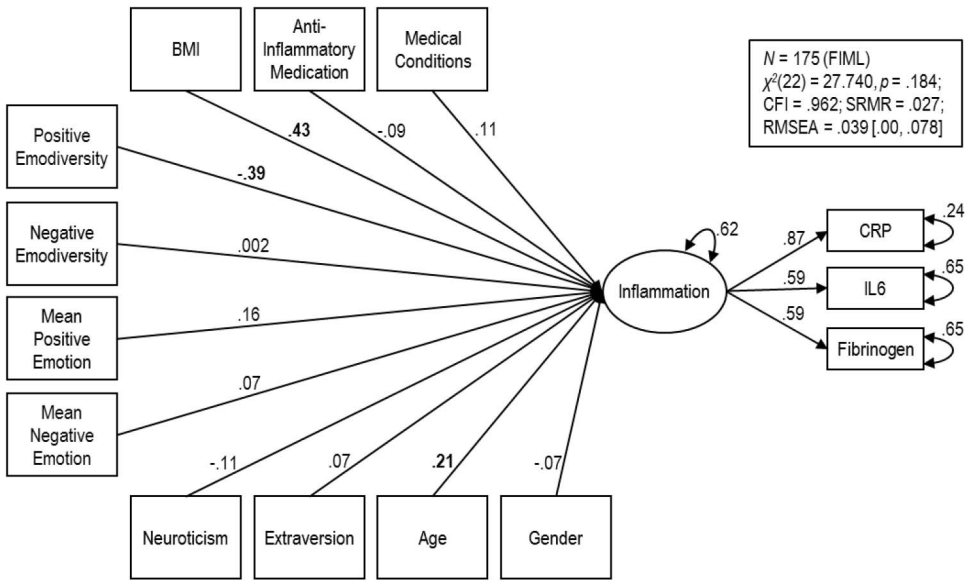


*Figure 2.* Structural and measurement models depicting results from Model 4. Values are standardized path coefficients and variances. Bolded coefficients are significant at $p < .05$.

Table 4
*Results from Unadjusted Regression Models Examining Associations Between Global Emodiversity and Markers of Inflammation*

| | CRP ($N$ = 162) | | | IL6 ($N$ = 162) | | | Fibrinogen ($N$ = 158) | | |
|---|---|---|---|---|---|---|---|---|---|
| Predictor | Estimate | *SE* | *p* | Estimate | *SE* | *p* | Estimate | *SE* | *p* |
| Intercept | .37 | .10 | <.001 | .46 | .06 | <.001 | 5.79 | .02 | <.001 |
| Global emodiversity | −1.18 | .99 | .234 | −.76 | .58 | .189 | −.31 | .16 | .052 |
| Residual *SE* | 1.30 | | | .76 | | | .20 | | |
| Adjusted $R^2$ | .003 | | | .005 | | | .02 | | |

*Note.* CRP = C-Reactive Protein; IL6 = Interleukin-6; *SE* = standard error; *p* = *p*-value.

measure of emodiversity (see Kenny, Kashy, & Bolger, 1998; Tennen & Affleck, 1996). A more systematic investigation of the relations between global emodiversity—assessed at multiple time scales—and inflammation is warranted to better understand the nature and health implications of individual differences in emodiversity. The study samples also differed in terms of cultural background. The sample in the Quoidbach et al. (2014) study was European (i.e., French and Belgian), whereas the sample in the current study was from the southwestern United States. Potential cultural differences in the links between global emodiversity and inflammation should be examined more closely in future work.

A second goal of the current study was to examine unique associations of positive and negative emodiversity with inflammation. As predicted, greater diversity in day-to-day positive emotions was related to lower systemic inflammation. This association remained significant after accounting for differences in demographic characteristics, BMI, medication use, medical conditions, personality, and mean levels of emotion. The finding is consistent with other studies examining links between positive affect and inflammation using conventional, single-occasion indices (Stellar et al., 2015; Steptoe et al., 2008). Importantly, the results are in line with prior work suggesting that intraindividual variability in positive emotions is important to psychological and physical health above and beyond mean levels (Gruber et al., 2013; Ong et al., 2013). Overall, these findings align with a functional account of "discrete" positive emotions that suggests biopsychosocial environments encountered in daily life can activate a diversity of positive emotions (e.g., pride, amusement, contentment), each serving a specific adaptive purpose (Shiota et al., in press; Shiota et al., 2014). In contrast, there was no association between negative emodiversity and inflammation. Prior research demonstrates that older adults show less intraindividual variability in negative emotions than younger adults (Brose et al., 2015; Grühn et al., 2013; Röcke, Li, & Smith, 2009). It is possible that the lack of association between emodiversity and inflammation in this study may reflect reduced intraindividual variability in participants' negative emotions. Future studies should attempt to replicate these findings in more age-heterogeneous samples.

This investigation also showed that higher positive emodiversity was associated with lower levels of CRP and fibrinogen. Further, the single-outcome regression models revealed that the association between positive emodiversity and CRP was unchanged when age, gender, anti-inflammatory medications, BMI, medical conditions, personality, and mean levels of positive and negative emotions were included as covariates. While not all associations were significant, it is worth noting that the substantive pattern of findings across all three markers of inflammation was in the predicted direction (i.e., higher positive emodiversity associated with lower inflammation). Mirroring the findings from the structural equation models, negative emodiversity was not associated with any of the biomarkers of inflammation in the separate regression models.

## Limitations

Our conclusions are limited by some features of our methods and analyses. First, our sample consisted of a cross-section of relatively healthy middle-aged adults. Both the restricted age range (age 45 to 60 years) and sample size ($N$ = 175) limit the gener-

Table 5
*Results from Adjusted Regression Models Examining Associations Between Global Emodiversity and Markers of Inflammation*

| | CRP ($N$ = 153) | | | IL6 ($N$ = 153) | | | Fibrinogen ($N$ = 150) | | |
|---|---|---|---|---|---|---|---|---|---|
| Predictor | Estimate | *SE* | *p* | Estimate | *SE* | *p* | Estimate | *SE* | *p* |
| Intercept | .37 | .10 | <.001 | .48 | .06 | <.001 | 5.78 | .02 | <.001 |
| Global emodiversity | −1.37 | 1.02 | .181 | −.49 | .61 | .419 | −.29 | .17 | .089 |
| BMI | .09 | .02 | <.001 | .03 | .01 | <.001 | .01 | .002 | <.001 |
| Anti-inflammatory medication | −.22 | .23 | .330 | −.32 | .14 | .021 | −.04 | .04 | .326 |
| Medical conditions | .11 | .10 | .251 | .10 | .06 | .080 | .02 | .02 | .270 |
| Neuroticism | −.01 | .02 | .777 | −.002 | .01 | .886 | −.004 | .003 | .153 |
| Extraversion | .01 | .01 | .668 | .005 | .01 | .568 | −.004 | .002 | .133 |
| Age | .02 | .01 | .098 | .03 | .01 | <.001 | .003 | .002 | .118 |
| Gender | −.17 | .20 | .405 | −.02 | .12 | .886 | −.04 | .03 | .185 |
| Residual *SE* | 1.19 | | | .71 | | | .19 | | |
| Adjusted $R^2$ | .19 | | | .16 | | | .11 | | |

*Note.* CRP = C-Reactive Protein; IL6 = Interleukin-6; *SE* = standard error; *p* = *p*-value.

Table 6
*Results from Adjusted Regression Models Examining Associations Between Positive and Negative Emodiversity and Markers of Inflammation*

| | CRP ($N$ = 153) | | | IL6 ($N$ = 153) | | | Fibrinogen ($N$ = 150) | | |
|---|---|---|---|---|---|---|---|---|---|
| Predictor | Estimate | *SE* | *p* | Estimate | *SE* | *p* | Estimate | *SE* | *p* |
| Intercept | .37 | .09 | <.001 | .47 | .06 | <.001 | 5.78 | .02 | <.001 |
| Positive emodiversity | −3.40 | .85 | <.001 | −.73 | .53 | .167 | −.30 | .15 | .045 |
| Negative emodiversity | .27 | .59 | .652 | −.37 | .36 | .311 | −.08 | .10 | .432 |
| BMI | .08 | .01 | <.001 | .03 | .01 | <.001 | .01 | .003 | .002 |
| Anti-inflammatory medication | −.16 | .22 | .465 | −.28 | .14 | .044 | −.03 | .04 | .376 |
| Medical conditions | .09 | .09 | .352 | .10 | .06 | .100 | .02 | .02 | .320 |
| Neuroticism | −.01 | .02 | .424 | −.0003 | .01 | .979 | −.005 | .003 | .099 |
| Extraversion | .02 | .01 | .103 | .01 | .01 | .265 | −.002 | .003 | .404 |
| Age | .03 | .01 | .015 | .03 | .01 | .001 | .004 | .002 | .063 |
| Gender | −.15 | .19 | .440 | −.01 | .12 | .945 | −.05 | .03 | .167 |
| Residual *SE* | 1.14 | | | .70 | | | .19 | | |
| Adjusted $R^2$ | .26 | | | .17 | | | .12 | | |

*Note.* CRP = C-Reactive Protein; IL6 = Interleukin-6; *SE* = standard error; *p* = *p*-value.

alizability of results. Although we attempted to examine the extent to which associations between emodiversity and inflammatory markers (i.e., IL-6, CRP, fibrinogen) were independent of potential confounding variables (e.g., age, gender, anti-inflammatory medications, BMI, medical conditions, personality, mean level of emotions), future research should explore whether the associations hold when accounting for a variety of other personal characteristics that may drive emodiversity (e.g., cognitive control). Second, our analyses of emodiversity relied heavily on emotion reports that were completed at the end of each day. It is well established the emotions vary both within day and across days (Clark, Watson, & Leeka, 1989; Watson, Wiese, Vaidya, & Tellegen, 1999). Our emotion reports were similarly limited by the number of emotions (16 positive emotions and 16 negative emotions) participants were asked to report on a daily basis, thereby restricting the degree to which the breadth of participants' emotional experience could be adequately captured (for a discussion, see Brown & Coyne, in press). Thus, future research should include more intensive experience sampling approaches (Steptoe & Wardle, 2011) that allow for modeling of diurnal and circadian patterns in emotion, and emotion scales that allow for a greater number of emotions to be reported. Third, our data do not speak to the underlying mechanisms of emodiversity. Emodiversity may act to reduce negative appraisals of stress and facilitate adaptive coping. Alternatively, emodiversity may impact behaviors relevant to health in general, irrespective of its influence on stress responses. It may be that systemic inflammation is among the mediating factors linking emodiversity to subsequent psychological morbidity. These hypothesized processes have yet to be empirically investigated. Finally, because our study was observational in nature, the directionality of the observed associations cannot be determined. For example, it is possible that a lack of diversity of both positive and negative emotional experience may result from heightened inflammatory responses. This issue highlights the need for longitudinal assessments to better characterize the temporal relationships between emodiversity and inflammation.

Table 7
*Results from Adjusted Regression Models Examining Associations Between Positive and Negative Emodiversity, Mean Positive and Negative Emotion, and Markers of Inflammation*

| | CRP ($N$ = 153) | | | IL6 ($N$ = 153) | | | Fibrinogen ($N$ = 150) | | |
|---|---|---|---|---|---|---|---|---|---|
| Predictor | Estimate | *SE* | *p* | Estimate | *SE* | *p* | Estimate | *SE* | *p* |
| Intercept | .36 | .09 | <.001 | .47 | .06 | <.001 | 5.78 | .02 | <.001 |
| Positive emodiversity | −5.03 | 1.25 | <.001 | −.75 | .77 | .329 | −.27 | .22 | .213 |
| Negative emodiversity | .45 | .74 | .546 | −.83 | .46 | .071 | −.05 | .13 | .698 |
| Mean positive emotion | .36 | .19 | .060 | .07 | .12 | .571 | −.01 | .03 | .768 |
| Mean negative emotion | .09 | .36 | .801 | .42 | .22 | .056 | −.03 | .06 | .628 |
| BMI | .07 | .01 | <.001 | .03 | .01 | <.001 | .01 | .003 | .002 |
| Anti-inflammatory medication | −.12 | .22 | .600 | −.30 | .14 | .032 | −.03 | .04 | .400 |
| Medical conditions | .09 | .09 | .328 | .08 | .06 | .149 | .02 | .02 | .304 |
| Neuroticism | −.01 | .02 | .507 | −.002 | .01 | .843 | −.005 | .003 | .109 |
| Extraversion | .02 | .01 | .137 | .01 | .01 | .231 | −.002 | .003 | .409 |
| Age | .03 | .01 | .034 | .03 | .01 | .002 | .004 | .002 | .060 |
| Gender | −.15 | .19 | .425 | −.02 | .12 | .898 | −.05 | .03 | .173 |
| Residual *SE* | 1.13 | | | .70 | | | .19 | | |
| Adjusted $R^2$ | .26 | | | .18 | | | .11 | | |

*Note.* CRP = C-Reactive Protein; IL6 = Interleukin-6; *SE* = standard error; *p* = *p*-value.

## Conclusions

Despite these limitations, the findings add to the evidence that positive affective states are related to favorable profiles of biological functioning that may contribute to reduced risk of chronic disease, while suggesting that diversity in day-to-day positive emotions is related to reduced levels of systemic inflammation.

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