

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 (Duijvis 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; Duijvis 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, & Mollay, 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 individual's 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

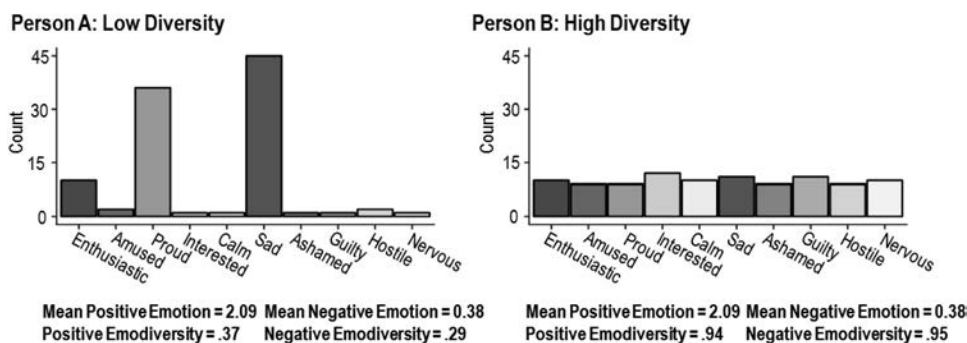


Figure 1. Individual differences in *emodiversity*—the breadth (the number of discrete emotions experienced) and evenness (the distribution of experiences across discrete emotions) of emotional experience. Left panel: Person A has low *emodiversity*, with emotion experiences that are relatively homogenous 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,

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

where c_{ij} is the count of individual i 's emotion experiences within $j = 1$ to m emotion types, indexed in ascending order ($c_{ij} \leq c_{i,j+1}$) 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 $G_A = 1 - \left(\frac{2 \sum_{j=1}^m j c_{ij}}{5 \sum_{j=1}^m c_{ij}} - \frac{5+1}{5} \right) = 1 - .63 = .37$. 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 $G_B = 1 - \left(\frac{2 \sum_{j=1}^m j c_{ij}}{5 \sum_{j=1}^m c_{ij}} - \frac{5+1}{5} \right) = 1 - .06 = .94$. 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 psy-

chological 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 Emodyersity and Latent Inflammation

Predictor	Model 1				Model 2			
	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 Emodyersity → 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								
χ^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 χ^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			
	<i>Unstd</i>	<i>SE</i>	<i>Std</i>	<i>p</i>	<i>Unstd</i>	<i>SE</i>	<i>Std</i>	<i>p</i>
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								
χ^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)

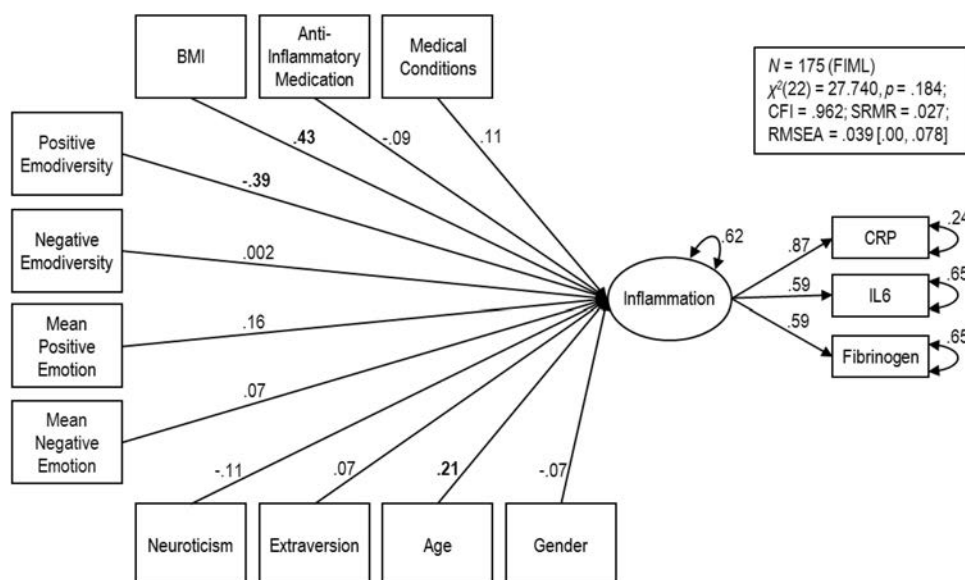


Figure 2. Structural and measurement models depicting results from Model 4. Values are standardized path coefficients and variances. Bolded coefficients are significant at $p < .05$.

Table 4

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

Predictor	CRP (<i>N</i> = 162)			IL6 (<i>N</i> = 162)			Fibrinogen (<i>N</i> = 158)		
	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>
Intercept	.37	.10	<.001	.46	.06	<.001	5.79	.02	<.001
Global emodiversity	-1.18	.99	.234	-.76	.58	.189	-.31	.16	.052
Residual <i>SE</i>	1.30			.76			.20		
Adjusted <i>R</i> 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; Grünh 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

Predictor	CRP (<i>N</i> = 153)			IL6 (<i>N</i> = 153)			Fibrinogen (<i>N</i> = 150)		
	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>
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 <i>SE</i>	1.19			.71			.19		
Adjusted <i>R</i> 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

Predictor	CRP (<i>N</i> = 153)			IL6 (<i>N</i> = 153)			Fibrinogen (<i>N</i> = 150)		
	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>
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 <i>SE</i>	1.14			.70			.19		
Adjusted <i>R</i> 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 exper-

ience 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

Predictor	CRP (<i>N</i> = 153)			IL6 (<i>N</i> = 153)			Fibrinogen (<i>N</i> = 150)		
	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>	Estimate	<i>SE</i>	<i>p</i>
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 <i>SE</i>	1.13			.70			.19		
Adjusted <i>R</i> 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.

References

- Adler, J. M., & Hershfield, H. E. (2012). Mixed emotional experience is associated with and precedes improvements in psychological well-being. *PLoS ONE*, *7*, e35633. <http://dx.doi.org/10.1371/journal.pone.0035633>
- Ai, A. L., Kronfol, Z., Seymour, E., & Bolling, S. F. (2005). Effects of mood state and psychosocial functioning on plasma Interleukin-6 in adult patients before cardiac surgery. *International Journal of Psychiatry in Medicine*, *35*, 363–376. <http://dx.doi.org/10.2190/2ELG-RDUN-X6TU-FG8C>
- Barrett, K., & Campos, J. (1987). Perspectives on emotional development: II. A functionalist approach to emotion. In J. Osofsky (Ed.), *Handbook of infant development* (Vol. 2, pp. 555–578). New York, NY: Wiley.
- Barrett, L. F. (1998). Discrete emotions or dimensions? The role of valence focus and arousal focus. *Cognition and Emotion*, *12*, 579–599. <http://dx.doi.org/10.1080/026999398379574>
- Barrett, L. F. (2006). Solving the emotion paradox: Categorization and the experience of emotion. *Personality and Social Psychology Review*, *10*, 20–46. http://dx.doi.org/10.1207/s15327957pspr1001_2
- Barrett, L. F., Gross, J., Christensen, T. C., & Benvenuto, M. (2001). Knowing what you're feeling and knowing what to do about it: Mapping the relation between emotion differentiation and emotion regulation. *Cognition and Emotion*, *15*, 713–724. <http://dx.doi.org/10.1080/02699930143000239>
- Barrett, L. F., Mesquita, B., Ochsner, K. N., & Gross, J. J. (2007). The experience of emotion. *Annual Review of Psychology*, *58*, 373–403. <http://dx.doi.org/10.1146/annurev.psych.58.110405.085709>
- Benson, L., Ram, N., Almeida, D., Zautra, A., & Ong, A. D. (in press). Fusing biodiversity metrics into investigations of daily life and healthy aging: Recommendations and illustrations with emodiversity. *The Journals of Gerontology: Series B, Psychological Sciences and Social Sciences*.
- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, *107*, 238–246. <http://dx.doi.org/10.1037/0033-2909.107.2.238>
- Blomberg, B. B., Alvarez, J. P., Diaz, A., Romero, M. G., Lechner, S. C., Carver, C. S., . . . Antoni, M. H. (2009). Psychosocial adaptation and cellular immunity in breast cancer patients in the weeks after surgery: An exploratory study. *Journal of Psychosomatic Research*, *67*, 369–376. <http://dx.doi.org/10.1016/j.jpsychores.2009.05.016>
- Brose, A., de Roover, K., Ceulemans, E., & Kuppens, P. (2015). Older adults' affective experiences across 100 days are less variable and less complex than younger adults'. *Psychology and Aging*, *30*, 194–208. <http://dx.doi.org/10.1037/a0038690>
- Brown, N., & Coyne, J. C. (in press). Emodiversity: Robust predictor of outcomes or statistical artifact? *Journal of Experimental Psychology: General*.
- Budescu, D. V., & Budescu, M. (2012). How to measure diversity when you must. *Psychological Methods*, *17*, 215–227. <http://dx.doi.org/10.1037/a0027129>
- Carstensen, L. L., Pasupathi, M., Mayr, U., & Nesselroade, J. R. (2000). Emotional experience in everyday life across the adult life span. *Journal of Personality and Social Psychology*, *79*, 644–655. <http://dx.doi.org/10.1037/0022-3514.79.4.644>
- Carstensen, L. L., Turan, B., Scheibe, S., Ram, N., Ersner-Hershfield, H., Samanez-Larkin, G. R., . . . Nesselroade, J. R. (2011). Emotional experience improves with age: Evidence based on over 10 years of experience sampling. *Psychology and Aging*, *26*, 21–33. <http://dx.doi.org/10.1037/a0021285>
- Cesari, M., Penninx, B. W., Newman, A. B., Kritchevsky, S. B., Nicklas, B. J., Sutton-Tyrrell, K., . . . Pahor, M. (2003). Inflammatory markers and cardiovascular disease. *The American Journal of Cardiology*, *92*, 522–528. [http://dx.doi.org/10.1016/S0002-9149\(03\)00718-5](http://dx.doi.org/10.1016/S0002-9149(03)00718-5)
- Clark, L. A., Watson, D., & Leeka, J. (1989). Diurnal variation in the positive affects. *Motivation and Emotion*, *13*, 205–234. <http://dx.doi.org/10.1007/BF00995536>
- Cohen, S., Alper, C. M., Doyle, W. J., Treanor, J. J., & Turner, R. B. (2006). Positive emotional style predicts resistance to illness after experimental exposure to rhinovirus or influenza a virus. *Psychosomatic Medicine*, *68*, 809–815. <http://dx.doi.org/10.1097/01.psy.0000245867.92364.3c>
- Coifman, K. G., Bonanno, G. A., & Rafaeli, E. (2007). Affect dynamics, bereavement and resilience to loss. *Journal of Happiness Studies*, *8*, 371–392. <http://dx.doi.org/10.1007/s10902-006-9014-5>
- Demiralp, E., Thompson, R. J., Mata, J., Jaeggi, S. M., Buschkuhl, M., Barrett, L. F., . . . Jonides, J. (2012). Feeling blue or turquoise? Emotional differentiation in major depressive disorder. *Psychological Science*, *23*, 1410–1416. <http://dx.doi.org/10.1177/0956797612444903>
- Deverts, D. J., Cohen, S., DiLillo, V. G., Lewis, C. E., Kiefe, C., Whooley, M., & Matthews, K. A. (2010). Depressive symptoms, race, and circulating C-reactive protein: The Coronary Artery Risk Development in Young Adults (CARDIA) study. *Psychosomatic Medicine*, *72*, 734–741. <http://dx.doi.org/10.1097/PSY.0b013e3181ec4b98>
- Dickerson, S. S., Kemeny, M. E., Aziz, N., Kim, K. H., & Fahey, J. L. (2004). Immunological effects of induced shame and guilt. *Psychosomatic Medicine*, *66*, 124–131. <http://dx.doi.org/10.1097/01.PSY.0000097338.75454.29>
- Duivis, H. E., de Jonge, P., Penninx, B. W., Na, B. Y., Cohen, B. E., & Whooley, M. A. (2011). Depressive symptoms, health behaviors, and subsequent inflammation in patients with coronary heart disease: Prospective findings from the heart and soul study. *The American Journal of Psychiatry*, *168*, 913–920. <http://dx.doi.org/10.1176/appi.ajp.2011.10081163>
- Enders, C. K. (2010). *Applied missing data analysis*. New York, NY: Guilford Press.
- Epel, E. S., & Lithgow, G. J. (2014). Stress biology and aging mechanisms: Toward understanding the deep connection between adaptation to stress and longevity. *The Journals of Gerontology*, *69*, S10–S16. <http://dx.doi.org/10.1093/gerona/glu055>
- Friedman, E. M., & Herd, P. (2010). Income, education, and inflammation: Differential associations in a national probability sample (The MIDUS study). *Psychosomatic Medicine*, *72*, 290–300. <http://dx.doi.org/10.1097/PSY.0b013e3181cfe4c2>
- Gini, C. (1921). Measurement of inequality of incomes. *The Economic Journal*, *31*, 124–126.
- Grossmann, I., Huynh, A. C., & Ellsworth, P. C. (2016). Emotional complexity: Clarifying definitions and cultural correlates. *Journal of Personality and Social Psychology*, *111*, 895–916. <http://dx.doi.org/10.1037/pspp0000084>
- Gruber, J., & Bekoff, M. (2017). A cross-species comparative approach to positive emotion disturbance. *Emotion Review*, *9*, 72–78. <http://dx.doi.org/10.1177/1754073915615430>
- Gruber, J., Kogan, A., Quoidbach, J., & Mauss, I. B. (2013). Happiness is best kept stable: Positive emotion variability is associated with poorer psychological health. *Emotion*, *13*, 1–6. <http://dx.doi.org/10.1037/a0030262>
- Grühn, D., Lumley, M. A., Diehl, M., & Labouvie-Vief, G. (2013). Time-based indicators of emotional complexity: Interrelations and correlates. *Emotion*, *13*, 226–237. <http://dx.doi.org/10.1037/a0030363>
- Hamilton, E. (1942). *The Greek way*. New York, NY: Norton.

- Hay, E. L., & Diehl, M. (2011). Emotion complexity and emotion regulation across adulthood. *European Journal of Ageing, 8*, 157–168. <http://dx.doi.org/10.1007/s10433-011-0191-7>
- Hershfield, H. E., Scheibe, S., Sims, T. L., & Carstensen, L. L. (2013). When feeling bad can be good: Mixed emotions benefit physical health across adulthood. *Social Psychological & Personality Science, 4*, 54–61. <http://dx.doi.org/10.1177/1948550612444616>
- Hostinar, C. E., Ross, K. M., Chen, E., & Miller, G. E. (2015). Modeling the association between lifecourse socioeconomic disadvantage and systemic inflammation in healthy adults: The role of self-control. *Health Psychology, 34*, 580–590. <http://dx.doi.org/10.1037/hea0000130>
- Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with C-reactive protein, IL-1, and IL-6: A meta-analysis. *Psychosomatic Medicine, 71*, 171–186. <http://dx.doi.org/10.1097/PSY.0b013e3181907c1b>
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6*, 1–55. <http://dx.doi.org/10.1080/10705519909540118>
- Hülür, G., Hoppmann, C. A., Ram, N., & Gerstorf, D. (2015). Developmental associations between short-term variability and long-term changes: Intraindividual correlation of positive and negative affect in daily life and cognitive aging. *Developmental Psychology, 51*, 987–997. <http://dx.doi.org/10.1037/a0039341>
- Janicki-Deverts, D., Cohen, S., Doyle, W. J., Turner, R. B., & Treanor, J. J. (2007). Infection-induced proinflammatory cytokines are associated with decreases in positive affect, but not increases in negative affect. *Brain, Behavior, and Immunity, 21*, 301–307. <http://dx.doi.org/10.1016/j.bbi.2006.09.002>
- John, O. P., & Srivastava, S. (1999). The Big Five trait taxonomy: History, measurement, and theoretical perspectives. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed., pp. 102–138). New York, NY: Guilford.
- Kashdan, T. B., & Farmer, A. S. (2014). Differentiating emotions across contexts: Comparing adults with and without social anxiety disorder using random, social interaction, and daily experience sampling. *Emotion, 14*, 629–638. <http://dx.doi.org/10.1037/a0035796>
- Keltner, D., & Gross, J. J. (1999). Functional accounts of emotions. *Cognition and Emotion, 13*, 467–480. <http://dx.doi.org/10.1080/02699399379140>
- Kenny, D. A., Kashy, D. A., & Bolger, N. (1998). Data analysis in social psychology. In S. T. Fiske & D. T. Gilbert (Eds.), *The handbook of social psychology* (4th ed., pp. 233–265). New York, NY: McGraw-Hill.
- Koffer, R. E., Ram, N., Conroy, D. E., Pincus, A. L., & Almeida, D. M. (2016). Stressor diversity: Introduction and empirical integration into the daily stress model. *Psychology and Aging, 31*, 301–320. <http://dx.doi.org/10.1037/pag0000095>
- Larsen, J. T., & McGraw, A. P. (2014). The case for mixed emotions. *Social and Personality Psychology Compass, 8*, 263–274. <http://dx.doi.org/10.1111/spc3.12108>
- Lee, S., Koffer, R. E., Sprague, B. N., Charles, S. T., Ram, N., & Almeida, D. M. (2016). Activity diversity and its associations with psychological well-being across adulthood. *The Journals of Gerontology*. Advance online publication. <http://dx.doi.org/10.1093/geronb/gbw118>
- Li, Y., Zhang, G., Feng, Y., & Wu, C. (2015). An entropy-based social network community detecting method and its application to scientometrics. *Scientometrics, 102*, 1003–1017. <http://dx.doi.org/10.1007/s11192-014-1377-5>
- Lindquist, K., & Barrett, L. F. (2008). Emotional complexity. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *The handbook of emotion* (3rd ed., pp. 513–530). New York, NY: Guilford Press.
- McCaffery, J. M., Marsland, A. L., Strohacker, K., Muldoon, M. F., & Manuck, S. B. (2012). Factor structure underlying components of allostatic load. *PLoS ONE, 7*, e47246. <http://dx.doi.org/10.1371/journal.pone.0047246>
- Miller, G. E., & Blackwell, E. (2006). Turning up the heat: Inflammation as a mechanism linking chronic stress, depression, and heart disease. *Current Directions in Psychological Science, 15*, 269–272. <http://dx.doi.org/10.1111/j.1467-8721.2006.00450.x>
- Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*, 959–997. <http://dx.doi.org/10.1037/a0024768>
- Miller, G. E., Rohleder, N., Stetler, C., & Kirschbaum, C. (2005). Clinical depression and regulation of the inflammatory response during acute stress. *Psychosomatic Medicine, 67*, 679–687. <http://dx.doi.org/10.1097/01.psy.0000174172.82428.ce>
- Miyamoto, Y., & Ryff, C. D. (2011). Cultural differences in the dialectical and non-dialectical emotional styles and their implications for health. *Cognition and Emotion, 25*, 22–39. <http://dx.doi.org/10.1080/02699931003612114>
- Miyamoto, Y., Uchida, Y., & Ellsworth, P. C. (2010). Culture and mixed emotions: Co-occurrence of positive and negative emotions in Japan and the United States. *Emotion, 10*, 404–415. <http://dx.doi.org/10.1037/a0018430>
- Moons, W. G., Eisenberger, N. I., & Taylor, S. E. (2010). Anger and fear responses to stress have different biological profiles. *Brain, Behavior, and Immunity, 24*, 215–219. <http://dx.doi.org/10.1016/j.bbi.2009.08.009>
- Moons, W. G., & Shields, G. S. (2015). Anxiety, not anger, induces inflammatory activity: An avoidance/approach model of immune system activation. *Emotion, 15*, 463–476. <http://dx.doi.org/10.1037/emo000055>
- Moreno, P. I., Moskowitz, A. L., Ganz, P. A., & Bower, J. E. (2016). Positive affect and inflammatory activity in breast cancer survivors: Examining the role of affective arousal. *Psychosomatic Medicine, 78*, 532–541. <http://dx.doi.org/10.1097/PSY.0000000000000300>
- Morin, P. J. (1999). *Community ecology*. Malden, MA: Blackwell Publishing.
- Ong, A. D., & Bergeman, C. S. (2004). The complexity of emotions in later life. *The Journals of Gerontology Series B, Psychological Sciences and Social Sciences, 59*, 117–122. <http://dx.doi.org/10.1093/geronb/59.3.P117>
- Ong, A. D., Exner-Cortens, D., Riffin, C., Steptoe, A., Zautra, A., & Almeida, D. M. (2013). Linking stable and dynamic features of positive affect to sleep. *Annals of Behavioral Medicine, 46*, 52–61. <http://dx.doi.org/10.1007/s12160-013-9484-8>
- Petrov, M. E., Davis, M. C., Belyea, M. J., & Zautra, A. J. (2016). Linking childhood abuse and hypertension: Sleep disturbance and inflammation as mediators. *Journal of Behavioral Medicine, 39*, 716–726. <http://dx.doi.org/10.1007/s10865-016-9742-x>
- Pitsavos, C., Panagiotakos, D. B., Papageorgiou, C., Tsetsekou, E., Soldatos, C., & Stefanadis, C. (2006). Anxiety in relation to inflammation and coagulation markers, among healthy adults: The ATTICA study. *Atherosclerosis, 185*, 320–326. <http://dx.doi.org/10.1016/j.atherosclerosis.2005.06.001>
- Prather, A. A., Marsland, A. L., Muldoon, M. F., & Manuck, S. B. (2007). Positive affective style covaries with stimulated IL-6 and IL-10 production in a middle-aged community sample. *Brain, Behavior, and Immunity, 21*, 1033–1037. <http://dx.doi.org/10.1016/j.bbi.2007.04.009>
- Quoidbach, J., Gruber, J., Mikolajczak, M., Kogan, A., Kotsou, I., & Norton, M. I. (2014). Emodiversity and the emotional ecosystem. *Journal of Experimental Psychology: General, 143*, 2057–2066. <http://dx.doi.org/10.1037/a0038025>
- Ram, N., Conroy, D. E., Pincus, A. L., Hyde, A. L., & Molloy, L. (2012). Tethering theory to method: Using measures of intraindividual variability to operationalize individuals' dynamic characteristics. In G. Hancock & J. Harring (Eds.), *Advances in longitudinal modeling in the social and*

- behavioral sciences* (pp. 81–110). Charlotte, NC: Information Age Publishing.
- Ram, N., & Gerstorf, D. (2009). Time-structured and net intraindividual variability: Tools for examining the development of dynamic characteristics and processes. *Psychology and Aging, 24*, 778–791. <http://dx.doi.org/10.1037/a0017915>
- Ready, R. E., Carvalho, J. O., & Weinberger, M. I. (2008). Emotional complexity in younger, midlife, and older adults. *Psychology and Aging, 23*, 928–933. <http://dx.doi.org/10.1037/a0014003>
- Robles, T. F., Brooks, K. P., & Pressman, S. D. (2009). Trait positive affect buffers the effects of acute stress on skin barrier recovery. *Health Psychology, 28*, 373–378. <http://dx.doi.org/10.1037/a0014662>
- Röcke, C., Li, S. C., & Smith, J. (2009). Intraindividual variability in positive and negative affect over 45 days: Do older adults fluctuate less than young adults? *Psychology and Aging, 24*, 863–878. <http://dx.doi.org/10.1037/a0016276>
- Rosseel, Y. (2012). lavaan: An R package for structural equation modeling. *Journal of Statistical Software, 48*, 1–36. <http://dx.doi.org/10.18637/jss.v048.i02>
- Russell, J. A. (1980). A circumplex model of affect. *Journal of Personality and Social Psychology, 39*, 1161–1178. <http://dx.doi.org/10.1037/h0077714>
- Schneiderman, N., Ironson, G., & Siegel, S. D. (2005). Stress and health: Psychological, behavioral, and biological determinants. *Annual Review of Clinical Psychology, 1*, 607–628. <http://dx.doi.org/10.1146/annurev.clinpsy.1.102803.144141>
- Schwarz, N. (1990). Feelings as information: Informational and motivational functions of affective states. In R. M. Sorrentino & E. T. Higgins (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 527–561). New York, NY: Guilford Press.
- Schwarz, N., & Clore, G. L. (1983). Mood, misattribution, and judgments of well-being: Informative and directive functions of affective states. *Journal of Personality and Social Psychology, 45*, 513–523. <http://dx.doi.org/10.1037/0022-3514.45.3.513>
- Sepah, S. C., & Bower, J. E. (2009). Positive affect and inflammation during radiation treatment for breast and prostate cancer. *Brain, Behavior, and Immunity, 23*, 1068–1072. <http://dx.doi.org/10.1016/j.bb.2009.06.149>
- Sherwin, W. B. (2010). Entropy and information approaches to genetic diversity and its expression: Genomic geography. *Entropy, 12*, 1765–1798. <http://dx.doi.org/10.3390/e12071765>
- Shiota, M. N., Campos, B., Oveis, C., Hertenstein, M. J., Simon-Thomas, E., & Keltner, D. (in press). Beyond happiness: Building a science of discrete positive emotions. *American Psychologist*.
- Shiota, M. N., Neufeld, S. L., Danvers, A. F., Osborne, E. A., Sng, O., & Yee, C. I. (2014). Positive emotion differentiation: A functional approach. *Social and Personality Psychology Compass, 8*, 104–117. <http://dx.doi.org/10.1111/spc3.12092>
- Sims, T., Tsai, J. L., Jiang, D., Wang, Y., Fung, H. H., & Zhang, X. (2015). Wanting to maximize the positive and minimize the negative: Implications for mixed affective experience in American and Chinese contexts. *Journal of Personality and Social Psychology, 109*, 292–315. <http://dx.doi.org/10.1037/a0039276>
- Stellar, J. E., John-Henderson, N., Anderson, C. L., Gordon, A. M., McNeil, G. D., & Keltner, D. (2015). Positive affect and markers of inflammation: Discrete positive emotions predict lower levels of inflammatory cytokines. *Emotion, 15*, 129–133. <http://dx.doi.org/10.1037/emo0000033>
- Steptoe, A., O'Donnell, K., Badrick, E., Kumari, M., & Marmot, M. (2008). Neuroendocrine and inflammatory factors associated with positive affect in healthy men and women: The Whitehall II study. *American Journal of Epidemiology, 167*, 96–102. <http://dx.doi.org/10.1093/aje/kwm252>
- Steptoe, A., & Wardle, J. (2011). Positive affect measured using ecological momentary assessment and survival in older men and women. *Proceedings of the National Academy of Sciences of the United States of America, 108*, 18244–18248. <http://dx.doi.org/10.1073/pnas.1110892108>
- Sturgeon, J. A., Arewasikporn, A., Okun, M. A., Davis, M. C., Ong, A. D., & Zautra, A. J. (2016). The psychosocial context of financial stress: Implications for inflammation and psychological health. *Psychosomatic Medicine, 78*, 134–143. <http://dx.doi.org/10.1097/PSY.00000000000000276>
- Suarez, E. C. (2003). Joint effect of hostility and severity of depressive symptoms on plasma interleukin-6 concentration. *Psychosomatic Medicine, 65*, 523–527. <http://dx.doi.org/10.1097/01.PSY.0000062530.94551.EA>
- Tennen, H., & Affleck, G. (1996). Daily processes in coping with chronic pain: Methods and analytic strategies. In N. S. Endler & M. Zeidner (Eds.), *Handbook of coping: Theory, research, applications* (pp. 151–177). Oxford, UK: Wiley.
- Tomko, R. L., Lane, S. P., Pronove, L. M., Treloar, H. R., Brown, W. C., Solhan, M. B., . . . Trull, T. J. (2015). Undifferentiated negative affect and impulsivity in borderline personality and depressive disorders: A momentary perspective. *Journal of Abnormal Psychology, 124*, 740–753. <http://dx.doi.org/10.1037/abn0000064>
- Tugade, M. M., Fredrickson, B. L., & Feldman-Barrett, L. (2004). Psychological resilience and positive emotional granularity: Examining the benefits of positive emotions on coping and health. *Journal of Personality and Social Psychology, 72*, 1161–1190. <http://dx.doi.org/10.1111/j.1467-6494.2004.00294.x>
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070. <http://dx.doi.org/10.1037/0022-3514.54.6.1063>
- Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology, 76*, 820–838. <http://dx.doi.org/10.1037/0022-3514.76.5.820>
- Wessman, A. E., & Ricks, D. F. (1966). *Mood and personality*. Oxford, UK: Holt, Rinehart, and Winston.
- West, S. G., & Hepworth, J. T. (1991). Statistical issues in the study of temporal data: Daily experiences. *Journal of Personality, 59*, 609–662. <http://dx.doi.org/10.1111/j.1467-6494.1991.tb00261.x>
- Zautra, A. J., Affleck, G. G., Tennen, H., Reich, J. W., & Davis, M. C. (2005). Dynamic approaches to emotions and stress in everyday life: Bolger and Zuckerman reloaded with positive as well as negative affects. *Journal of Personality, 73*, 1511–1538. <http://dx.doi.org/10.1111/j.0022-3506.2005.00357.x>

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