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Is Anger, but Not Sadness, Associated With Chronic Inflammation and Illness in Older Adulthood?

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The discrete emotion theory of affective aging postulates that anger, but not sadness, becomes increasingly maladaptive during older adulthood in predicting health-relevant physiological processes and chronic disease (Kunzmann & Wrosch, 2018). However, it is largely unknown whether different negative emotions have distinct functional consequences in the development of older adults' physical disease. To start examining this possibility, we investigated whether older adults' daily experiences of anger and sadness were differentially associated with two biomarkers of chronic low-grade inflammation (interleukin-6 [IL-6] and C-reactive protein [CRP]) and the number of chronic illnesses (e.g., heart disease, cancer, etc.). In addition, we examined whether such divergent associations would become paramount in advanced, as compared with early, old age. A community-dwelling study of 226 older adults (age 59 to 93; $M = 74.99$, $SD = 7.70$) assessed participants' anger and sadness over 1 week, inflammatory processes, number of chronic illnesses, and relevant covariates. Regression analysis showed that anger predicted higher levels of IL-6 and chronic illness in advanced, but not in early, old age. The age effect of anger on chronic illness was mediated by increased IL-6 levels. Sadness exerted a reversed, but nonsignificant, association with IL-6 and chronic illness, independent of age. No emotion or age effects were obtained for CRP. The study's findings inform theories of health, emotion, and life span development by pointing to the age-related importance of discrete negative emotions in predicting a major physiological pathway to physical health across older adulthood.

Keywords: sadness, anger, chronic inflammation, chronic illness, older adulthood

Functional approaches to emotion posit that specific negative emotions can exert different functions, associated with distinct behavioral and physiological signatures (e.g., Ekman, 1999; Lazarus, 1991). Extending this view, the discrete emotion theory of affective aging (DEA) postulates that the adaptive value of specific negative emotions varies across the life course (Kunzmann, Kappes, & Wrosch, 2014). This perspective assumes that anger can support overcoming blocked, but attainable, goals, while sadness may facilitate disengagement from unattainable goals. As such, anger, but not sadness, may become increasingly maladaptive and forecast health-related problems during old age; a life period characterized by a decline in personal control and a corresponding increase in intractable losses and unattainable goals (Heckhausen, Wrosch, & Schulz, 2010). Although negative emotions may compromise health particularly in older adulthood (Charles, 2010), research has yet to examine whether the experience of different negative emotions, such as anger and sadness, is differentially predictive of physical health. We explored this possibility by examining the associations between older adults' daily experiences of anger and sadness with a major physiological pathway to age-related disease (i.e., chronic low-grade inflammation, Allin & Nordestgaard, 2011) and their levels of chronic illness. Assuming that the distinct motivational responses associated with sadness and anger could differently modulate the experience of challenging life circumstances and associated health-relevant processes, we hypothesized that anger, but not sadness, would reliably predict higher levels of chronic inflammation and associated illnesses, particularly in advanced old age. These differential associations of anger and sadness with chronic inflammation and illness, by contrast, were expected to be relatively reduced in early old age.

The Discrete Emotion Theory of Affective Aging

Theories highlight that different negative emotions serve unique roles in signaling and addressing imbalances between individuals and their environments (e.g., Ekman, 1999; Frijda, Kuipers, & ter Schure, 1989; Lazarus, 1991; Nesse, 2000). These functional ap-

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proaches assume that discrete negative emotions are associated with distinct patterns of physiological activity, action impulses, and cognitive appraisals, each designed to address a specific imbalance between individuals and their environment (Kreibig, 2010). The discrete emotion theory of affective aging extends functional approaches of emotion by offering a theoretical account that integrates life span developmental theory (DEA; Kunzmann et al., 2014). This approach assumes that opportunities and constraints for pursuing personal goals change across the adult life span. While young adulthood is characterized by plenty of opportunities for overcoming goal-related problems, older adults often confront increasing irreversible losses, a reduction of personal resources, and the frequent experience of unattainable goals (Baltes & Smith, 2003). To adjust to these age-related challenges effectively, life span theory and research has shown that individuals need to adjust their motivational responses to age-related opportunities and constraints by switching from attempts to overcome goal-related challenges at younger ages to adjusting psychologically to the experience of unattainable goals in older adulthood (Heckhausen et al., 2010).

To this end, the DEA posits that discrete negative emotions may support or hinder effective age-related adaptation processes. These emotions may reflect individual differences in response to specific situations (e.g., emotional states) or across different contexts (e.g., daily, weekly, or trait-level emotions). Irrespective of these different levels of functioning, the DEA assumes that the adaptive value of discrete emotions depends on the degree to which they enable the successful management of age-specific opportunities and constraints, residing in the individual, the environment, or both (Kunzmann et al., 2014; Kunzmann & Wrosch, 2017).

This theory has focused, so far, on anger and sadness. Anger has been described as an approach-oriented affect (Carver & Harmon-Jones, 2009) that can result from obstacles to goal pursuit (Lazarus, 1991). Anger is often associated with a highly aroused physiological profile and supports persistence in overcoming goal blockages or reversing injustice (e.g., Frijda et al., 1989; Keltner & Gross, 1999; Kunzmann, Rohr, Wieck, Kappes, & Wrosch, 2017). Sadness, by contrast, is typically experienced as a response to irreversible losses (e.g., Cole & Dendukuri, 2003; Kunzmann & Grühn, 2005; Kunzmann et al., 2017), and can facilitate the reprioritization of goals and the pursuit of realistic plans either directly or through the recruitment of social support (Heckhausen, Wrosch, & Schulz, 2019; Klinger, 1975; Nesse, 2000; Wrosch & Miller, 2009).

Based on the premise that different negative emotions serve distinct functions, research on the age-related trajectories of sadness and anger has shown that the salience of anger tends to decline from young adulthood to old age, while the salience of sadness increases with age, particularly during the later parts of older adulthood when many individuals confront an increasing number of irreversible developmental losses (e.g., Kunzmann & Thomas, 2014; Kunzmann, Richter, & Schmukle, 2013; Wrosch, Barlow, & Kunzmann, 2018). The emergence of age differences in the salience of different negative emotions could further imply that sadness and anger become differentially adaptive in older adulthood, considering that psychological processes are particularly prominent during life phases when they serve an adaptive function (cf. developmental evolutionary psychology, Buss, 1995). To this end, age-related declines in opportunities and increases in constraints during older adulthood could make it possible that anger is particularly maladaptive during this life period if it fosters the continued pursuit of unattainable goals and prompts repeated failure experiences (Kunzmann et al., 2014). Sadness, by contrast, may become relatively more functional among older adults if it facilitates the effective adjustment of unattainable goals (Nesse, 2000; Wrosch & Miller, 2009). Previous research has provided preliminary support for this proposition by showing that responding with sadness, but not anger, to an emotionally neutral film clip facilitated older adults' psychological well-being (Haase, Seider, Shiota, & Levenson, 2012).

Discrete Negative Emotions and Health-Related Processes in Old Age

Negative life events and stressful experiences can elicit a host of negative emotions that can become chronic and jeopardize health behaviors (e.g., physical activity, Roshanaei-Moghaddam, Katon, & Russo, 2009) or dysregulate physiological processes in the neuroendocrine and autonomic systems (e.g., cortisol; Cohen, Janicki-Deverts, & Miller, 2007; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002), which may further modulate immune function (e.g., chronic low-grade inflammation, Miller, Cohen, & Ritchey, 2002). Thus, stress-related psychological and physiological responses may fuel chronic inflammation and place individuals at risk of developing chronic disease. In support of this argument, the dysregulation of immune function has been considered a core physiological pathway to morbidity and mortality (Ershler & Keller, 2000). This process may become particular important in older adulthood, when individuals frequently show elevated levels of chronic inflammation (Franceschi & Campisi, 2014; Piazza, Almeida, Dmitrieva, & Klein, 2010), which can contribute to the development of chronic illness, including heart disease, osteoarthritis, diabetes, or some cancers (Allin & Nordestgaard, 2011; Danesh et al., 2004).

While negative emotions are widely thought to contribute to chronic illness by linking stressful experiences and chronic inflammation (Cohen et al., 2007), most of the extant work is limited in at least three respects. First, it has focused on a dimensional view of affect, combining different negative emotions. As such, there is a paucity of research examining whether distinct negative emotions, such as anger and sadness, are equally predictive of physical disease (Kunzmann & Wrosch, 2018; Suls, 2018). Considering their distinct physiological and motivational functions (Carver & Harmon-Jones, 2009; Frijda et al., 1989; Keltner & Gross, 1999; Nesse, 2000), however, it would be plausible to assume that anger and sadness are differentially related to inflammatory processes and physical health.

Second, the small literature on the health effects of specific negative emotions is mixed, with some studies demonstrating links between the experience of anger and increased IL-6 (Carroll et al., 2011) and others finding no association with indicators of inflammation (Moons & Shields, 2015). In addition, research comparing the effects of different negative emotions frequently relies on examining trait-level emotions (Suls, 2018). However, trait-level negative emotions, as compared with more transient and fluctuating situation-specific emotional experiences, generalize across circumstances and may become disconnected from their original functions. As such, trait-level negative emotions may be frequently

maladaptive, independent of the specific emotion experienced, which could explain their widely observed adverse effects on physical disease (Cohen et al., 2007; Suls, 2018). For example, at a trait level, all negative emotions may tend to be dysfunctional, given that they are arguably decoupled from contextual demands. Thus, trait sadness and anger, which is not elicited by particular events, may serve maladaptive functions and jeopardize individuals' physical health (e.g., Glaser, Robles, Sheridan, Malarkey, & Kiecolt-Glaser, 2003). By contrast, at a state-level, the differential functions of specific negative emotions (e.g., in situations, over days, or over weeks) may become more evident, and their distinct consequences may at times be functional and, thus, not health-damaging. For example, the experience of sadness in response to irreversible losses may foster necessary psychological adaptation and ameliorate health-related physiological processes (Heckhausen et al., 2019).

Third, and perhaps most importantly, past work has not considered the possibility that the associations between discrete negative emotions and physical health may depend on an individual's age. Different from earlier life phases, older adulthood is characterized as a life phase that involves increasingly frequent experiences of intractable losses and unattainable goals (Heckhausen et al., 2010). In addition, theory and research point to older adults' increased vulnerability to the adverse health effects of emotional distress due to an age-related decrease in the organism's ability to down-regulate physiological arousal (Charles, 2010; Graham, Christian, & Kiecolt-Glaser, 2006). Although the latter argument may suggest a general age-related physiological mechanism, in which a wide range of negative emotions could increase older adults' vulnerability to inflammation and disease, our approach further considers that age-adapted emotions can help individuals to overcome age-normative problems in their daily lives. From this perspective, older adults generally encounter relatively few circumstances in which anger could contribute to overcoming emerging losses and problems with futile goals. Instead, older adults may more frequently experience situations that require them to respond with sadness to emerging challenges, fostering necessary goal adjustment and ameliorating individuals' stress experiences and associated inflammation and disease. Thus, an age-adapted experience of negative emotions, characterized by reduced anger and comparably enhanced sadness in response to situational challenges, could exert some important functions in old age. In addition, the instigation of successful adaptation processes could prevent these emotions from becoming chronic and render them less likely to affect disease-related processes (Kunzmann & Wrosch, 2018).

Considering both the generally enhanced vulnerability to the adverse health effects of negative emotions in older adulthood as well as the different motivational consequences of distinct negative emotions, we would expect an adverse association to emerge particularly between older adults' anger and their levels of chronic inflammation and physical health. By contrast, adverse effects of sadness on health-related outcomes should be considerably reduced or even reversed as long as these emotions are elicited in response to specific challenges and do not become chronic (Kunzmann & Wrosch, 2018). Importantly, however, such a differential pattern may be observed particularly in advanced old age, when many individuals confront an increasing number of intractable losses (Gerstorf et al., 2010). In early old age, by contrast, individuals are more likely to encounter still manageable challenges that could be resolved through active behavioral responses (e.g., during the onset of age-related declines; for distinguishing early from advanced old age, see Baltes & Smith, 2003). As such, in early old age, anger could at times still contribute to overcoming emerging problems, while sadness may exert a comparatively maladaptive function (Wrosch et al., 2018). A corollary of this argument is that reliable differences in the relative effects of anger and sadness on inflammatory processes and chronic illness should become paramount in advanced old age, but potentially reduced in early old age.

The Present Study

This study examined the age-related associations between older adults' daily experiences of anger and sadness with indicators of chronic low-grade inflammation (i.e., interleukin-6 [IL-6] and C-reactive protein [CRP]) and chronic illness (e.g., arthritis, cancer, or diabetes) in an age-heterogeneous sample of community-dwelling older adults. Given that anger could become increasingly maladaptive, and sadness increasingly adaptive during older adulthood, we hypothesized interaction effects to emerge between sadness and anger with age in predicting older adults' chronic inflammation and illness. That is, anger was expected to be positively associated with levels of inflammation and illness in advanced old age, but to a lesser extent in early old age. Sadness, by contrast, was expected to be unrelated to or to even predict reduced levels of chronic inflammation and illness, particularly in advanced, as compared with early, old age. Finally, considering that a dysregulation of immune function could underlie the experience of chronic disease, we hypothesized that the age-related associations between anger and sadness with chronic illness would be statistically mediated by levels of chronic inflammation.

Method

Participants and Procedures

Data were drawn from the Montreal Aging and Health Study (MAHS). The MAHS originally sampled 215 community-dwelling older adults aged from the Montreal, Quebec, Canada area. After 10 years of data collection, the MAHS sample was refreshed, and new measures that are pertinent for the present study (e.g., IL-6) were added. Consequently, only cross-sectional data from this time point were analyzed. Participants were recruited via newspaper advertisements in the Montreal area. The MAHS was approved by the institutional ethics review board. Written informed consent was obtained from participants prior to participation.

The current sample included 268 participants (96 original and 172 newly recruited participants) who were assessed in their homes or in the laboratory. Study attrition of the original participants from baseline to 10-year follow up was attributable to death ($n = 43$), refusal to participate in the study ($n = 17$), loss of contact ($n = 20$), withdrawal due to personal reasons ($n = 9$), inability to follow study directions ($n = 3$), or unknown reasons ($n = 27$). At the original baseline of the study, participants who had dropped out were not significantly different from those 96

participants who remained in the study, for most of the available study variables (i.e., sex, socioeconomic status [SES], body mass index [BMI], smoking, chronic illness, sadness, and anger; all *p*s $>$ .40). However, participants who dropped out were significantly older than those who remained in the study, $t(211.74) = 3.73$, $p < .01$.

The assessment consisted of a general questionnaire, the collection of blood drops, and subsequent daily assessment of emotions. Because we were interested in participants' normative experiences, for the daily assessment they were instructed to complete short questionnaires over the course of 1 week toward the end of three nonconsecutive typical days (days during which they did not expect extraordinary events; e.g., an unusual doctor's appointments). Sample size was determined a priori, based on power calculations reported in the associated grant proposal. The analytic sample was restricted to 226 participants (age range = 59 to 93) with available IL-6, CRP, and discrete emotions data. Six of the excluded participants had CRP scores that exceeded 10 mg/L, indicating the likely presence of acute infections (Pearson et al., 2003). Excluded participants had significantly higher levels of IL-6 ($M = 0.60$, $SD = 0.57$) and CRP ($M = 0.24$, $SD = 0.70$) than those retained (IL-6: $M = 0.12$, $SD = 0.34$; $t(7.183) = -2.41$, $p = .05$; CRP: $M = -0.06$, $SD = 0.42$; $t(24.82) = -2.10$, $p = .05$), but did not differ on any of the other main study variables (*t*s $<$ 1.13, *p*s $>$ .27).

Instrumentation

Chronic inflammation. IL-6 and CRP were measured using dried blood spots. Research assistants collected up to five drops of whole capillary blood using a finger prick with a disposable single-use lancet, and filter paper designed for this purpose (Whatman 903, GE Health care, Piscataway, NJ). The filter paper was left out to dry and then stored in a freezer. After completion of the study, the samples were analyzed in the Laboratory for Human Biology Research at Northwestern University, using a high sensitivity enzyme-linked immunosorbent assay protocol to quantify IL-6 and CRP levels. This protocol has demonstrated appropriate levels of precision, reliability, accuracy, and high correlations with serum-based results (McDade, Burhop, & Dohnal, 2004; Miller & McDade, 2012). In the present study, the averaged interassay coefficient of variation was 7.51% for IL-6 and 5.84% for CRP.

Chronic illness. Participants were asked to respond to a previously used checklist (Wrosch, Schulz, Miller, Lupien, & Dunne, 2007) that asked them to report whether they were diagnosed with 17 different common age-related chronic illnesses (e.g., cardiovascular problems, arthritis, cancer, or diabetes). Chronic illness was indexed by counting the number of medical diagnoses reported. 10.2% of participants reported no chronic illness, 20.8% reported one, 23.5% reported two, 22.6% reported three, 9.7% reported four, 6.2% reported five, and 6.9% reported six or more chronic illnesses.

Sadness and anger. Participants were asked to report the extent to which they experienced specific emotions during the day at the end of three nonconsecutive typical days. They completed the assessment within 1 week, and nonconsecutive days were chosen to decrease the likelihood that emotional experiences were related to one single event. Sadness and anger were measured with single items, using five-point Likert-type scales ranging from 0 (*very slightly or not at all*) to 4 (*extremely*). To obtain scores of discrete emotions, the sum scores of anger and sadness were separately computed across days. Positive associations were obtained across the 3 days for both anger ($r = .23$ to .42, *p*s $<$ .01) and sadness scores ($r = .54$ to .58, *p*s $<$ .01), indicating some stability of emotional experiences across days. The magnitude of these correlations indicates an intermediate level of emotional experience, in between situation-specific and trait-like emotions, and is consistent with longitudinal research (using the same methodology) showing low to moderate size positive associations over 2-year intervals (Wrosch et al., 2018).

Sociodemographic variables. Information was collected on participants' age, sex, objectively measured BMI (weight in kilograms divided by height in meters squared), smoking status (0 = no, 1 = yes), education (ranging from 0 = *no education* to 16 = *doctorate*), income (0 = less than $17,000, 1 = up to $34,000, 2 = up to $51,000, 3 = up to $68,000, 4 = up to $85,000, 5 = more than $85,000), and perceived socioeconomic status (ranging from 1 = *low* to 10 = *high* on a ladder diagram). A composite mean score of education, income, and perceived socioeconomic status (*r*s = .32 to .48, *p*s $<$ .01) was computed to obtain a reliable measure of SES. Sex, BMI, smoking status, and SES were included in the analysis as covariates to control for their possible confounding effects on inflammation and disease reported in other research (Danesh et al., 2000; Duncan et al., 2003).

Data Analysis

Prior to data analyses, values of IL-6 and CRP were log transformed to stabilize variance. The distribution of chronic illness was found to be approximately normal (skewness = 1.38, kurtosis = 3.35; Kline, 2009). Preliminary analyses were conducted to describe the sample and examine zero-order correlations among study variables. The study's hypotheses were tested using three separate multiple regression analyses, predicting levels of IL-6, CRP and chronic illness (SPSS V. 23.0; IBM Corp, 2015). These analyses followed the same procedure. Predictor variables were standardized prior to conducting the main regression analyses. Given the proportion of missing data was less than 2% on any one variable, missing scores of covariates were replaced with the sample mean. In the first step, the main effects and covariates (anger, sadness, age, sex, BMI, smoking status, and SES) were entered into the regression equation to predict individuals' levels of IL-6, CRP, or chronic inflammation. In the second step, the interaction effects of anger and age, and of sadness and age were additionally and separately entered into the model. Significant interactions were followed up by estimating the simple slopes and regions of significance of the associations between specific emotions and inflammation and health separately for individuals in early and advanced old age (i.e., +/–1 *SD* about the sample mean). Further, follow-up mediation analyses were conducted by calculating the indirect effects in bootstrap analyses (95% bias-corrected confidence interval [BCI] using 5,000 bootstraps) using PROCESS (Hayes, 2017) to determine if the significant age-related interaction effects of sadness and anger in predicting chronic illness were mediated by levels of chronic inflammation.

Results

Preliminary Analyses

Descriptive statistics are reported in Table 1. Participants were on average 75 years old ($SD = 7.81$), and approximately 62% of the sample was female. They were largely balanced across income levels (34% had an income less than $34,000, 36% had an income between $34,001 and $68,000, and 30% had an income greater than $68,000), and the sample was well educated (47% received at least a bachelor's degree). The average perceived social status was somewhat above midrange. Finally, the average BMI was in the normal range, only a minority of participants reported smoking (5%), and participants reported an average of 2.57 chronic illnesses.

The zero-order correlations between the main study variables are reported in Table 2. Of note, anger and sadness were moderately positively correlated ($r = .37$, $p < .01$), suggesting that these emotions are related, but distinct constructs. Both indicators of chronic inflammation were associated with higher BMI (IL-6: $r = .23$, $p < .01$; CRP: $r = .28$, $p < .01$), lower SES (IL-6: $r = -.16$, $p = .01$; CRP: $r = -.18$, $p < .01$), and more chronic illness (IL-6: $r = .22$, $p < .01$; CRP: $r = .13$, $p = .04$). Levels of IL-6 and CRP were moderately positively associated ($r = .40$, $p < .01$), and IL-6 levels were higher in male compared with female participants ($r = -.15$, $p < .05$), whereas CPR was associated with smoking ($r = .16$, $p < .05$). In addition, age was associated with higher IL-6 ($r = .23$, $p < .01$), more chronic illness ($r = .21$, $p < .01$), less smoking ($r = -.17$, $p = .01$), and a lower SES ($r = -.19$, $p < .01$). Finally, smoking was associated with a lower BMI ($r = -.14$, $p < .05$).

Table 1
Means, Standard Deviations, and Frequencies of Main Study Variables (N = 226)

Construct	*M* (*SD*) or %	Range
Interleukin–6 (log IL-6)	.12 (.34)	−.96–1.26
C-reactive protein (log CRP)	−.06 (.42)	−1.22–.86
Number of Chronic Illnesses	2.57 (1.92)	0–12
Anger	.62 (1.27)	0–9
Sadness	1.11 (1.94)	0–12
Age	75.00 (7.81)	59–93
Female	61.5	
Body mass index	27.01 (4.62)	14.81–43.25
Smoking	4.5	
Education		
Primary school/other	5.2	
High school	34.8	
College/trade	13.6	
Bachelor's degree	27.7	
Master's degree/PhD	18.8	
Annual income		
Less than $17,000	9.2	
$17,001–$34,000	25.1	
$34,001–$51,000	25.6	
$51,001–$68,000	10.6	
>$68,000	29.5	
Perceived social status	6.74 (1.70)	1–10

Main Analyses

The results of the regression analyses are reported in Table 3. The first step, including all covariates and main effect variables, showed significant model effects for IL-6, $F(7, 218) = 6.97$, $p < .01$, $R^2 = .18$, CRP, $F(7, 218) = 5.58$, $p < .01$, $R^2 = .15$, and chronic illness, $F(7, 218) = 2.64$, $p = .01$, $R^2 = .08$. Of the covariates, a higher BMI (IL-6: $\beta = 0.08$ $SE = .02$, $t = 3.73$, $p < .01$, $R^2 = .05$; CRP: $\beta = 0.13$, $SE = .03$, $t = 4.90$, $p < .01$, $R^2 = .09$) and being a smoker (IL-6: $\beta = 0.06$, $SE = .02$, $t = 2.99$, $p < .01$, $R^2 = .03$; CRP: $\beta = 0.09$, $SE = .03$, $t = 3.14$, $p < .01$, $R^2 = .04$) were significantly associated with higher levels of IL-6 and CRP. Finally, the main effects model showed that age was associated with IL-6 ($\beta = 0.09$, $SE = .02$, $t = 3.94$, $p < .01$, $R^2 = .06$) and chronic illness ($\beta = 0.39$, $SE = .13$, $t = 3.00$, $p < .01$, $R^2 = .04$), indicating that older, as compared with younger, participants had higher levels of IL-6 and more chronic illnesses. No other covariate or main effects emerged.

The second step of the analysis showed significant interaction effects between anger and age on levels of IL-6 and chronic illness (see Table 3; IL-6: $\beta = 0.06$, $SE = .02$, $t = 2.39$, $p = .02$, $R^2 = .02$; chronic illness: $\beta = 0.30$, $SE = .14$, $t = 2.12$, $p = .04$, $R^2 = .02$). The interaction effect between sadness and age did not predict levels of IL-6 or chronic illness, and no statistically significant interactions emerged for CRP. The significant interaction effects are plotted in Figure 1. The observed pattern suggests that participants in advanced old age, who experienced high levels of anger, exhibited the highest levels of IL-6 and chronic illnesses. Comparatively lower levels of chronic inflammation and illness were observed among young-old participants, who experienced high levels of anger, and among participants, who generally experienced low levels of anger (independent of age). Follow-up analyses of the simple slope supported this interpretation. Higher levels of anger were significantly associated with higher levels of IL-6 and chronic illness among participants in advanced old age (IL-6: $\beta = 0.10$, $SE = .03$, $t = 3.00$, $p < .01$; chronic illness: $\beta = 0.52$, $SE = .20$, $t = 2.57$, $p = .01$), but not among their relatively younger counterparts (IL-6: $\beta = -0.02$, $SE = .03$, $t = -0.41$, $p = .68$; chronic illness: $\beta = -0.09$, $SE = .19$, $t = -0.46$, $p = .64$). Conversely, a higher age was significantly associated with greater levels of IL-6 and chronic illness among individuals who experienced high levels of anger (IL-6: $\beta = 0.15$, $SE = .03$, $t = 4.40$, $p < .01$; chronic illness: $\beta = 0.72$, $SE = .20$, $t = 3.58$, $p < .01$), but not among individuals with comparatively lower levels of anger (IL-6: $\beta = 0.03$, $SE = .03$, $t = 1.09$, $p = .28$; chronic illness: $\beta = 0.11$, $SE = .19$, $t = 0.61$, $p = .54$). Further, a slope region of significance test (Preacher, Curran, & Bauer, 2006) revealed that the slope among participants in advanced old age became significant at the age of 75.00 for IL-6, and 76.60 for chronic illness. The slope for IL-6 or chronic illness did not become significant within the observed age range among relatively younger participants.

Mediation analyses were conducted to determine if the interaction of anger and age for predicting chronic illness was mediated by IL-6. The results of the mediation analyses are illustrated in Figure 2 and showed that adding IL-6 to the regression model rendered the interaction between anger and age on chronic illness nonsignificant ($\beta = 0.26$, $SE = 0.14$, $t = 1.79$, $p = .08$). In addition, they confirmed a significant indirect effect of IL-6 in explaining the interaction effect of anger and age on the number of

Table 2
Zero-Order Correlations Between Study Variables (N = 226)

Variable	1	2	3	4	5	6	7	8	9	10
1. Interleukin-6 (log IL-6)	—									
2. C-reactive protein (log CRP)	.40**	—								
3. Number of chronic illnesses	.22**	.13*	—							
4. Anger	.08	.02	.09	—						
5. Sadness	−.04	.06	−.00	.37**	—					
6. Age	.23**	.05	.21**	−.11	−.07	—				
7. Female	−.15*	.01	.02	.03	−.05	−.04	—			
8. Body mass index	.23**	.28**	.11	.04	−.12	−.03	−.07	—		
9. Smoking	.12	.16*	−.10	−.00	.08	−.17*	−.01	−.14*	—	
10. Socioeconomic status	−.16*	−.18**	−.12	−.11	−.07	−.19**	−.11	−.10	−.07	—

* $p < .05$. ** $p < .01$.

chronic illnesses (95% BCI [0.003, 0.138]). This indirect effect was significant among older participants (+1 *SD*: 95% BCI [0.055, 0.232]), but not among younger participants (−1 *SD*: 95% BCI [−0.080, 0.031]). These findings suggest that the higher number of chronic illnesses reported among participants in advanced old age who also experienced higher levels of anger, were partially mediated by their elevated levels of IL-6.[1]

Discussion

This study showed in a sample of community-dwelling older adults that the association between the daily experience of anger with levels of chronic inflammation and illness was moderated by participants' chronological age. In particular, the findings indicated that anger was related to higher levels of IL-6 and chronic illness, but only among individuals in advanced, and not early, old age. Further, individuals' IL-6 levels mediated the age-related association between anger and chronic illness. Sadness, by contrast, showed a negative, but nonsignificant, association with levels of IL-6 and chronic illness across older adulthood, but did not exert age-differential effects.

The reported results contribute to the extant literature on emotion and health by highlighting the distinct roles of anger and sadness for predicting chronic inflammation and disease in older adulthood More specifically, they suggest that anger, but not sadness, was associated with higher levels of IL-6 and chronic illness in advanced old age. Further, the age-related associations between anger and IL-6 remained significant when controlling for a number of sociodemographic and health-relevant covariates (see Table 3) as well as trait-like high arousal negative affect (e.g., hostile, afraid, or upset) Footnote 1). These results suggest that the divergent effects of these two negative emotions on chronic inflammation and illness across older adulthood may not be due to the generalized experience of negative affect or other measured covariates. They are further consistent with the theoretical claims that anger and sadness can exert different age-related functions, and that health effects of these two discrete negative emotions may depend on individuals' age-related opportunities and constraints for addressing developmental challenges (Kunzmann et al., 2014). However, we note that our study did not explicitly examine age-related opportunities and constraints and only included a trait-like measure of high-arousal negative affect. As such, it did not include a trait-measure of sadness, and future research is needed to replicate the observed findings and substantiate our conclusions. Such research may examine specific emotion-by-situation interactions and corresponding trait measures of emotions to disentangle health effects of situation-specific negative emotions from the corresponding experience of generalized negative affect.

The study's results may inform research postulating that anger could contribute to motivational attempts aimed at overcoming pressing problems and blocked goals (Carver & Harmon-Jones, 2009; Frijda et al., 1989; Keltner & Gross, 1999), whereas sadness is likely to facilitate adjustment to unattainable goals by promoting effective goal disengagement processes (Klinger, 1975; Nesse, 2000; Wrosch & Miller, 2009). In particular, toward the end of life, many individuals are likely to encounter a steep increase in intractable developmental losses across different areas of life (Baltes & Smith, 2003; Gerstorf et al., 2010). Here, the experience of anger may become particularly dysfunctional for effectively adjusting to uncontrollable age-related declines if it interferes with adaptive motivational processes by promoting persistence instead of needed adjustment to unattainable goals (Wrosch, Scheier, & Miller, 2013). In addition to targeting older adults' generally enhanced vulnerability to the health-related consequences of negative emotions (Charles, 2010), anger may prolong stressful circumstances and facilitate a dysregulation of neuroendocrine processes in advanced old age, which could have significant implications for individuals' inflammatory processes and chronic illness (Cohen et al., 2007). In early old age, by contrast, individuals are more likely to experience the onset of age-related problems that may still be partially controllable. Here, the motivational concomitants of anger (e.g., persistence) may allow individuals to effectively counteract emerging age-related losses more frequently and ameliorate stressful experiences, thereby preventing adverse effects of challenges on chronic inflammation and disease.

[1] Additional analyses were conducted to determine the impact of trait-level negative affect on the reported results. To this end, our study included a measure of high-arousal negative affect, experienced during the last year (e.g., hostile, afraid, or upset), which has been shown trait-like stability (Watson, Clark, & Tellegen, 1988). When negative affect was included in the analyses, the obtained interaction effects (IL-6: ß = 0.06, $SE = .02$, $t = 2.57$, $p = .01$, $R^2 = .02$; chronic illness: ß = 0.28, $SE = .14$, $t = 1.96$, $p = .05$, $R^2 = .02$) and mediation effect remained significant (overall: 95% BCI [0.002, 0.075]; older participants: 95% BCI [0.006, 0.132]; younger participants: 95% BCI [−0.043, 0.019]).

Table 3
Multiple Regression Analyses Predicting Chronic Low-Grade Inflammation (IL-6), C-Reactive Protein (CRP), and Chronic Illness

	Interleukin-6 (log IL-6)			C–reactive protein (log CRP)			Chronic illness		
Predictors	ß (*SE*)	*t*	*p*	ß (*SE*)	*t*	*p*	ß (*SE*)	*t*	*p*
Step 1									
Intercept	.12 (.02)	5.56**	.00	−.06 (.03)	−2.38**	.00	2.57 (.13)	20.64**	.00
Female	−.05 (.02)	−2.17*	.03	.01 (.03)	.41	.68	.04 (.13)	.32	.75
SES	−.03 (.02)	−1.37	.17	−.05 (.03)	−1.76	.08	−.13 (.13)	−.97	.33
BMI	.08 (.02)	3.73**	.00	.13 (.03)	4.90**	.00	.19 (.13)	1.49	.14
Smoking	.06 (.02)	2.99**	.00	.09 (.03)	3.14**	.00	−.10 (.013)	−.81	.42
Age	.09 (.02)	3.94**	.00	.03 (.03)	1.16	.25	.39 (.13)	3.00**	.00
Anger	.04 (.02)	1.81	.07	−.01 (.03)	−.47	.64	.20 (.14)	1.47	.14
Sadness	−.02 (.02)	−1.07	.29	.04 (.03)	1.33	.19	−.02 (.14)	−.18	.85
Step 2									
Age × Anger	.06 (.02)	2.39*	.02	.02 (.03)	.61	.54	.30 (.14)	2.12*	.03
Age × Sadness	.01 (.02)	.42	.68	.02 (.03)	.55	.58	.06 (.13)	.44	.66

Note. The interaction effects were tested in separate models. SES = socioeconomic status; BMI = body mass index.
* $p < .05$. ** $p < .01$.

The reported results further revealed that the age-related association between anger and chronic illness was statistically mediated by older adults' levels of IL-6. This pattern of results demonstrates that the stronger effect of anger on higher levels of chronic illness in advanced, as compared with early, old age was partially explained by participants' elevated levels of IL-6. It is further consistent with the notion that IL-6 represents a major physiological pathway to several age-related diseases, including heart disease, osteoarthritis, diabetes, or cancer (Allin & Nordestgaard, 2011; Danesh et al., 2004). Note, however, that these results were not replicated for predicting CRP. This may be the case because our study showed only a moderate correlation between CRP and IL-6 ($r = .40$ see Table 2). While the strength of the relation between these two biomarkers can depend on the physiological context (Czarkowska-Paczek et al., 2005), associations of similar magnitude have been shown in other community-dwelling samples of older adults (Puzianowska-Kuznicka et al., 2016). In addition, IL-6 appears to be more strongly related to affective symptoms than CRP (Howren, Lamkin, & Suls, 2009), which could imply that IL-6 is a particular potent mediator for linking negative emotions and chronic illness. Finally, we acknowledge that the obtained differential associations between age-related negative emotions and different markers of inflammations could be due to measurement error. In this regard, we note that IL-6, but not CRP, was positively correlated with age in our sample (see Table 2). Since research has shown that chronic inflammation typically increases with age (Franceschi & Campisi, 2014; Piazza et al., 2010), our measure of CRP may have incorporated some measurement error, which would explain its weaker association with participants' age and emotional experiences. Thus, future studies should obtain multiple samples of biomarkers to address measurement error in their analyses.

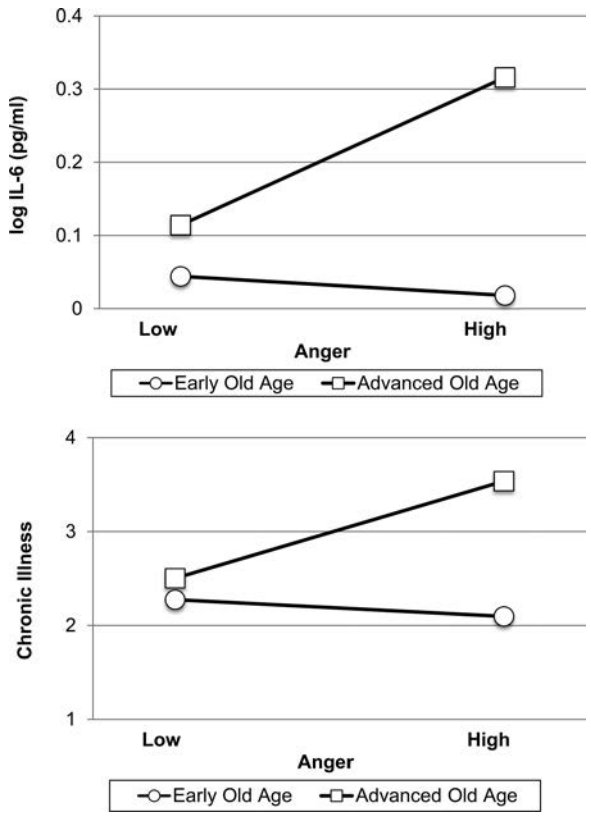

Figure 1. The association between anger and levels of interleukin-6 (IL-6; upper panel), and number of chronic illnesses (lower panel), separately for individuals in early old age (−1 *SD*) and advanced old age (+1 *SD*). High and low anger is plotted at one standard deviation above and below the sample mean.

Note that the obtained age-related associations between anger, IL-6, and chronic illness were based on cross-sectional data, which could lead to alternative interpretations of the data. One issue with cross-sectional data frequently relates to possible "third variables" that could explain associations with health-related variables (e.g., neuroticism, Watson & Pennebaker, 1989). Since our study included objectively measured health-relevant processes, however, we minimized the emergence of method overlap and consider this possibility as relatively unlikely. Nonetheless, chronic illness could also trigger certain negative emotions and associated chronic inflammation. In addition, such a process may be observed to a larger extent in advanced old age when physical health problems

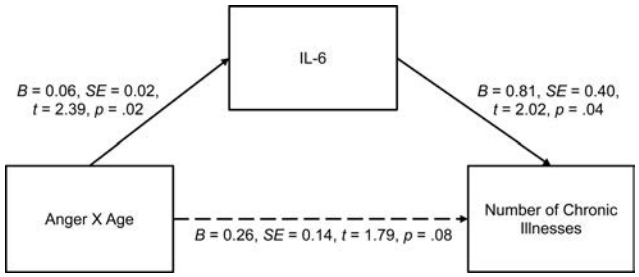

Figure 2. The indirect effect of interleukin-6 (IL-6) in the association between the interaction effect of anger and age with the number of chronic illnesses.

typically become more severe. We think that this possibility is plausible as well and does not compromise our interpretation of age-related effect of discrete negative emotions and health-related variables. In fact, theoretical models in life span developmental psychology have conceptualized bidirectional and reciprocal associations between negative emotions and health, and recent work suggests that such an association can increase with advancing age (Barlow, Wrosch, Heckhausen, & Schulz, 2017; Kunzmann, Schilling, et al., 2018; Wrosch, Schulz, & Heckhausen, 2004). Thus, the observed age-related link between anger, inflammation, and chronic illness could be triggered or become aggravated by disturbances in each of the processes involved and result in a downward spiral that compromises both older adults' emotional experience and their health.

Of note, although anger was negatively correlated with age in our study, this association did not reach statistical significance. Considering that past research has documented that older adults experience less anger than young adults (for a review, see Kunzmann & Wrosch, 2017), the latter finding seems somewhat inconsistent with this extant literature. However, it should be noted that a study including middle-aged adults demonstrated that anger may begin to decline in early midlife and can rebound slightly in older adulthood (Kunzmann et al., 2013). Therefore, it may be that a nonlinear relation between anger and age has blunted the association, or that the levels of anger have already begun to plateau in our sample of community-dwelling older adults.

Further, it is important to address that sadness showed a negative, but nonsignificant, association with older adults' levels of IL-6 and chronic illness. The absence of an association between sadness and health-relevant outcomes does in our view not rule out the possibility that sadness could have exerted some beneficial health functions in our study. Given that older adults are generally vulnerable to the physiological and health consequences of negative emotions (Charles, 2010), the experience of sadness may have rendered such an adverse process less health-disruptive by enabling older adults to psychologically adjust to uncontrollable age-related challenges (Wrosch et al., 2013). This possibility would be consistent with our data in that anger, but not sadness, predicted enhanced inflammation and chronic disease in advanced old age.

We also note that our theoretical framework expected that sadness could be relatively more adaptive during the later, as compared with earlier, parts of older adulthood. The reported data, however, did not support this hypothesis. In this regard, it could be possible that different types of sadness-related phenomena (e.g., ordinary feelings of sadness vs. chronic sadness or depression, cf. Andrews & Thomson, 2009) exerted opposing effects on health-relevant outcomes in our study. For example, it has been argued that although feelings of sadness could provide some benefit to well-being and health by facilitating adaptive goal disengagement processes, the experience of chronic feelings of sadness could contribute to undesirable outcomes by depleting a person's motivational resources altogether or trigging dysfunctional health behaviors (Heckhausen et al., 2019; Seligman, 1975; Wrosch & Miller, 2009). Such opposing health effects may have prevented the detection of age effects for sadness in our study. In fact, steep reductions in well-being, including depressive symptomatology, have been reported as individuals enter advanced old age (Dunne, Wrosch, & Miller, 2011), and may have counteracted potential age-related associations of ordinary sadness. To shed further light on these possibilities, more research is needed to reveal the circumstances in which sadness and anger exert unique effects on individuals' health. We feel that such work is warranted and likely to illuminate the consequences of discrete emotional experiences on health-relevant outcomes across the adult life span.

Limitations and Future Directions

To the best of our knowledge, the present study is unique in providing a theory-based investigation of the divergent age-related effects of anger and sadness on a major physiological pathway to chronic illness. Indeed, research examining whether different negative emotions can exert varying effects on physical health outcomes has only begun, and often lacks a theoretical foundation (Kunzmann & Wrosch, 2018; Suls, 2018). Although the reported findings lend support to a life span perspective on discrete emotions and health-relevant outcomes, more theoretical and empirical work is needed to substantiate our conclusions. Here, we address the study's limitations and outline future research that may be needed to arrive at a comprehensive theory of discrete emotional experiences and developmental health outcomes across the adult life span.

First, the study's results were drawn from cross-sectional data, making it impossible to determine causality. Future research should therefore replicate our findings using longitudinal and experimental designs. Second, our study asked participants to report their daily emotional experiences at the end of 3 days during 1 week. This approach should be complemented by studies using ecological momentary assessment of emotions (Shiffman, Stone, & Hufford, 2008), as well as measuring other emotion response systems (e.g., emotional expression and physiology; Levenson, 2000), trait-level emotions, and personality factors (e.g., neuroticism) that could influence the experience of emotions and health outcomes. Such work could reduce biases in the report of emotions and establish whether diverging effects of different negative emotions on inflammatory processes and health may generalize across different emotion response systems and varying stability of emotional reactions. In addition, it may identify important individual difference variables that could influence the situation-specific experience of negative emotions and exert associated effects on individuals' health. Third, potential mediators of the link between different negative emotions and chronic inflammation and illness should be studied. Such research may be capable of illustrating the

entire process including individuals' emotional experiences in specific contexts, their mediating behavioral and physiological responses, and long-term health outcomes. For example, our theoretical model assumes that certain motivational processes (i.e., persistence vs. disengagement, Heckhausen et al., 2010; Wrosch et al., 2013) and states of psychological and physiological stress (e.g., cortisol output, Cohen et al., 2007) could explain the different links between specific negative emotions and health-related outcomes. Fourth, the present study was restricted to older participants and future research should broaden the age range of inquiry. Since opportunities for overcoming developmental challenges change significantly from young adulthood to old age (Heckhausen et al., 2010), such research may determine stronger age effects of discrete negative emotions on emerging physiological risk factors of physical disease. Fifth, our approach considered age as a proxy for reductions in individuals' opportunities to overcome common developmental challenges. Future research should therefore assess age-related opportunity structures more directly by studying individuals' particular problems and exposure to stressors as well as the likelihood they could overcome them. Sixth, research should improve measurement by distinguishing ordinary sadness from chronic and severe feelings of sadness. There may be a tipping-point, when extreme and lasting negative emotional responses associated with depression reverse some of the potentially adaptive functions of sadness and result in maladaptive behavioral and health outcomes (Wrosch & Miller, 2009). Finally, the study examined only sadness and anger because our theoretical model has focused thus far on the age-related functions of these two discrete emotions. Nonetheless, other negative emotions and different positive emotions may also serve divergent age-related functions. More theoretical work and subsequent empirical research is needed to illuminate the effects of a variety of discrete emotions on psychological processes, specific physiological pathways, and physical health outcomes across the life span.

Conclusion

The present study showed that the daily experience of anger, but not sadness, predicted elevated levels of low-grade inflammation and chronic illness in advanced, but not early, old age, when many individuals encounter a steep increase in insurmountable developmental losses. In addition, it suggested that the age-related effects of anger on chronic illness were partially explained by individuals' levels of IL-6. These findings support propositions from a discrete emotion approach to physical health (Kunzmann & Wrosch, 2018) by documenting divergent effects of different negative emotions on a major physiological pathway to a number of age-related diseases. Negative emotional experiences, such as anger, may exert strong effects on physical health if they are mismatched with the declining opportunities and motivational affordances of older adulthood. By contrast, other negative emotional experiences, such as sadness, could facilitate psychological adjustment to irreversible age-related losses in old age and buffer older adults' increased vulnerability to the health effects of emotional distress (Charles, 2010). A discrete emotion approach to physical health across the adult life span may thus represent a promising theoretical model that is capable of addressing both the potential benefits and adverse effects of different negative emotions.

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Correction to Carpenter and Schacter (2018)

In the article "Flexible Retrieval Mechanisms Supporting Successful Inference Produce False Memories in Younger but Not Older Adults," by Alexis C. Carpenter and Daniel L. Schacter (*Psychology and Aging*, 2018, Vol. 33, No. 1, pp. 134–143, http://dx.doi.org/10.1037/pag0000210), the script that was used to calculate the trial counts was found to be incorrect when the experiments reported in the article were reanalyzed. Thus, although the statistical conclusions of the article have not changed, data reported throughout the Results section and in Tables 1 and 2 were incorrect. The online version of this article has been corrected.

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