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Anger Expression, Momentary Anger, and Symptom Severity in Patients with Chronic Disease

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Abstract

Background—Anger expression styles are associated with physical health, and may affect health by modulating anger experience in daily life. Research examining this process in the daily lives of clinically relevant populations, such as patients with chronic disease, is needed.

Method—Community adults with asthma (N=97) or rheumatoid arthritis (RA; N=31) completed measures of trait-level anger expression styles (anger-in and anger-out), followed by ecological momentary assessments of anger and physical health 5 times daily for 7 days.

Results—High anger-in predicted greater momentary anger, physical limitations, and greater asthma symptoms. High anger-out predicted *reduced* RA symptoms. Momentary anger was robustly associated with more severe symptoms in daily life. Three-way interactions showed anger-in moderated these momentary anger-symptom associations more consistently in men.

Conclusions—Anger expression styles, particularly anger-in, may affect the day-to-day adjustment of patients with chronic disease in part by altering the dimensions of everyday anger experience, in ways that appear to differ by gender.

Keywords

Anger Expression and Suppression; Asthma; Arthritis; Ecological Momentary Assessment; Gender Differences

Frequent suppression (*anger-in*) and frequent expression (*anger-out*) of angry emotion have each been associated with a wide range of health complications, including cardiovascular disease (1, 2), respiratory illness and musculoskeletal problems (3), and pain disorders (4, 5). Anger-in and anger-out represent two broad styles of anger expression describing how people manage or regulate anger arousal (6). Despite close conceptual links between anger expression styles and anger experiences, few studies have examined their interplay in the daily lives of patients with chronic illness, for whom the dynamic regulation of anger experiences in daily life may play an important role in disease adjustment. The current study uses ecological momentary assessment (EMA) to examine these issues.

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Compliance with Ethical Standards

The authors have no actual or potential conflicts of interest to report. All procedures were approved by both university and hospital institutional review boards for human research. Informed consent was obtained from all participants.

Anger expression styles characterized by suppression or outward anger expression may affect health insofar as they may represent ineffective emotion regulation strategies (7, 8), with their habitual use potentially leading to anger dysregulation, perhaps evidenced by more frequent and more severe/intense anger episodes. For patients with asthma and arthritis, the experience of frequent and intense anger episodes may lead to greater symptom expression and increased physical limitations (e.g., via inflammatory processes relevant to both diseases, such as interleukin-6 and immune complement C3; see 9, 10). The research base for the interplay between anger expression styles, momentary anger experience, and disease adjustment in the daily lives of patients with chronic disease is small, so it is not well understood how anger expression styles may influence the dimensions of momentary anger experiences.

The relationship between trait anger expression style and health may best be described as a *transactional process*, whereby trait anger expression leads to health problems by: (i) increasing a person's frequency and/or severity of anger arousal (i.e., greater "exposure" to one's own anger), which may lead to more health problems; and (ii) increasing sensitivity to anger experiences in the moment, leading patients to experience greater symptom "reactivity" to anger arousal (for examples of transactional processes, see refs 11, 12). Evidence for the "exposure" pathway comes from studies showing that trait anger expression styles predict greater frequency of anger experiences in people's everyday lives (13) as well as evidence that both experimentally induced and naturally occurring anger experiences affect diverse physical parameters such as blood pressure, heart rate, and pain experiences (14–16).

Evidence for the "reactivity" pathway, however, comes primarily from experimental findings suggesting that associations between anger experiences and momentary physiological parameters are stronger for individuals with high trait anger-in (17) as well as for those with high trait anger-out (18–20). For example, early laboratory work (17) found that harassment conditions had the strongest effects on cardiovascular reactivity for men with high anger suppression and high trait anger. Another study (18) showed that those with high anger-in as well as those with high anger-out showed greater cardiovascular reactivity to experimental harassment, but only if they were instructed to use their "non-preferred" mode of anger expression (i.e., if individuals with high anger-out were instructed to suppress anger or if individuals with high anger-in were instructed to express anger). More recent studies are extending this research base by using ambulatory assessment designs, such as daily diary and EMA, in which individuals repeatedly provide data as they navigate their daily lives (typically responding daily in diary designs and multiple times per day in EMA). These designs provide for a more nuanced understanding of temporal dynamics, reduced recall bias in self-reports, and enhanced ecological validity (21–23). For example, a large daily diary study of women with fibromyalgia (24) found that patients with high trait anger-out experienced less end-of-day pain, but only if they reported using anger expression (anger-out) in response to an anger-inducing situation that day. An EMA study (16) found that momentary behavioral anger expression predicted subsequent pain intensity, but the lagged effects from momentary anger expression to pain were not moderated by trait anger-in or trait anger-out. Finally, a recent paper (25) showed that state anger arousal and momentary anger expression predicted subsequent pain and pain-related interference, again showing a

lagged effect, whereas momentary anger inhibition showed only concurrent associations with pain interference and decreases in function.

Although such studies provide valuable information about the health-related effects of anger arousal and anger expression, no study has yet concurrently tested whether trait-level anger-in and anger-out (a) predict greater frequency and intensity of momentary anger arousal or experience, (b) predict greater momentary symptom severity and physical limitation, and (c) moderate momentary associations between anger arousal and momentary health indicators in the daily lives of patients with chronic disease. In addition to providing empirical evaluation of the transactional model described above, these tests may have clinical application by helping us understand *who* is at risk (via anger expression styles), as well as *when* they are most at risk (when they are angry versus not angry) for experiencing increased symptom severity and physical limitation in daily life. This information may inform for standard clinical care, but also holds value for the design and implementation of personalized interventions, tailored both to the person as well as the moment, and sensitive to the interaction between the two.

The current study uses EMA to obtain repeated measures of anger experience and momentary health indicators (disease-specific symptom severity, and physical limitations due to symptoms) in patients with asthma or rheumatoid arthritis (RA). Five hypotheses were tested in the current study. First, we expected that individuals with high trait anger-in or anger-out would experience more frequent and more severe anger episodes in their daily lives compared to people low in these dimensions. Second, we expected that people high in trait anger-in and trait anger-out would also show greater momentary negative affect (excluding anger) and less momentary positive affect than people low on these dimensions. Third, we expected that people with high versus low trait anger-in or anger-out would experience more disease-specific symptoms, as well as greater symptom interference and physical limitation, in their daily lives. Fourth, we expected that patients would show increased severity of physical limitations and disease-specific symptoms during moments when they experienced anger, compared to themselves when they did not experience anger. Fifth, we expected that patients with high versus low trait anger-in or anger-out would show greater sensitivity to anger experiences—that is, stronger momentary associations between angry affect and momentary health indicators. Because numerous studies report gender differences in relationships between anger expression styles, momentary anger, and health (17, 26–28), we tested moderating effects of gender in an exploratory manner.

Method

Participants

The data used in this report were drawn from the baseline measurement period of a larger intervention study that utilized EMA to assess momentary well-being, stress, mood, pain expression, social support, coping, and health behaviors in a sample of 128 community participants with a physician-verified diagnosis of either asthma ($n=97$) or RA ($n=31$). The larger study examined the relationship of an expressive writing emotional regulation intervention on the health status of asthmatic and arthritic symptoms; again, all data used in

this report are from baseline (pre-randomization and prior to any intervention). For the procedures and data reported on herein, participants were compensated \$50.

Procedure

Individuals diagnosed with asthma or RA were recruited for a study on the relationship of daily experiences to health and well-being in persons diagnosed with chronic illness. After providing informed consent, participants completed self-report questionnaires that evaluated a range of psychological, social, and disease-related factors. Participants were also trained on how to provide EMA assessments on a palmtop computer. Subsequently, participants followed a signal-contingent design EMA (i.e. providing reports when notified by a schedule of beeps) five times a day for one week (i.e., up to 35 assessments per patient). When prompted, participants reported momentary experiences (mood, activities, location, interaction with others, stressful occurrences, symptom interference/restrictions, disease-specific symptom severity, peak flow ratings for patients with asthma, use of medication and consumption of caffeine, alcohol and tobacco). Additional details of the study procedure are also described elsewhere (29).

Baseline Measures

Anger expression styles were assessed using the Anger-In and Anger-Out scales of the State-Trait Anger Expression Inventory (STAXI)(6). The Anger-In scale indicates the frequency with which angry feelings are held in or suppressed; the Anger-Out scale measures the frequency with which angry feelings are expressed outwardly, toward other people or objects in the environment. Each scale consists of 8 items. Cronbach alphas were 0.77 and 0.70 for anger-in and anger-out scales, respectively. Anger-in and anger-out were centered on their sample means ($M_s=15.9$ and 14.5 , respectively) in all OLS and multilevel regression analyses.

EMA Measures

Anger experiences were measured as part of the Positive and Negative Affect Schedule (PANAS; 30) at each EMA beep. Participants rated their current level of anger (along with 4 other negative and 4 positive emotions) on a seven-point rating scale ranging from 0 (not at all) to 6 (extremely). The raw anger rating scale (ranging from 0–6) was used as the measure of *momentary anger severity*. A measure of *momentary anger experiences* (0=not angry, 1=angry) was created by dichotomizing the raw anger severity score such that ratings of 0 corresponded to non-angry moments and ratings of 1 to 6 (i.e., any anger reported) corresponded to angry moments and were recoded to 1. Finally, an *anger frequency* score was created by (a) taking the average of the momentary anger experience measure (0=not angry, 1=angry) for each person, which corresponds to the proportion of EMA occasions each person reported being angry (versus not), and (b) multiplying this proportion by 100 to yield a percentage. EMA reports where anger information was not provided were treated as missing and were not included in the calculation of the anger frequency score.

Positive and Negative Affect were also measured as part of the PANAS (30) at each EMA beep, with participants rating their current level of each affective state on a 0 (not at all) to 6 (extremely) scale. Positive affect was the momentary average of participants' ratings across

the adjectives *happy, joyful, (experiencing) enjoyment, and pleased* (alpha at the measurement occasion level was 0.92). Negative affect was the momentary average of participants' ratings across the adjectives *frustrated, worried, depressed, and unhappy* (alpha at the measurement occasion level was 0.82). Anger is typically included as one of the items of the PANAS negative affect scale; however, for the current study anger was removed to avoid inflating associations between anger expression styles and negative affect.

Physical Limitations were reported at each EMA beep. All participants answered the following questions (with only the appropriate disease classification indicated) scaled from 0 (not at all) to 6 (extremely): "How much did your [asthma or arthritis] interfere with your daily routine since the last beep?" and "How much did your [asthma or arthritis] force you to restrict activities since the last beep?" These 2 items were highly correlated ($r = .90, p < .001$) and were averaged to create a measure of physical limitations (the two items demonstrated good internal consistency at the measurement occasion level, .86).

Disease-specific symptoms were also assessed at each EMA beep. Participants with asthma answered reported symptoms (i.e. "How bad was your coughing/wheezing since last beep?") on a scale from 0 (not at all) to 6 (extremely). Patients with RA answered the following questions: "Rate severity of stiffness as you were beeped"; "Rate severity of pain as you were beeped"; "Rate severity of joint tenderness/swelling as you were beeped" on a scale from 0 (not at all) to 6 (extremely). These items were averaged together to form a composite index of momentary RA symptom severity (the three items demonstrated good internal consistency at the measurement occasion level, .85).

Statistical Analyses

Descriptive Statistics—We calculated descriptive statistics for sample characteristics (ethnicity, age, disease status, and trait anger expression styles) and momentary reports (anger experiences, negative and positive affect, physical limitations, and disease-specific symptoms). Descriptive statistics for EMA reports are sample averages of individual-level means for each measure. Gender differences in sample characteristics and EMA reports were tested using independent samples *t*-tests for continuous outcomes and chi-squared tests of independence for categorical outcomes. Overall, participants completed 81.3% of EMA reports.

Statistical Modeling

Overview—Study hypotheses were tested using ordinary least squares (OLS) regressions for outcomes that varied only at the between-person level (anger frequency) and multilevel models (MLM) for outcomes that varied both between and within people (anger severity, positive and negative affect, physical limitations, and disease-specific symptoms). Unadjusted and covariate adjusted models were estimated for each of these outcomes, and separate models were run with anger-in and anger-out as predictors. All covariate adjusted models included the following covariates: sample characteristics—gender, age (sample mean centered), ethnicity, and disease group (asthma or RA). Covariate-adjusted multilevel models also included within-person covariates for weekend (where Friday, Saturday, or Sunday=1 and Monday through Thursday=0) and within-day EMA "beep" number (where

1=morning, 5=evening, corresponding to the 5 reports across the day). Effect size estimates are provided using semipartial r^2 (r^2_{sp}) in OLS regression models, and r effect size estimates (r_{effect}) in multilevel models (see p. 441 in reference 31). Denominator df for within-person variables were conservatively calculated using the N of subjects rather than the N of observations (see ref 32).

Multilevel models testing main effects of anger expression styles—Equation 1 shows an example MLM testing the main effect of anger expression style on anger severity.

$$AngSev_{ij} = \beta_0 + \beta_1 * (AES_j) + u_{0j} + e_{ij} \quad (1)$$

In equation 1, anger severity at time i for person j is predicted by the population intercept β_0 , a random effect for the intercept that varies at the person level (u_{0j}), anger expression style for person j (anger-in or anger-out, AES_j), and an assessment-specific residual term (e_{ij}). Models of the same form as equation 1 were also estimated for physical limitations, asthma symptoms, and RA symptoms as outcomes to test the first 3 hypotheses.

Multilevel models testing associations between momentary anger and disease adjustment—Equation 2 shows an example MLM testing the concurrent association between momentary anger experiences (0=not angry, 1=angry) and physical limitations.

$$PhysLim_{ij} = \beta_0 + \beta_1 * (Anger_{ij}) + \beta_2 * (AngerFreq_j) + u_{0j} + e_{ij} \quad (2)$$

In equation 2, physical limitations at time i for person j are predicted by a population average intercept (β_0), a random effect for the intercept (u_{0j}), the within-person effect describing the change in physical limitations across angry versus non-angry moments ($\beta_1 * (Anger_{ij})$), and a between-person covariate measuring individual differences in anger frequency ($\beta_2 * (AngerFreq_j)$) which, when added to the model, removes the between-person variance in the $Anger_{ij}$ variable and ensures its associated β_1 coefficient describes a within-person association (33). Anger frequency was centered at its sample mean. Models of the same form as equation 2 were also estimated for asthma and RA symptoms and used to test our fourth hypothesis.

Multilevel models testing interactions between anger expression styles and momentary anger—To test whether the momentary associations between anger and symptom-related outcomes (physical limitations, coughing/wheezing, and RA symptoms) differ based on anger expression styles, interaction terms between momentary anger experiences and anger expression styles were estimated as depicted in Equation 4.

$$PhysLim_{ij} = \beta_0 + \beta_1 * (Anger_{ij}) + \beta_2 * (AngerFreq_j) + \beta_3 * (AES_j) + \beta_4 * (AES_j \times Anger_{ij}) + u_{4j} + e_{ij} \quad (3)$$

The β_4 parameter in Equation 3 tests whether the momentary relationship between anger experiences and physical limitations is stronger for patients with high anger-in (or high anger-out). Gender differences were tested by adding Gender, Gender $\times$ AES, Gender $\times$

Anger, and Gender $\times$ AES $\times$ Anger terms to the model in Equation 3. These models were used to test our fifth hypothesis.

Results

Table 1 shows descriptive statistics for sample characteristics and momentary reports, both for the full sample and split by gender. No significant differences were found between men and women across any of the study variables. Compared to patients with asthma, patients with arthritis were older (arthritis $M_{age}=50.0$ years, asthma $M_{age}=42.3$ years, $t(126)=2.71$, $p=0.008$), reported less positive affect (arthritis $M=2.50$, asthma $M=2.82$, $t(73.69)=-2.10$, $p=0.039$), and more physical limitations (arthritis $M=2.16$, asthma $M=1.08$, $t(115)=4.49$, $p<0.001$). No other differences were found between patients with asthma versus RA.

Hypothesis 1: Trait Anger Expression Styles will Predict Increased Momentary Anger (Both Frequency and Severity)

Anger frequency—OLS Regression models showed that trait anger-in predicted more frequent momentary anger in daily life (*unadjusted* $b=2.53$, $SE=0.67$, 95% CI: 1.21, 3.86, $p<0.001$, $r^2_{sp}=0.12$; *adjusted* $b=2.59$, $SE=0.68$, 95% CI: 1.24, 3.94, $p<0.001$, $r^2_{sp}=0.12$). Trait anger-out did not predict momentary anger frequency (*unadjusted* $b=0.72$, $SE=0.84$, 95% CI: -0.94 , 2.39, $p=0.39$, $r^2_{sp}=0.007$; *adjusted* $b=0.40$, $SE=0.87$, 95% CI: -1.33 , 2.13, $p=0.65$, $r^2_{sp}=0.002$). We found no evidence for gender differences in these associations.

Anger severity—Multilevel regression models showed that trait anger-in predicted greater severity of momentary anger (*unadjusted* $b=0.07$, $SE=0.01$, 95% CI: 0.05, 0.10, $p<0.001$, $r_{effect}=0.45$; *adjusted* $b=0.07$, $SE=0.01$, 95% CI: 0.05, 0.10, $p<0.001$, $r_{effect}=0.46$) whereas trait anger-out did not predict anger severity (*unadjusted* $b=0.02$, $SE=0.02$, 95% CI: -0.01 , 0.06, $p=0.24$, $r_{effect}=0.11$; *adjusted* $b=0.02$, $SE=0.02$, 95% CI: -0.02 , 0.05, $p=0.37$, $r_{effect}=0.09$). Additionally, model results showed evidence for a significant two-way interaction between trait anger-in and gender predicting momentary anger severity (*unadjusted* interaction $b=-0.07$, $SE=0.03$, 95% CI: -0.12 , -0.01 , $p=0.017$, $r_{effect}=0.23$; *adjusted* interaction $b=-0.07$, $SE=0.03$, 95% CI: -0.13 , -0.02 , $p=0.012$, $r_{effect}=0.24$). Simple slopes analyses from the covariate-adjusted model showed that the relationship between anger-in and momentary anger severity was stronger for men ($b=0.12$, $SE=0.02$, 95% CI: 0.07, 0.16, $p<0.001$, $r_{effect}=0.47$) than for women ($b=0.05$, $SE=0.02$, 95% CI: 0.01, 0.08, $p=0.012$, $r_{effect}=0.24$), but was nonetheless significant for both genders. For men with high trait anger-in, predicted anger severity was 1.19 versus 0.26 for men with low trait anger-in. For women with high trait anger-in, predicted anger severity was 0.76 versus 0.40 for women with low trait anger-in. No evidence was found for an interaction between gender and anger-out.

Hypothesis 2: Trait Anger Expression Styles will Predict Increased Momentary Positive and Negative Affect

Negative affect—Multilevel models showed that trait anger-in predicted higher momentary negative affect (*unadjusted* $b=0.10$, $SE=0.02$, 95% CI: 0.06, 0.13, $p<0.001$,

$r_{\text{effect}}=0.48$; *adjusted* $b=0.09$, $SE=0.02$, 95% CI: 0.06, 0.13, $p<0.001$, $r_{\text{effect}}=0.48$). Anger-out did not significantly predict momentary negative affect (*unadjusted* $b=-0.01$, $SE=0.02$, 95% CI: -0.05, 0.03, $p=0.66$, $r_{\text{effect}}=0.04$; *adjusted* $b=-0.01$, $SE=0.02$, 95% CI: -0.06, 0.03, $p=0.56$, $r_{\text{effect}}=0.06$). These relationships did not differ by gender.

Positive affect—Neither trait anger-in (*unadjusted* $b=-0.04$, $SE=0.02$, 95% CI: -0.08, 0.003, $p=0.07$, $r_{\text{effect}}=0.17$; *adjusted* $b=-0.04$, $SE=0.02$, 95% CI: -0.08, 0.001, $p=0.06$, $r_{\text{effect}}=0.19$) nor trait anger-out (*unadjusted* $b=-0.03$, $SE=0.02$, 95% CI: -0.08, 0.01, $p=0.15$, $r_{\text{effect}}=0.13$; *adjusted* $b=-0.03$, $SE=0.02$, 95% CI: -0.08, 0.02, $p=0.18$, $r_{\text{effect}}=0.13$) were significantly associated with momentary positive affect. These relationships did not differ by gender.

Hypothesis 3: Trait Anger Expression Styles will Predict Greater Momentary Physical Limitation and Symptom Severity

Trait anger-in—Table 2 shows two findings on the relationships between trait anger-in and momentary disease adjustment. First, trait anger-in predicted more physical limitations in the full sample (*unadjusted* $p=0.030$, *adjusted* $p=0.006$), and separate models by disease status showed that the effect of anger-in on physical limitations was similar across patient groups, although only significant among patients with asthma (*unadjusted* $p=0.011$, *adjusted* $p=0.011$) versus patients with RA (*unadjusted* $p=0.10$, *adjusted* $p=0.35$). Second, high trait anger-in predicted more coughing/wheezing in the daily lives of patients with asthma (*unadjusted* $p=0.027$, *adjusted* $p=0.040$), but did not, however, predict more arthritis symptoms in RA patients (*unadjusted* $p=0.29$, *adjusted* $p=0.78$). These relationships did not differ by gender.

Trait anger-out—Table 2 also shows two findings regarding associations between trait anger-out and momentary disease adjustment. First, trait anger-out did not significantly predict physical limitations when models were run in the full sample (*unadjusted* $p=0.25$, *adjusted* $p=0.34$), nor when models were run only among patients with asthma (*unadjusted* $p=0.77$, *adjusted* $p=0.70$). In patients with arthritis, trait anger-out predicted greater physical limitations (*unadjusted* $p=0.023$), but this relationship did not remain significant after covariate adjustment (*adjusted* $p=0.06$). Second, high trait anger-out did not predict severity of coughing/wheezing among patients with asthma (*unadjusted* $p=0.70$, *adjusted* $p=0.76$), but high trait anger-out significantly predicted *lower* severity of RA symptoms patients with arthritis (*unadjusted* $p=0.008$; *adjusted* $p=0.022$). Relationships between anger-out, physical limitations, and disease-specific symptoms did not differ by gender.

Hypothesis 4: Momentary Physical Limitation and Symptom Severity will be Higher During Angry (versus non-Angry) Moments

Contemporaneous associations—Table 2 shows the within-person relationships between momentary anger experiences and physical symptoms. Three findings emerged. First, in the full sample, patients experienced significantly more physical limitations when experiencing anger versus not (*unadjusted* $p<0.001$; *adjusted* $p<0.001$). Separate models by disease status showed that both patients with asthma (*unadjusted* $p<0.001$; *adjusted* $p<0.001$) and patients with arthritis (*unadjusted* $p=0.015$; *adjusted* $p=0.010$) reported more physical

limitations when they were angry compared to themselves when they were not angry. Second, momentary anger experiences were associated with increases in coughing/wheezing among patients with asthma (*unadjusted* $p < 0.001$, *adjusted* $p < 0.001$) as well as increases in symptom severity among patients with RA (*unadjusted* $p = 0.002$, *adjusted* $p = 0.002$). None of these effects differed by gender.

Lagged associations—In an effort to determine if momentary anger experiences predicted subsequent disease adjustment, we ran lagged models testing whether anger from the previous assessment (M time lag=2.06 hours prior, SD=1.20 hours, min=0.02 hours, max=5.82 hours) predicted subsequent changes in symptoms and limitations. Lagged symptoms/limitations were included in both unadjusted and adjusted models to control for the autocorrelation in momentary health states. Lagged anger experiences did not predict momentary disease-specific symptoms or physical limitations (all $ps > .05$). Given that the associations between momentary anger experiences and symptoms/limitations could also plausibly operate in *the reverse direction*, with increases in symptoms/limitations predicting increased odds of subsequent anger experiences, we also examined *reverse* lagged associations testing whether physical limitations and disease specific symptoms from the previous assessment predicted whether or not anger was experienced at the current assessment (controlling for previous anger experience). These tests were performed using multilevel models that specified a binomial distribution and a logit link in SAS PROC GLIMMIX. Only one reverse lagged effect was significant, albeit in the opposite direction compared to its contemporaneous counterpart. Among patients with rheumatoid arthritis, for each unit increase in momentary physical limitations, the odds of experiencing anger at the next assessment *decreased* by 33% (*unadjusted* OR=0.77, 95% CI: 0.61, 0.97, $p = 0.028$; *adjusted* OR=0.77, 95% CI: 0.61, 0.97, $p = 0.030$).

Hypothesis 5: Associations between Momentary Health Indicators and Momentary Anger Experience will be Heightened for Patients with High versus Low Trait Anger-In or Anger-Out

Two-way interaction models testing anger expression style (anger-in or anger-out) X momentary anger experience interactions yielded no significant effects. Table 3 shows that significant three-way Gender $\times$ Trait Anger-In $\times$ Momentary Anger Experience interactions were found for physical limitations in the full sample (*unadjusted* $p = 0.017$, *adjusted* $p < 0.001$), physical limitations among patients with RA (*unadjusted* $p = 0.005$, *adjusted* $p = 0.009$), and symptom severity among patients with RA (although this last interaction was not significant after covariate adjustment; *unadjusted* $p = 0.026$; *adjusted* $p = 0.08$). Although unadjusted three-way Gender $\times$ Trait Anger-In $\times$ Momentary Anger Experience interactions predicting physical limitations (*unadjusted* $p = 0.11$) and coughing/wheezing symptoms (*unadjusted* $p = 0.20$) were not significant for patients with asthma, these interactions became significant in the covariate-adjusted models (physical limitations *adjusted* $p = 0.001$; coughing/wheezing *adjusted* $p = 0.008$), suggesting suppression effects. No three-way interactions were significant for trait anger-out.

Table 4 shows the covariate adjusted within-person relationships (Level 1 slopes) between anger experiences and momentary health, separately by gender and high (Mean + 1 SD)

versus low (Mean – 1 SD) trait anger-in, and displays four results. First, in the full sample, men with high versus low trait anger-in showed larger increases in physical limitations when they were angry versus not angry, as evidenced by a significant and higher within-person slope for men with high trait anger-in ($p < 0.001$) compared to the nonsignificant and lower slope for men with low trait anger-in ($p = 0.26$), and a significant anger-in X anger experience interaction for men ($p < 0.001$, see Table 4). These effects were also observed when analyses were run separately by disease status (asthma only and arthritis only, see Table 4). Second, among patients with asthma, men with high trait anger-in showed larger differences in coughing/wheezing across angry versus non-angry moments ($p = 0.006$) compared to men with low trait anger-in ($p = 0.30$; interaction for men $p = 0.005$). Third, although men with high ($p = 0.030$) versus low trait anger-in ($p = 0.48$) also showed larger differences in RA symptoms across angry versus non-angry moments, the interaction was not significant ($p = 0.15$). Fourth, among women, trait anger-in moderated the momentary relationship between anger experiences and physical limitations, but only for female patients with arthritis ($p = 0.040$). These interactions, however, were in the *opposite* direction of those in men. That is, women with *low* trait anger-in showed *higher* levels of physical limitations when angry versus not angry compared to women with high trait anger-in (see Table 4). All significant ($p < 0.05$) three-way interaction results are depicted in Figure 1.

A Test of Specificity: Can the Same Pattern of Results be Achieved by Replacing Anger Suppression/Anger-In with Trait Anxiety?

Given past evidence for a modest association between trait anger-in and trait negative affectivity/neuroticism (13), we ran separate models to test whether a similar pattern of results could be obtained using trait anxiety, as opposed to trait anger-in, as the main independent variable. These tests served to determine the specificity of the observed associations between trait anger-in, anger experiences, and momentary symptoms/limitations. Trait anxiety was measured at baseline using the Trait Anxiety Inventory (34). Trait anxiety was significantly correlated with anger-in ($r = .40$, $p < 0.001$) but was not significantly correlated with anger-out ($r = 0.13$, $p = 0.17$). Results of these models suggested that while some associations may be specific to anger-in, such as the main effects of anger-in on anger frequency, anger severity, physical limitations, and disease-specific symptom severity, the moderating effects of trait anger-in appear somewhat nonspecific (as many were duplicated with trait anxiety substituted). Model results for trait anxiety are available upon request from the first author.

Discussion

The current study examined the interplay between trait-level anger expression styles, momentary anger experiences, and physical health using EMA in the daily lives of patients with chronic disease. The following findings emerged from this investigation. First, we found that high versus low trait anger-in predicted greater anger frequency and greater anger severity (testing hypothesis 1). The relationship between trait anger-in and momentary anger severity was stronger for men. Second, trait anger-in predicted greater momentary negative affect, but did not predict greater momentary positive affect (testing hypothesis 2). Third, high versus low trait anger-in predicted more severe physical limitations and asthma-specific

symptoms in daily life, whereas high versus low trait anger-out predicted *less* severe RA-specific symptoms (testing hypothesis 3). Fourth, momentary anger experiences showed strong within-person associations with momentary physical health, with patients reporting greater symptom severity and physical limitations when angry compared to themselves when not angry (testing hypothesis 4). We found little evidence for lagged associations between anger experience and momentary health indicators. Fifth, although we found no evidence for two-way interactions between trait anger expression styles and momentary anger experiences (hypothesis 5), we did find evidence of three-way interactions between gender, trait anger-in, and momentary anger experiences in predicting momentary health indicators. Specifically, we found that for men, higher trait anger-in predicted stronger momentary associations between anger experiences and momentary health indicators; whereas for women, *low* trait anger-in predicted stronger momentary associations between anger and physical limitations, especially for patients with arthritis. The moderation results were not fully specific to anger-in, however, as a similar pattern of results was achieved by substituting trait anxiety as a moderator of these momentary associations. This suggests that the moderating effects of trait anger-in may be partly due to a general disposition toward negative affectivity, rather than anger suppression specifically; future research might explore this issue directly. Overall, our hypotheses were confirmed with regard to anger-in, but not with regard to anger-out.

This study is novel in that it is the first, to our knowledge, to examine the interplay between trait anger expression styles, momentary anger experiences, and momentary health in the daily lives of patients with asthma or RA. Past experimental studies using harassment conditions (provoking situational anger) have shown that the associations between trait anger expression styles and physiological reactivity are amplified in harassment conditions (17, 18, 20, 35, 36), and previous ambulatory assessment studies have shown that trait anger expression styles and state anger expression interact to influence health outcomes in trait $\times$ state interaction models (16, 24). Our study examines the interplay between trait anger expression styles (anger-in and anger-out) and momentary anger experience during normal day to day experiences. Generally, our findings align with, and extend, past experimental findings by showing that trait anger-in and momentary anger experience or arousal interact to influence momentary symptom severity and physical limitation in patients' daily lives. Moreover, as the extant literature in this area has focused largely on pain disorders, our study offers novel information about these processes in patients with asthma.

Our findings show some consistency with those from previous ambulatory assessment studies. For example, our finding that trait anger-in, but not anger-out, was predictive of greater daily life anger frequency corresponds with Martin and Watson (13), where a relationship between trait anger-in and anger frequency was found in the daily lives of female undergraduates. We also found that trait anger expression styles predicted momentary indices of physical health in the daily lives of patients with asthma and arthritis. Past research has shown that trait levels of anger (37) as well as trait levels of anger suppression/anger-in (3, 38) and anger expression/anger-out (3, 4) have been linked to respiratory diseases such as asthma and chronic pain diseases such as arthritis, but few studies have examined the effects of these trait anger expression styles on momentary health indicators in the daily lives of patient populations. Our results showed that high trait anger-in

was detrimental to the daily health experiences of patients with chronic disease, especially for patients with asthma, extending previous work on the link between trait anger-in and respiratory disease (3). For patients with arthritis, however, trait anger-in was not associated with disease-specific symptoms, and high trait anger-out may have a slightly *beneficial* effect. Although prior work suggests anger-out may have analgesic effects among chronic pain patients, the literature largely shows that high trait anger-out is associated with more frequent or severe experiences of chronic pain (see reference 4 for review). Additional examination of the effects of anger expression in the daily lives of chronic pain patients is needed.

We found clear and consistent evidence that momentary anger experiences were associated with greater physical limitations and more disease-specific symptoms in patients' daily lives, as we hypothesized given past EMA research with chronic pain patients (25) and evidence suggesting that anger arousal is associated with inflammatory biomarkers involved in the pathogenesis of both asthma and RA, such as immune complement C3 (9) and interleukin-6 (10). This finding adds to the accumulating body of evidence suggesting that momentary anger has a reliable influence on physical health parameters (15, 39, 40) and suggests that anger experiences may be important contributors to daily health and adjustment. We found no evidence of lagged associations for anger experiences predicting any of the momentary health indicators, and some (albeit very limited) evidence for reverse lagged associations, with momentary physical limitations predicting *reduced* odds of subsequent anger. This stands in contrast to an excellent recent study (25) showing that lagged state anger (3 hours earlier) was significantly associated with some measures of pain behavior in patients with chronic low back pain, and that lagged patient reported downtime was associated with increased state anger 3 hours later. The reason for the divergence between our findings and theirs is not yet entirely clear, although differences in samples, methods, measures, and patient populations may of course be contributing. More generally, however, we believe continued careful exploration of the lagged associations between momentary anger and momentary health indicators using ambulatory methods is warranted.

We found that momentary associations between anger experiences and physical limitations/symptoms differed significantly by patient gender and by level of trait anger-in. Specifically, men with high versus low trait anger-in reported relatively more health problems when they were angry versus not angry than did women, suggesting that anger-in amplified the momentary relationships between anger and physical health among men. For women, however, trait anger-in also moderated the momentary association between momentary anger experiences and physical limitations, but in the opposite direction, and only for women with RA. Overall, the pattern of gender differences tentatively suggests that *higher* anger suppression may be problematic for men with chronic disease, whereas *lower* anger suppression may be problematic for women. Previous studies on gender differences in anger expression style and health have shown that anger suppression has worse health effects for men versus women (17, 26) although not always (see 24, 27), but these studies have not shown that high anger suppression is *protective* or *beneficial* among women. Regarding anger-out, we did not find evidence that trait anger-out moderated the relationship between momentary anger and physical health, despite previous evidence showing that high trait anger-out affects (a) pain sensitivity when anger is induced in laboratory studies (35, 36, 41)

and (b) pain reports when anger is experienced in daily life (16). Future research replicating this pattern of findings and further examining these questions in the daily lives of independent patient samples is needed.

What might explain the observed associations between anger-in, increased anger arousal, and increased symptom severity, but the relative *lack* of such findings for anger-out? A number of theoretical models exist, most used to explain the associations between anger expression styles and pain. Burns and colleagues (5, 42), drawing on Wegner's ironic process model (43), suggest that attempts to suppress negative emotion (i.e., anger) may paradoxically *increase* this emotion, and this negative emotion may contaminate the perception of somatic states, including symptoms (25, 42). They provide evidence for this model in both healthy samples and in patients with chronic low back pain, showing that suppression of anger arousal is associated with greater anger during anger induction, as well as greater subsequent levels of both anger arousal and pain sensitivity during a subsequent pain induction (5, 42). Experiential avoidance models, such as those of Hayes and colleagues (8) and Gardner and Moore (44), predict that the avoidance of anger arousal – potentially through anger suppression – may ultimately serve to intensify and prolong anger. Given that the expression of anger via aggressive behavior is also discussed as a means of anger avoidance in Gardner and Moore's model, however, it is perplexing that we did not observe these effects for trait anger-out. Alternative models focusing on the physiological aspects of these relationships suggest that greater symptom severity should be associated with *both* anger-in and anger-out (45–47). As such, it is not yet entirely clear why we observed divergent effects.

The “match” between trait anger expression style and situational use of anger expression versus suppression may be a potential explanation for the divergence between anger-in and anger-out results. Laboratory and daily diary studies suggest the association between trait anger expression style and physiological or symptom-related reactivity to anger induction can be attenuated if the person uses their “preferred” style of anger expression, and intensified if they do not use this preferred style (18, 19, 24). It is possible that those with high trait anger-out were more consistently able to engage in their preferred style of anger expression in daily life (than were those with high trait anger-in), thus attenuating the associations between trait anger-out, momentary anger experience, and momentary health indicators. This remains speculation, however, as the current study did not include momentary measures of anger suppression or expression and could not test this hypothesis. Yet another potential reason for the divergence in anger-in versus anger-out associations may have to do with the item content of the scales used in the current study; the anger-in scale contains explicit references to anger experience (e.g., “I boil inside but don't show it”; “I am angrier than I am willing to admit”) whereas the latter focuses largely on aggressive behaviors enacted when anger is experienced but without explicit reference to anger experience (e.g., “I lose my temper,” “I do things like slam doors”; see reference 6).

The current study has several limitations. First, as mentioned above, we did not assess the use of anger suppression and anger expression strategies *in the moment*, preventing us from (a) knowing whether either of these strategies were used in moments when anger was reported and (b) testing the match or mismatch between trait and state anger suppression or

expression and how these were associated with symptoms. We note, however, that other work has examined the impact of both trait and state measures of anger-in and anger-out on pain and interference (see, e.g., references 16, 25). Nonetheless, our study provides a novel test of the interaction between anger expression styles and *anger arousal* in the daily lives of patients with chronic disease, thus examining a transactional model of anger expression styles and health—suggesting that anger expression styles may affect health by both changing the way an individual experiences, and responds to, anger experiences in everyday life. Second, although the use of EMA is a strength, our findings are based on self-reports. Third, we do not know if these momentary fluctuations in physical limitations and disease-specific symptoms are related to long-term disease prognosis. Fourth, although the gender differences found in our results are intriguing, the small number of men warrants caution in interpreting these findings until replicated in a larger sample.

This study nonetheless provides some preliminary contributions to research and practice. For research, the current study continues to extend understanding of the potential mechanisms linking anger expression styles and health in the daily lives of patients with chronic disease, suggesting that (a) anger-in may be more consistently related to poor health than anger-out, (b) anger-in is associated with more frequent and more severe anger experiences in patients' daily lives, which are robustly associated with physical limitations and disease specific symptoms; and (c) male patients with high versus low anger-in show larger increases in symptoms and limitations when they experience momentary anger, whereas female patients with high anger-in show opposite patterns—low anger-in was associated with greater anger sensitivity than high anger-in. For practice, the current study offers some preliminary evidence for *who* is likely to show greater symptom severity and physical limitation in daily life (men with high trait anger-in) as well as *when* these patients may be most at risk (when they experience anger). This may not only assist with standard practice, but may ultimately assist in the development of personalized and “just-in-time” intervention. Overall, the current findings map onto longstanding ideas that anger suppression relates to poor health, and extend previous work by providing a look inside the “black box” of daily life, examining the momentary processes and mechanisms that may relate anger expression style and disease adjustment in the daily lives of patients with chronic disease.

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Figure 1.

Gender $\times$ Trait Anger-In $\times$ Momentary Anger predicting momentary health outcomes. Predicted values depicted in these graphs were generated from covariate adjusted models with all covariates held at their means. High anger-in and anger-out are equal to the mean of each measure + 1 SD; low anger-in and anger-out are equal to the mean of each measure - 1 SD. Panel A shows the three-way interaction predicting momentary physical health in the full sample (asthma and arthritis patients). Panel B shows the interaction predicting physical limitations only among patients with asthma; Panel C shows this interaction only among patients with arthritis. Panel D shows the three-way interaction predicting coughing/wheezing symptoms in asthma patients.

Table 1

Sample characteristics and Momentary Averages, Full sample and By Gender

	Full Sample	Men (N=35)	Women (N=94)
<i>Sample Characteristics</i>			
Anger In, <i>M</i> (SD)	15.88 (3.98)	15.94 (5.06)	15.86 (3.50)
Anger Out, <i>M</i> (SD)	14.53 (3.35)	14.97 (3.97)	14.35 (3.07)
<i>Ethnicity</i>			
Caucasian	85%	89%	84%
African-American	9%	6%	10%
Other	6%	6%	6%
Age, <i>M</i> (SD)	44.16 (14.20)	46.69 (16.36)	43.20 (13.27)
<i>Disease Status</i>			
Asthma	76%	77%	75%
RA	24%	23%	25%
<i>EMA Measures</i>			
	Full Sample	Men	Women
<i>Anger Experiences</i>			
	<i>M</i> (SD)	<i>M</i> (SD)	<i>M</i> (SD)
Anger Frequency	33.20 (30.01)	36.29 (31.99)	31.98 (29.30)
Anger Severity	0.64 (0.68)	0.73 (0.83)	0.60 (0.62)
<i>Affect</i>			
Positive Affect	2.74 (0.87)	2.63 (0.81)	2.78 (0.90)
Negative Affect	1.15 (0.83)	1.04 (0.79)	1.19 (0.85)
<i>Momentary Health—Full Sample</i>			
Physical Limitations	1.36 (1.24)	1.17 (1.08)	1.44 (1.30)
<i>Momentary Health—Asthma Only</i>			
	Full Asthma Sample	Men (N=25)	Women (N=61)
Physical Limitations	1.08 (1.09)	1.01 (1.03)	1.10 (1.12)
Cough	1.38 (1.04)	1.34 (1.09)	1.39 (1.03)
<i>Momentary Health—RA Only</i>			
	Full RA Sample	Men (N=8)	Women (N=23)
Physical Limitations	2.16 (1.32)	1.69 (1.13)	2.33 (1.36)
RA Symptoms	2.36 (1.33)	1.99 (1.23)	2.46 (1.37)

EMA=ecological momentary assessment, RA=Rheumatoid arthritis. *M* = mean of each measure, SD = standard deviation.

Table 2

Relationships between trait anger expression styles, momentary anger experiences, and momentary health.

	Anger-In Models		Anger-Out Models		Anger Experience Models	
	Anger-In Effect (Unadjusted)	Anger-In Effect (Adjusted)	Anger-Out Effect (Unadjusted)	Anger-Out Effect (Adjusted)	Anger Experiences (Unadjusted)	Anger Experiences (Adjusted)
	b(SE) [95% CI]	b(SE) [95% CI]	b(SE) [95% CI]	b(SE) [95% CI]	b(SE) [95% CI]	b(SE) [95% CI]
<i>Full Sample</i>						
Physical Limitations	0.06 (0.03)* [0.006, 0.12] $r_{\text{effect}}=0.21$	0.07 (0.03)** [0.02, 0.12] $r_{\text{effect}}=0.26$	-0.04 (0.03) [-0.11, 0.03] $r_{\text{effect}}=0.11$	-0.03 (0.03) [-0.09, 0.03] $r_{\text{effect}}=0.09$	0.19 (0.04)*** [0.11, 0.27] $r_{\text{effect}}=0.42$	0.20 (0.04)*** [0.12, 0.27] $r_{\text{effect}}=0.42$
<i>Asthma Only</i>						
Physical Limitations	0.07 (0.03)* [0.02, 0.12] $r_{\text{effect}}=0.28$	0.07 (0.03)* [0.02, 0.12] $r_{\text{effect}}=0.28$	0.01 (0.04) [-0.06, 0.08] $r_{\text{effect}}=0.03$	0.01 (0.04) [-0.06, 0.09] $r_{\text{effect}}=0.04$	0.17 (0.04)*** [0.09, 0.25] $r_{\text{effect}}=0.42$	0.17 (0.04)*** [0.09, 0.25] $r_{\text{effect}}=0.41$
Coughing/Wheezing	0.06 (0.03)* [0.007, 0.11] $r_{\text{effect}}=0.24$	0.05 (0.03)* [0.003, 0.11] $r_{\text{effect}}=0.23$	0.01 (0.03) [-0.05, 0.08] $r_{\text{effect}}=0.04$	0.01 (0.04) [-0.06, 0.08] $r_{\text{effect}}=0.03$	0.22 (0.05)*** [0.13, 0.31] $r_{\text{effect}}=0.46$	0.21 (0.05)*** [0.12, 0.30] $r_{\text{effect}}=0.45$
<i>RA Only</i>						
Physical Limitations	0.12 (0.07) [-0.02, 0.27] $r_{\text{effect}}=0.31$	0.07 (0.08) [-0.09, 0.24] $r_{\text{effect}}=0.19$	-0.15 (0.06)* [-0.28, -0.02] $r_{\text{effect}}=0.41$	-0.13 (0.06) [-0.26, 0.003] $r_{\text{effect}}=0.37$	0.24 (0.09)* [0.05, 0.43] $r_{\text{effect}}=0.43$	0.26 (0.09)* [0.06, 0.45] $r_{\text{effect}}=0.45$
Symptoms	0.08 (0.08) [-0.07, 0.24] $r_{\text{effect}}=0.20$	0.02 (0.08) [-0.14, 0.18] $r_{\text{effect}}=0.06$	-0.17 (0.06)** [-0.30, -0.05] $r_{\text{effect}}=0.48$	-0.15 (0.06)* [-0.27, -0.02] $r_{\text{effect}}=0.44$	0.27 (0.08)** [0.11, 0.44] $r_{\text{effect}}=0.52$	0.28 (0.08)** [0.11, 0.45] $r_{\text{effect}}=0.52$

p<0.001**
p<0.01*
p<0.05.

Significant estimates (p<0.05) are presented in bold. All estimates are from multilevel models.

Table 3

Trait Anger-In $\times$ Momentary Anger Experiences Predicting Momentary Physical Limitations and Symptoms

	Gender $\times$ Anger-In $\times$ Anger Experiences (Unadjusted)	Gender $\times$ Anger-In $\times$ Anger Experiences (Adjusted)
	b(SE) [95% CI]	b(SE) [95% CI]
<i>Full Sample</i>		
Physical Limitations	-0.05 (0.02)* [-0.09, -0.01] $r_{\text{effect}}=0.23$	-0.08 (0.02)*** [-0.13, -0.04] $r_{\text{effect}}=0.34$
<i>Asthma Only</i>		
Physical Limitations	-0.03 (0.02) [-0.08, 0.01] $r_{\text{effect}}=0.18$	-0.08 (0.02)** [-0.12, -0.03] $r_{\text{effect}}=0.36$
Cough	-0.03 (0.02) [-0.08, 0.02] $r_{\text{effect}}=0.14$	-0.07 (0.03)** [-0.12, -0.02] $r_{\text{effect}}=0.29$
<i>RA Only</i>		
Physical Limitations	-0.24 (0.08)** [-0.40, -0.08] $r_{\text{effect}}=0.52$	-0.22 (0.08)** [-0.38, -0.06] $r_{\text{effect}}=0.48$
RA Symptoms	-0.16 (0.07)* [-0.30, -0.02] $r_{\text{effect}}=0.42$	-0.13 (0.07) [-0.27, 0.01] $r_{\text{effect}}=0.34$

p<0.001

**
p<0.01

*
p<0.05.

Significant estimates (p<0.05) are presented in bold. All estimates are from multilevel models.

Table 4

Within-person associations between anger and physical symptoms, by gender and trait anger-in

	Men			Women		
	High Anger-In	Low Anger-In	Interaction	High Anger-In	Low Anger-In	Interaction
	b (SE) [95% CI]	b (SE) [95% CI]	b (SE) [95% CI]	b (SE) [95% CI]	b (SE) [95% CI]	b (SE) [95% CI]
<i>Momentary Associations (Full Sample)</i>						
Anger Moments with Physical Limitations	0.36 (0.10)*** [0.16, 0.56] r _{effect} =0.32	-0.12 (0.11) [-0.34, 0.10] r _{effect} =0.11	0.06 (0.02)*** [0.03, 0.10] r _{effect} =0.31	0.14 (0.07)* [0.01, 0.28] r _{effect} =0.20	0.33 (0.07)*** [0.19, 0.48] r _{effect} =0.40	-0.02 (0.01) [-0.05, 0.003] r _{effect} =0.17
<i>Momentary Associations (Asthma Only)</i>						
Anger Moments with Physical Limitations	0.26 (0.10)* [0.06, 0.45] r _{effect} =0.28	-0.24 (0.12) [-0.48, 0.005] r _{effect} =0.22	0.06 (0.02)*** [0.03, 0.10] r _{effect} =0.37	0.17 (0.07)* [0.03, 0.31] r _{effect} =0.26	0.28 (0.08)*** [0.13, 0.43] r _{effect} =0.38	-0.01 (0.01) [-0.04, 0.01] r _{effect} =0.11
Anger Moments with Cough	0.32 (0.11)** [0.09, 0.54] r _{effect} =0.31	-0.15 (0.14) [-0.42, 0.13] r _{effect} =0.12	0.06 (0.02)** [0.02, 0.10] r _{effect} =0.31	0.21 (0.08)* [0.05, 0.36] r _{effect} =0.28	0.29 (0.09)** [0.12, 0.47] r _{effect} =0.36	-0.01 (0.02) [-0.04, 0.02] r _{effect} =0.08
<i>Momentary Associations (RA Only)</i>						
Anger Moments with Physical Limitations	1.15 (0.44)* [0.25, 2.06] r _{effect} =0.46	-0.02 (0.24) [-0.52, 0.48] r _{effect} =0.01	0.15 (0.07)* [0.002, 0.29] r _{effect} =0.38	-0.02 (0.17) [-0.37, 0.32] r _{effect} =0.03	0.56 (0.18)** [0.19, 0.93] r _{effect} =0.52	-0.07 (0.03)* [-0.14, -0.004] r _{effect} =0.39
Anger Moments with RA sx	0.90 (0.39)* [0.09, 1.70] r _{effect} =0.41	0.15 (0.22) [-0.29, 0.60] r _{effect} =0.14	0.09 (0.06) [-0.04, 0.22] r _{effect} =0.28	0.12 (0.15) [-0.19, 0.43] r _{effect} =0.16	0.41 (0.16)* [0.08, 0.73] r _{effect} =0.44	-0.04 (0.03) [-0.10, 0.03] r _{effect} =0.23

Table 4 Notes. p<0.001**
p<0.01*
p<0.05. r_{effect} = r effect size.

Significant estimates (p<0.05) are presented in bold. All estimates were generated via postestimation tests from covariate-adjusted multilevel models.