Physical activity moderates stressor-induced rumination on cortisol reactivity

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Abstract

Objective—Physically active individuals have lower rates of morbidity and mortality, and recent evidence indicates that physical activity may be particularly beneficial to those experiencing chronic stress. The tendency to ruminate increases and prolongs physiological stress responses, including hypothalamic-pituitary adrenal (HPA) axis responses as indexed by cortisol reactivity to stressful experiences. We examined the association between ruminating in response to a laboratory stressor task and HPA axis reactivity and recovery, and whether a physically active lifestyle moderates the associations between rumination and cortisol output trajectories.

Methods—Forty-six post-menopausal women underwent the Trier Social Stress Test while salivary cortisol was repeatedly measured. Twenty-five minutes after the end of the stressor, participants reported level of rumination in response to the stress.

Results—Findings indicate that physical activity moderated the initial rate ($B = -.10$, $SE = .04$, $p < .05$) and curvature ($B = -.03$, $SE = .01$, $p = .06$) of the relationship between rumination and log-transformed cortisol trajectory. Among sedentary participants, those who responded to the stressor with higher levels of rumination had a more rapid initial increase in cortisol (0.26 vs 0.21, $p < .001$), a later peak (56 vs. 39 minutes), and a delayed recovery (curvature -0.07 vs. -0.08, $p < .001$) compared to those with lower levels of rumination. In active participants, cortisol trajectories were equivalent, regardless of level of rumination.

Conclusions—In sum, individuals who maintain a physically active lifestyle may be protected against the effects of rumination on HPA axis reactivity to and recovery from acute stress.

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Keywords

acute stress; rumination; physical activity; mixed modeling; cortisol reactivity

Numerous longitudinal and intervention studies have shown the direct benefits of exercise on health. For example, exercise and increased fitness reduce risk for health problems, including depression (1–5), cognitive impairment and dementia (6–10), cardiovascular disease (11–13), diabetes (14; 15), and mortality (16–18).

A growing body of evidence suggests that remaining physically active is particularly beneficial for those undergoing chronic stress (19–23). Sources of chronic stress, such as caregiving and low socioeconomic status, are associated with increased risk for cardiovascular disease, metabolic syndrome, and other markers of disease (24–27). These relationships are likely mediated at least partially through repeated and prolonged activation of the stress-responsive hypothalamic-pituitary adrenocortical (HPA) axis. Exposure to even a brief stressor can stimulate the HPA axis and produce an elevated cortisol level that can persist for approximately one hour after exposure (28). While adaptive in the short term, repeated and chronic stimulation of the HPA axis and release of cortisol can lead to allostatic load (i.e. the physiological effects of repeated or chronic exposure to stress that lead to pathophysiological state) and accelerated biological aging (29–31).

Perseverative cognitions can prolong affective and physiological stress responses (32–36). A hallmark of perseverative cognition is rumination, the tendency to perseverate on self-relevant negative content and emotion often directed at one's past experiences (37–40). A disposition to ruminate, as measured by trait rumination measures, appears to be higher in women (41), predicts the severity and number of major depressive episodes (42; 43), and is also considered a feature of some psychiatric disorders (42; 44). Trait rumination may unfold daily by increasing ruminative responses to daily stressors (45). Such responses may in turn lead to increases in daily negative affect (45–47) and related increases in daily cortisol output (48). There is also evidence that laboratory-induced rumination affects physiological systems, prolonging stress-related stimulation of the HPA axis as evidenced by increased cortisol reactivity to the stressor (49). Therefore in this study, we examined the relationship between state rumination (i.e. rumination after a stressor) on concurrent cortisol responses.

Previous studies of exercise and fitness effects on the stress response have compared fit individuals (i.e. trained athletes and physically fit individuals) and unfit individuals and how they respond physiologically to laboratory induced acute mental stress. These have shown that fit versus unfit individuals have reduced HPA (50–52), inflammatory (53), and cardiovascular reactivity and faster recovery (54–57; 50). For example, Rimmele and colleagues (51) demonstrated that elite young adult male athletes had significantly lower cortisol and autonomic responses to the Trier Social Stress Task (TSST) compared to untrained men, and they additionally maintained positive mood and calmness in the face of stress. In another study, Traustadottir and colleagues (52) examined physiological stress reactivity in response to a laboratory stressor in unfit young as well as fit and unfit postmenopausal women. Fit post-menopausal women had similar cortisol responses to a laboratory stressor as the unfit younger women. On the other hand, unfit post-menopausal women had significantly greater output compared to their matched-age cohort and younger unfit equivalents. The findings from these studies suggest that being physically active appears to promote a healthier physiological response to stress in postmenopausal women.

In the present study, we examined the association between laboratory-induced rumination and cortisol reactivity and recovery in post-menopausal women. We additionally examined whether the relations between stress-induced rumination and cortisol varied as a function of activity level. We hypothesized that higher levels of rumination would be associated with faster initial cortisol reactivity and slower rate of recovery in response to the laboratory challenge tasks. In addition, we examined whether these associations would be more pronounced in sedentary compared to physically active participants.

Growth curve modeling was employed to capture reactivity and recovery from stress (58). Measuring cortisol output repeatedly permits the examination of the trajectory of cortisol in response to stress across time. Growth curve modeling can successfully attend to our outcomes of interest, such as whether rumination is related to participants' baseline cortisol levels, initial rate of cortisol increase (i.e. reactivity) in response to stress induction, and recovery over time. From these models we can then extrapolate number of minutes the average person takes to peak in cortisol levels and return to baseline. Modeling the overall pattern of HPA responses to stress, including initial rise, time to peak and time to recovery from stress to basal state, addresses increasing calls in the literature for a more nuanced understanding of HPA axis response to stress (59–61).

Methods

Post-menopausal women aged between 54 and 82 years were recruited through flyers and posters in the community and from service providers serving the elderly in the San Francisco Bay Area. Participants were part of a prospective study on caregiving and its effects on physical and psychological well-being that began in May 2005. All data presented are from participants' baseline visit. Women included both healthy women who were providing at least 4 hours of care to a relative with dementia per day and who reported high levels of perceived stress and age-matched non-caregiver controls reporting low levels of daily stress. Exclusion criteria included the presence of major medical conditions such as heart disease, cancer, or diabetes, use of medications containing agents known to affect stress hormone levels, and regular smoking. The study protocol was approved by the Institutional Review Board of the University of California, San Francisco. Written, informed consent was obtained from all participants.

Procedures

Women who called or emailed indicating interest were screened for eligibility criteria by telephone. They had a physical exam, fasting blood draw, and provided written informed consent at the UCSF Clinical and Translational Science Institute's Clinical Research Center (CCRC). They were scheduled to return on a separate afternoon one week later to undergo the TSST. During the week between visits, participants completed three consecutive days of daily diary assessment on mood, daily events, coping, and physical activity. On their return to the laboratory, they ate a standardized lunch provided by the CCRC metabolic kitchen, and had an intravenous forearm catheter inserted around 1300 hr. Participants had a one-hour resting baseline period while listening to relaxing music using headphones after catheter insertion. At the end of this baseline period (Time 0), the first saliva sample was collected. A modified form of the TSST (62) including performance of a speech and math task was administered. The phases of the stressor included four 5-min stressful periods (20 min total), including introduction to two trained evaluators who described the task, a preparatory period for the speech, a speech (about strengths and weaknesses, instead of a job interview, to fit the age group which includes many retirees), and lastly, a math task (serial subtraction of consecutive prime numbers). In line with the TSST, evaluators maintained neutral expressions throughout the tasks and followed a script to provide neutral feedback throughout the tasks.

Materials

Sociodemographics

Participants' *age* was calculated from date of birth. *Ethnicity* was assessed with a list from which participants selected one option (i.e. White, Black, Hispanic, Asian, Pacific Islander, Native-American). Participants selected from the following choices for *education*: 'Less than 12 years,' 'high school graduate,' 'some college or technical degree,' 'Associate in Arts degree,' 'Bachelor's degree,' and 'advanced degree.' *Income* was assessed by providing 22 categories of income ranges (lowest category, $0–$3,000 - highest category, $250,000 and above).

Saliva sample collections

Saliva samples were collected via passive drool method, using polypropylene saliva tubes at six time points throughout for the assessment of cortisol. Samples were collected at the following times in minutes: 0 (baseline), 15 (after the speech task), 20 (after the stressor ended), 30 (to capture cortisol peak), 50 (short term recovery), and 90 min (long term recovery, which is 70 min after the stressor ended, when cortisol is typically back to baseline levels) (63). Saliva samples were kept on ice and frozen at the end of each session, and sent for batch assay to Dresden, Germany (laboratory of Clemens Kirschbaum). Salivary cortisol was assayed using a chemiluminescence immunoassay (CLIA). Intra-assay CV was 2.9% for high levels and 7.7% for low levels. The inter-assay CV was 5.7% for high levels and 9.1% for low levels. The sensitivity lower limit was 0.16 ng/ml. Cortisol values ranged from 0.58 ng/ml to 33.75 ng/ml, well within the range of detection of the assays. Slight skewness (skewness statistic = 2.15) existed in the data and thus all values were log transformed for correction. Log-transformed values were normally distributed, and no outliers existed in the data.

Psychological Measures

Stress-induced rumination—Rumination items were adapted from the Ruminative Responses Scale (RRS) (37; 63). Twenty-five minutes after completion of the TSST, approximately when we sampled their 50-minute cortisol, participants completed the following rumination items based on the RRS and in response to completing the tasks: "I thought 'Why do I always react this way?'" "I thought about the tasks, wishing they would have gone better," and "I thought 'Why can't I handle things better?' "Participants responded on a 4-point Likert scale from 0 (never) to 3 (always). Average scores were calculated for the three questions for each participant. Due to concerns about item overlap between rumination and depression (64), we used items from the brooding subscale of the RRS without the items that included references to depressive symptomatology. Examples of excluded statements include "I thought about how hard it is to concentrate" and "I thought about all my shortcomings, failings, faults, mistakes?" The TSST-induced rumination subscale had good internal consistency (Cronbach's alpha=.75).

Physical activity—Participants reported at the end of each day, on three consecutive days, the number of minutes they engaged in vigorous exercise. They were asked, "Did you exercise today?" and were given the definition of vigorous activity that produced "increased heart rate and/or sweating." If participants reported exercising, they were then asked, 'How long did you exercise today?' For those participants who did not exercise on a particular day, minutes of exercise for that day were recoded to 0. All participants who participated in the daily component of the study answered these questions on all three days. Higher levels of self-reported physical activity are associated with greater fitness (64). Retrospective reporting is plagued by recall bias (65), and thus daily reporting of behaviors is considered a stronger measure of behavior (66; 67). Daily reports of physical activity were extremely

skewed (range 0 to 300 minutes; 24 participants reported no exercise throughout the 3 day period) and therefore we split participants based on reported required amounts of physical activity for good health per week. The CDC(68), based on previous work (69), recommends an average of 75 minutes of vigorous activity per week (an average of 33 minutes for a three day period, which is the number of days participants recorded their activities). We have previously split participants based on meeting these recommended guidelines (less than 33 minutes over a 3 day period = 0, equal or greater than 33 minutes = 1), labeled here, for brevity, as "sedentary" vs. "active"(22).

Covariates—Body Mass Index (BMI; kg/m^2) was included based on associations between BMI and cortisol in previous work (70). Depressive symptomatology was measured with the Inventory of Depressive Symptomatology and was included as a covariate given its strong associations with rumination (71), physical activity (72), and cortisol reactivity to stress (73).

Statistical Approach

Descriptive statistics and figure 1 for cortisol trajectories are presented with raw values. Log transformed values of cortisol were included in all analyses. Successive measurement of cortisol during and following a stressor permits the examination of cortisol trajectory over time with multilevel growth curve modeling (58). Multilevel growth curve modeling requires the employment of mixed modeling statistical designs. Given our small sample size and skewed data, we fitted a restricted maximum likelihood (REML) to our mixed models. We also fitted an unstructured covariance structure. Two strengths of mixed modeling with REML are that it can accommodate missing and skewed data and that it computes unbiased estimators (58; 74). Analyses were conducted with SPSS 18.0, using the MIXED syntax as recommended by Singer and Willett (58). All analyses were replicated with BMI, age, depressive symptomatology and control versus caregiver group as covariates, and the findings were consistent with reported results. In addition, the results did not vary as a function of hormone replacement therapy (HRT) (either when the 3 on HRT were excluded or HRT was included as a covariate).

We first examined the unconditional means model (i.e. with no predictors) to determine amount of variation occurring at the within person level. Next, we examined the unconditional multilevel growth curve model to determine the cortisol reactivity and recovery trajectory across the whole sample. Multilevel growth curve modeling of cortisol over time (set at 30 minute intervals) permits the delineation of a series of parameters that increase our understanding of reactivity and recovery from laboratory stressors. Growth curve modeling estimates the intercept (B_0), initial rate of change (i.e. time at each sampling of cortisol, B_{rise}), and curvature of the cortisol trajectory (i.e. time-squared, $B_{curvature}$). Stressor reactivity is captured by the intercept, initial rate of change, and number of minutes to peak, all of which is estimated by the growth curve model. Minutes for cortisol to peak is calculated with equation: peak = $(-B_{rise}/(2*B_{curvature}))$ (58). Recovery was captured by the curvature in the trajectory and the minutes estimated to return to baseline.

Next, we conducted two analyses, first examining the interaction between rumination and time and, second, the interaction between physical activity and time. We used Cohen, Cohen, West, and Aiken's approach (75) to regression analyses to analyze a three-way interaction between two continuous variables (time and rumination) and a dichotomous variable (activity level). In line with Cohen and colleagues recommendations, significant three-way interactions would suggest that cortisol trajectory varies differentially as a function of rumination at the two different activity levels. To follow-up on a significant three-way interaction, we examined simple interactions between rumination and time at

activity= 0 and activity = 1. Significant simple interactions suggest that cortisol trajectories are significantly different at varying levels of rumination. As a result, simple slopes are examined at one standard deviation above and below the mean rumination score to determine the intercept, rise and curvature at these two levels of rumination. If the simple interactions are not significant at a particular activity level, simple slopes are not examined since this suggests that rumination does not predict trajectory at that activity level.

Results

Univariate and bivariate results

Forty-six postmenopausal women completed cortisol measurement during the TSST. Table 1 presents means and standard deviations of age, BMI, rumination, depression, and raw cortisol values (ng/ml) at each time point during the TSST for the entire sample of participants, as well as for active versus sedentary participants. The range of time spent exercising ranged from 0 to 300 minutes over the 3 days of reporting, with a mean of 43 minutes ($SD = 66.23$). When categorized by activity level, eighteen participants were in the active group and 28 were in the sedentary group. Of those categorized as sedentary, the majority (N=24, 86%) reported no activity over the three days, and the remaining 4 participants reported less than 20 minutes total time spent exercising over the three days. While there were no significant differences between caregivers and controls in activity levels ($p = .23$), the majority of controls (61%) were active and the majority of caregivers (61%) were sedentary. Furthermore, caregivers and controls did not significantly differ in rumination ($p = .72$). Bivariate analyses indicated that only depression scores were different between the groups, such that those who were more active had fewer depressive symptoms than those who were sedentary ($p = .05$). All cortisol measures (log-transformed) were moderately to strongly significantly correlated with one another (correlations range: .42 to .85) with the exception of baseline cortisol to 50 and 90 minutes post baseline ($p = .09$).

Unconditional Means model

We partitioned the between and within person variation in cortisol output in response to the TSST among our participants. The estimates of the residual and intercept covariance parameters were 10.07 ($SE = 0.96$) and 7.61 ($SE = 2.01$) respectively. The intraclass correlation was .43 – in other words, 57% of the variation in cortisol response occurred within person.

Growth Models

We examined model fit for time in log-transformed cortisol, and results indicated that across all participants, log-transformed cortisol output followed a curvilinear relationship where intercept was 0.65 ($SE = .04$, $p = .00$), initial rate (time) of increase was 0.23 ($SE = 0.04$, $p < .01$) with a curvature (time-squared) -0.08 ($SE = 0.01$, $p < .01$). The moment when the quadratic trajectory curve reached its peak and turned downward was at 44 minutes after onset of stressor for the entire sample.

Next, we examined cortisol trajectory as a function of rumination alone, activity level alone, and the interaction between activity and rumination. In analyses with rumination alone, rumination did not significantly predict the trajectory of cortisol in response to stress (interaction $B_{slope} = .04$, $SE = 0.05$, $p = .48$, interaction $B_{curvature} = -0.01$, $SE = 0.01$, $p = .68$). Similarly, in analyses with activity level alone, activity did not significantly predict the trajectory of cortisol in response to stress (interaction $B_{slope} = -.01$, $SE = 0.11$, $p = .95$, interaction $B_{curvature} = -0.00$, $SE = 0.03$, $p = .98$). However, as hypothesized, the interaction term between rumination and exercise was significant in relation to the initial rate of cortisol increase (interaction $B_{slope} = -0.10$, $SE = 0.04$, $p = .01$) and was marginally significant in

relation to curvature (interaction $B_{curvature} = -0.02$, $SE = 0.01$, $p = .06$). The nature of the three-way interaction (i.e. rumination, exercise, time) was further examined for sedentary versus active participants. Results indicated that the association between rumination and cortisol trajectory (slope and curvature), but not baseline levels, significantly varied as a function of activity.

Rumination and cortisol trajectory in sedentary participants

For sedentary participants, initial increase and curvature in cortisol trajectory varied significantly as a function of levels of rumination ($B_{slope} = .23$, $SE = .04$, $p < .001$ and $B_{curvature} = -08$, $SE = .01$, $p < .001$, respectively). For sedentary participants at one standard deviation below the mean of rumination, baseline levels of log-transformed cortisol were 0.70 ($SE = 0.06$, $p < .001$), cortisol initially increased at a rate of 0.21 ($SE = 0.04$, $p < .01$) and had a significant curvature of -0.08 ($SE = 0.01$, $p < .001$), corresponding to a peak in cortisol reactivity at 39 minutes. At one standard deviation above the mean of rumination, baseline levels of log-transformed cortisol were 0.63 ($SE = 0.05$, $p < .001$), cortisol initially increased at a rate of 0.26 ($SE = 0.04$, $p < .001$) and had a significant curvature of -0.07 ($SE = 0.01$, $p < .001$), corresponding to a peak in cortisol reactivity at 56 minutes, 17 minutes after the peak of sedentary low ruminators. Relatedly, while sedentary low ruminators were on average likely to have returned to their baseline levels by the end of the study (extrapolated time from the graph was 49 minutes after end of stressor), those high ruminators who were sedentary had on average not yet returned to their own baseline. Recovery to baseline did not occur during our measurement period in sedentary high ruminators, and extrapolation showed average recovery to baseline at approximately 115 minutes - nearly 90 minutes after the end of the TSST, and 36 minutes after the sedentary low ruminators. In summary, compared with sedentary low ruminators, sedentary high ruminators had a more pronounced initial rate of cortisol increase, a later cortisol peak, and a slower return to baseline cortisol levels.

Rumination and cortisol trajectory in active participants

For all active participants, there was a significant cortisol intercept ($B = 0.64$, $SE = 0.05$, $p < .001$), significant rise ($B_{slope} = 0.24$, $SE = 0.06$, $p < .001$) and significant curvature ($B_{curvature} = -0.08$, $SE = 0.02$, $p < .001$) to the stressor corresponding to a peak of 41 minutes and return to baseline 60 minutes post-stressor. However, rumination was unrelated to cortisol intercept ($p = .53$), initial rate of change ($p = .10$), or curvature ($p = .18$). In other words, all active participants had similar trajectories, regardless of level of rumination.

Figure 1 summarizes these interaction effects. As illustrated, sedentary high ruminators had a more marked stress response and slower recovery than both sedentary low ruminators and all active participants. Furthermore, it seems that sedentary, low ruminating participants had, on average, similar trajectories to those who were active.[1]

Discussion

The present study examined whether being physically active moderates the effects of rumination on the trajectory of cortisol responses to acute stress. We hypothesized that one pathway through which physical activity could lower the stress response is by mitigating the effects of stress-related cognitions on the body's stress arousal systems. Our findings

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[1]We examined interactive effects of rumination and activity on the trajectories of heart rate (HR), diastolic blood pressure (DBP), and systolic blood pressure (SBP). Our data suggests no direct or interaction effects on BP, DBP or SBP trajectories. However, for DBP, physical activity and rumination significantly interacted to predict DBP across all sampling times (matched to times of cortisol sampling). For inactive participants, higher rumination was significantly related to increased DBP across the entire recording during and after the TSST. However in active participants, rumination was unrelated to DBP.

support this notion by demonstrating that the cortisol trajectory was a function of stress-induced rumination only in those who were sedentary, and unrelated to rumination in those who were active. Specifically, sedentary participants who had higher self-reported levels of rumination in response to the stressor had faster and prolonged reactivity and delayed recovery to stress, evidenced by a more rapid initial increase, a later peak, and a delayed return to baseline cortisol compared to the sedentary lower ruminating participants. For active participants, cortisol trajectory was not a function of rumination, regardless of level of stressor-induced rumination.

It is interesting to note that we may be detecting a trait-like effect rather than just the immediate effect of state rumination on cortisol. Rumination was determined after the acute stressor had terminated. Sedentary ruminators were not just high in cortisol during the recovery phase when we measured their levels of rumination, but also during the initial onset of the stressor. Therefore, our measure of state rumination may be differentiating people who are different in their initial acute stress reactivity, possibly due to differential appraisal of the stressor. Furthermore, activity alone may not shape cortisol responses. Rather, activity may be especially beneficial to those responding to stress with rumination, given the findings that activity moderates the relationship between rumination and cortisol reactivity and recovery, but does not directly predict cortisol reactivity to and recovery from stress.

We have recently demonstrated that being physically active, as defined here, moderates the relationship between chronic stress and short leukocyte telomere length, a marker increasingly understood to capture the accumulated burden of genetics, life stressors, and health behaviors (22). Understanding the psychological and physiological mechanisms through which physical activity confers such effects to those most vulnerable to stress is critical for the development of interventions to enhance health, and for identifying those individuals for whom new interventions would be beneficial. The present findings address these issues and suggest that one pathway that may explain the physiological benefits of physical activity is reduced activation and enhanced recovery of stress arousal systems tied to psychological responses to stress. Overall, these findings lend support to the idea that being physically active may confer stress resistance, at least in post-menopausal women. Importantly, these findings appear to extend to elderly women.

Chronic stress is documented to produce different profiles of HPA axis dysregulation, including either hyperactive or blunted cortisol responses to stressors (29; 74). In this study, we examined HPA axis reactions to acute stress in the context of rumination, a potential psychological mechanism of chronic stress. A recent meta-analysis by Denson and colleagues (36) provides evidence that rumination may induce increased cortisol levels in order to maintain vigilance toward unresolved stressors. Repeatedly ruminating in response to stress in the real world may repeatedly stimulate the HPA axis resulting in chronically elevated levels of cortisol. Chronic exposure to elevated cortisol is in turn linked with insulin resistance, accumulation of abdominal fat, and increased risk for cardiovascular disease (29; 31). Healthy levels of physical activity may differentially affect high ruminating individuals by creating a physiological eustress state (76), and attenuating biological stress responses including cortisol responses (50–52). A caveat of our study is that the heightened cortisol reactivity and delayed recovery observed in high ruminators was defined as such based on comparisons with low ruminators. At present, to our knowledge, there are no standard trajectories that are understood to represent excessively high cortisol reactivity or delayed cortisol recovery.

Our study adds to the current literature on the benefits of physical activity in that it tests if being physically active moderates the physiological effects of commonly experienced stress-

related cognitions. There are a number of neurobiological and physiological mechanisms by which exercise may confer benefits, including increases in the expression of genes that encode brain-derived neurotrophic factor (BDNF) leading to increased cognitive functioning and neural plasticity (77–79), a heightened anti-inflammatory environment in the body (80–82), enhanced insulin sensitivity (83–86), and enhanced oxidative buffering capacity (76; 87). Physical activity reduces depressive symptoms among those genetically predisposed to lower BDNF levels (20), and reduces symptom reporting and doctor visits in those with increased numbers of stressful life events (19; 21). Here, we demonstrate that physical activity may attenuate the acute physiological response to stress, as indexed by HPA axis reactivity, in those particularly vulnerable to stress – ruminators.

Limitations of the study included our short-term and self-reported measure of physical activity. Physical activity was reported on a daily basis across three days, and participants reported the number of minutes they engaged in activities in which their heart rate was increased and/or they perspired. We assume that these three days of reporting represent typical exercise behaviors, and thus, a long-term lifestyle factor. However, we do not know if the short-term activity specifically occurring in the days before the TSST was important in the results here, or whether this was a proxy measure for general fitness. Although our measure represents an important advance over the usual retrospective report of physical activity, future studies should measure participants' baseline fitness levels and objectively capture daily physical activity with the use of accelerometers or assessments of current physical fitness. Our measurement across three days may be an over or under representation of actual engagement in physical activity per week, and thus extending measurement to longer than 3 days to a week or two would benefit future studies that aim to use daily measurement of vigorous activity as a measure of high activity level. These limitations of our measure of physical activity may explain why we did not detect a main effect of physical activity on cortisol trajectory, in contrast with other studies on fitness and cortisol output (52–54). Self-report of physical activity is not as strong a predictor of health as measurements of actual fitness levels (82). Finally, only a randomized controlled aerobic exercise intervention study can truly test the buffering effects of physical activity on HPA axis activation as a result of stress-induction. Our results are thus limited to between-group differences that can also be a function of personality and other between group differences.

Findings of the present study are also limited to post-menopausal women, and should be replicated in larger samples, including men as well as individuals with diverse ages and ethnic backgrounds. Of particular importance, our sample size is limited to 18 participants in the active group, and 28 in the sedentary one. While mixed models with REML estimation are considered unbiased and robust, it may be possible that our findings are limited to our specific sample. We thus consider these findings preliminary, and suggest that follow up studies with larger sample sizes and more diverse groups and ages may be worthwhile. Given the sample size, we were unable to stratify our results by caregiver group, age and BMI. Exposure to ongoing chronic stress may change psychological responses including ruminative responses to acute stressors. However, in our sample, caregivers and controls showed similar levels of rumination in response to the acute laboratory stress, and our findings were significant over and above the effects of caregiver status. Exposure to chronic stress may also influence physical activity levels. Although not statistically significant, the caregivers in our study were disproportionately sedentary compared with controls. While significant differences between caregivers and controls in our study were not apparent, possibly due to sample size limitations, the current findings may be particularly relevant to caregivers, given their lower rates of physical activity. Caregivers who ruminate about everyday stressors may benefit from physical exercise interventions.

Further, there may be sex differences, which we could not test in this all-female sample. It may be that rumination is not only more common in women but has different physiological effects in women. Thus including men in future studies is important to further our understanding of the moderating potential of being active on physiological consequences of rumination. Comparing these trajectories to younger, fit and unfit women who are high and low on rumination will also deepen our understanding of the interplay of age with psychological and physiological response to stress.

In summary, the findings reported in the present study are the first, to our knowledge, that demonstrate the moderating effects of being physically active on the physiological responses associated with rumination following acute laboratory stressors. We applied a statistical model, namely growth curve modeling, which better captures the nature of physiological responses by modeling initial increases, minutes to peak responses, and speed of recovery. The effects found in the present study were apparent, even after covarying age, BMI, caregiving group, and depressive symptomatology. Heightened increases in cortisol responses and delayed recoveries to repeated stressors across the day would possibly lead to sustained exposure to elevated cortisol levels, ultimately affecting physical health outcomes(60). Our study demonstrates the potential for physical activity to allow rapid recovery of the HPA axis after the induction of stress, especially in those who are ruminating, thus reducing the heightened cortisol response earlier and potentially protecting individuals from continued, prolonged exposure. It has been increasingly clear that it is important to understand factors that enhance physiological recovery from stressors in the service of promoting health. Exercise appears to be one promising way to promote physiological stress resistance, particularly in stressed ruminators who are prone to affective and physiological disorders.

Acknowledgments

Abbreviations

HPA	hypothalamic-pituitary adrenal
TSST	Trier Social Stress Task
RRS	The Ruminative Responses Scale
BMI	body mass index
REML	restricted maximum likelihood
HRT	hormone replacement therapy

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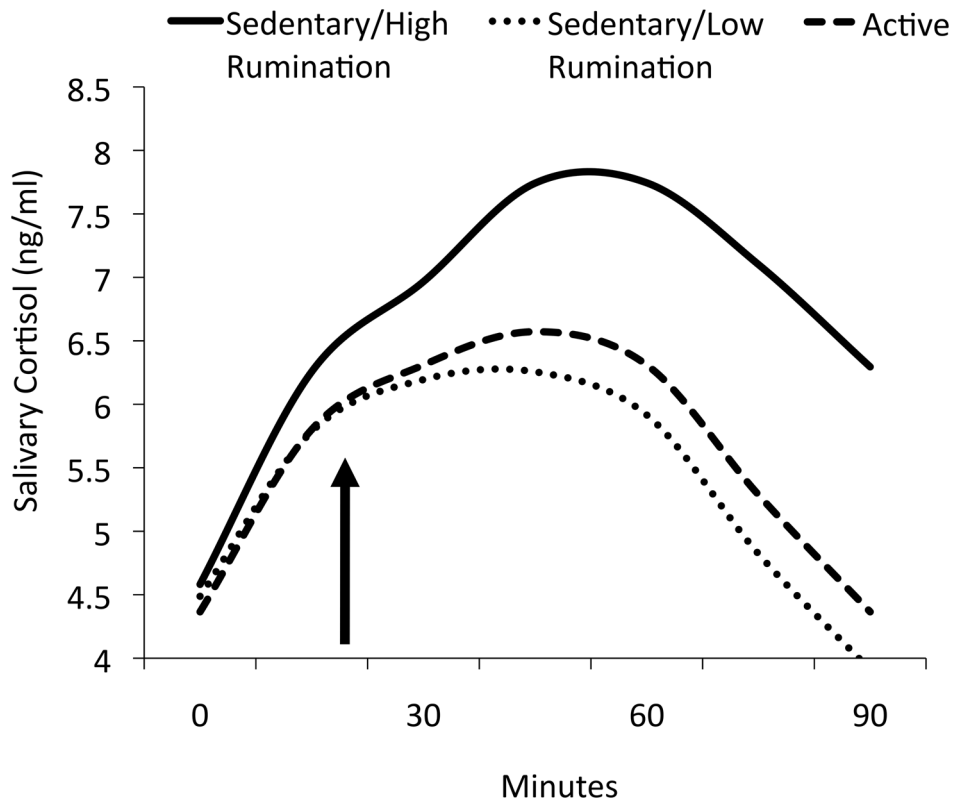
Figure 1.
Cortisol trajectory over the course of the TSST (90 minutes) as a function rumination and activity level.
Note. Values on the Y axis are solved Y values for salivary cortisol (ng/ml) from the quadratic equations for sedentary participants at low and high rumination and for all active participants. X-axis corresponds to time elapse over 90 minutes from beginning to the end of the TSST. The vertical black arrow corresponds to the approximate time the stressor ended. All active participants had similar trajectories, regardless of level of rumination, and thus are graphed as one group.

Table 1

Means and standard deviations for the overall sample and the active and sedentary sub-groups of participants,

	Total (N = 46)	Sedentary (N=28)	Active (N=18)
Group (Control N, %)	24 (52%)	11 (39%)	11 (61%)
BMI	26.40 (5.82)	27.16 (6.16)	25.22 (5.18)
Age	65.33 (5.81)	65.04 (5.98)	65.78 (5.67)
Depressive Symptoms	14.31 (10.73)	16.62 (11.99)	10.83 (7.54)
Rumination	1.81 (0.70)	1.89 (0.81)	1.72 (0.50)
2. Cortisol (ng/ml) 0min	5.04 (2.49)	5.08 (2.84)	4.98 (1.92)
3. Cortisol (ng/ml) 10min	5.88 (3.23)	6.32 (3.70)	5.14 (2.15)
4. Cortisol (ng/ml) 20min	8.04 (5.90)	8.88 (7.12)	6.72 (2.86)
5. Cortisol (ng/ml) 30min	7.90 (4.34)	7.61 (4.78)	8.32 (3.74)
6. Cortisol (ng/ml) 50min	7.36 (4.50)	7.49 (4.71)	7.16 (4.29)
7. Cortisol (ng/ml) 90min	5.06 (2.36)	5.33 (2.66)	4.62 (1.78)