Journal of Abnormal Psychology
2013, Vol. 122, No. 1, 45–50

0021-843X/13/$12.00 DOI: 10.1037/a0029881

Acute Exercise Attenuates Negative Affect Following Repeated Sad Mood Inductions in Persons Who Have Recovered From Depression

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Identifying factors that may protect individuals from developing Major Depressive Disorder (MDD) in the face of stress is critical. In the current study we experimentally tested whether such a potentially protective factor, engaging in acute exercise, reduces the adverse effects of repeated sad mood inductions in individuals who have recovered from depression. We hypothesized that recovered depressed participants who engage in acute exercise report a smaller increase in negative affect (NA) and a smaller decrease in positive affect (PA) when exposed to a repeated sad mood induction (i.e., habituation), whereas participants who do not exercise show sensitization (i.e., increased NA and decreased PA in response to a repeated adverse stimulus). Forty-one women recovered from MDD and 40 healthy control women were randomly assigned to either exercise for 15 minutes or quiet rest. Afterward, participants were exposed to two sad mood inductions and reported their levels of affect throughout the study. Recovered depressed participants who had not exercised exhibited higher NA after the second sad mood induction, a finding consistent with sensitization. In contrast, both recovered depressed participants who had engaged in acute exercise and healthy control participants showed no increase in NA in response to the repeated sad mood induction. Participants who exercised reported higher PA after the exercise bout; however, our hypothesis concerning reported PA trajectories following the sad mood inductions was not supported. Results suggest that exercise can serve as a protective factor in the face of exposure to repeated emotional stressors, particularly concerning NA in individuals who have recovered from depression.

Keywords: exercise, recovered Major Depressive Disorder, sad mood induction, repeated stress, physical activity

Major Depressive Disorder (MDD) is one of the most prevalent and burdensome of all psychiatric disorders. Importantly, MDD is a recurrent disorder; more than 80% of people with a history of MDD have more than one episode of depression (Kessler & Wang, 2009). Investigators examining factors involved in the onset and recurrence of depression have focused on the interaction of stress with vulnerability or protective factors (e.g., Caspi et al., 2003). Vulnerability-stress formulations posit that the development of depression is due to the interaction of stressful events with one or more vulnerability factors. Similarly, protective models focus on factors that may neutralize or reduce the impact of adverse experiences on the development of a negative outcome such as MDD (Fergus & Zimmerman, 2005).

Depression is characterized by elevated levels of negative affect (NA) and low levels of positive affect (PA), where NA represents the extent to which individuals feel subjective distress and unpleasurable engagement, and PA the extent to which they feel enthusiastic, active, or alert (Watson, Clark, & Tellegen, 1988). Behaviors that help to decrease NA and/or increase PA might act as protective factors in the face of stress. Acute exercise may be one such factor. Engaging in acute exercise has been found to increase PA (Reed & Ones, 2006) and result in fewer and less intense anxiety-related thoughts and attenuated physiological response following a psychosocial stressor (Rejeski, Thompson, Brubaker, & Miller, 1992). In a 1-week experience sampling study, Mata et al. (2012) found that depressed individuals had

This article was published Online First September 17, 2012.

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This research was supported by Grant Wi3496/41 from Deutsche Forschungsgemeinschaft to JM and NIMH Grant MH59259 to IHG. We thank Jessica Bowmer and Sarah Victor for their help with the study; Andrea Meyer and Rui Mata for advice on the article.

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higher levels of PA when they were physically active than during sedentary periods. Moreover, treatment studies with both unselected and clinical samples have documented that engaging in prescribed exercise significantly reduces levels of depressive symptoms, including NA (e.g., Mead et al., 2009). Indeed, investigators have found comparable benefits of treating MDD with sertraline (an antidepressant medication) and with exercise after 4 months (Hoffman et al., 2011) and 6 months (Babyak et al., 2000). Exercise can also reduce the likelihood of relapse of depression: in 10-month (Babyak et al., 2000) and 1-year (Hoffman et al., 2011) follow-up assessments, participants who exercised had significantly lower relapse rates than did participants who received medication.

Although most research concerning stress and depression has focused on the effects of major stressful life events like job loss or marital disruption, common repeated daily life adversities such as family arguments or work deadlines also have a cumulative effect and impair health and well-being (e.g., Almeida, 2005). Two general learning processes that affect response to repeated adverse experiences have been suggested: habituation and sensitization. Habituation is defined as a decreased level of response to a repeated stimulus; in contrast, sensitization refers to an increased level of response to a repeated stimulus (e.g., Eisenstein, Eisenstein, & Smith, 2001). For example, women have been found to exhibit higher heart rate and greater NA reactivity following a second than a first exposure to an adverse event, suggesting that they became sensitized to the event (Schmaus, Laubmeier, Boquiren, Herzer, & Zakowski, 2008).

Investigators have not examined the effects of one bout of acute exercise on the reaction to repeated stressors. In rodents, physical activity over time can facilitate habituation to repeated stressors: compared with rodents that did not exercise, the cortisol response to an acute stressor in rodents that exercised decreased more rapidly as they were exposed to more stressors (Nyhuis, Masini, Sasse, Day, & Campeau, 2010). Studying the effects of a 3-month exercise training on response to a repeated stressor, Blumenthal et al. (1988) randomly assigned 31 healthy men to an aerobic exercise program or a strength and flexibility program. Before and after the program, participants engaged in a stressful mental arithmetic task. Both groups showed a reduced physiological stress response, suggesting habituation; however, effects were stronger and applied to a higher number of physiological markers in the aerobic exercise group.

The present study was designed to examine experimentally the protective effect of exercise on affective responses to repeated stressors in individuals who have recovered from depression. We predicted that recovered depressed participants who engage in acute exercise will report a smaller increase in NA and a smaller decrease in PA when exposed to repeated sad mood inductions (i.e., habituation), whereas recovered depressed participants who do not exercise will show sensitization (i.e., increased NA and decreased PA).

Method

Participants

Eighty-seven women were recruited through advertisements in local newspapers, Internet bulletin boards, and university kiosks. Of these, four women were excluded because their scores on the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) exceeded the cut-off (see requirements below), and two dropped out before completing the second session, leaving a final sample of 81 women. Forty-one had recovered from MDD based on the Structured Clinical Interview for DSM (SCID; First, Spitzer, Gibbon, & Williams, 2002) and 40 were classified as never-disordered healthy control participants. Individuals were excluded from participating in the study if they had experienced severe head trauma, learning disabilities, psychotic symptoms, bipolar disorder, and/or alcohol or substance abuse within the last 6 months. Women in the "recovered" group had to meet *DSM–IV* criteria for a past major depressive episode. To confirm full recovery from depression, participants completed a slightly modified version of the SCID using guidelines recommended by the NIMH Collaborative Program on the Psychobiology of Depression (Keller et al., 1992): Participants had to have eight consecutive weeks with no more than two symptoms of no more than a mild degree (i.e., ratings of 1 = *no symptoms* or 2 = *minimal symptoms, no impairment*) in order to be classified as recovered from depression. Of the participants who had recovered from MDD, 11 participants met criteria for a current Axis-I disorder, and 27 participants for a lifetime diagnosis of an Axis-I disorder. Healthy controls could not have had a past or current diagnosis of MDD or any other Axis-I disorder and had to have a BDI-II score of 10 or less. All participants were native English speakers and between 18 and 58 years old. Participants provided informed consent and were compensated for their participation in the study. The Institutional Review Board at Stanford University approved the study.

Self-Report Measures

Depressive symptoms. Depressive symptoms were assessed with the BDI-II, Cronbach's Alpha = .84.

Affect. Affect was assessed throughout the experimental session with the following two questions: "How positive are you feeling right now?" and "How negative are you feeling right now?" on a 7-point Likert scale from 1 (*not at all*) to 7 (*very*) (cf. Samanez-Larkin et al., 2007). Before answering these scales for the first time, participants were thoroughly instructed on what was meant by feeling positive and negative: At the far left of the PA scale (1) they would feel completely neutral, neither positive nor negative; at the far right (7) they would feel very positive, such as happy, pleased, satisfied, competent, or some other positive feeling. At the far right of the NA scale they would be feeling very negative, which could be unhappy, frustrated, sad, depressed, or some other negative feeling.

Physical activity. Physical activity was assessed with the 7-Day Physical Activity Recall (Sallis, Haskell, & Wood, 1985), a structured interview to determine minutes spent in physical activity over the course of an average week; results were converted into weekly kilocalorie expenditure per kilogram of body weight.

Experimental Measures

Mood inductions. All participants viewed two film clips that have reliably been shown to induce negative mood (see Joormann & Gotlib, 2007, for a detailed description of the clips). Films were counterbalanced across participants and lasted approximately 6 minutes each.

Physiological measures. Heart rate and respiration were recorded with an integrated system and software package (Biopac MP150, AcqKnowledge; Biopac Systems, Goleta, CA) at a sampling rate of 1 kHz. Cardiovascular signals were recorded with the electrocardiogram (ECG) amplifier module and pregelled disposable electrodes. ECG data were scored offline using ANSLAB software (Wilhelm & Peyk, 2005), inspected for artifacts, false positives, and nonrecognized R-waves, and converted to heart rate (HR) using the corrected interbeat intervals. Exercise intensity during cycling was determined using percent heart rate reserve (HRR; Karvonen, Kentala, & Mustala, 1957). HRR is operationalized as the difference between resting and maximum heart rate. HRR is preferable to the simpler age-predicted percent heart rate because it takes into account the possible range of an individual's heart rate.

Procedure

Participants meeting eligibility criteria in a telephone screen were invited to complete two separate sessions. In Session 1, participants were administered the SCID. Our team of interviewers has previously achieved excellent interrater reliability for the assessment of recovery from a major depressive episode ($k = .91$) and for classifying participants as nonpsychiatric controls ($k = .92$; e.g., Levens & Gotlib, 2010). Next, the 7-Day Physical Activity Recall Interview was administered and participants completed demographic and BDI-II questionnaires.

Session 2 was conducted approximately 1 week later. Participants were asked to wear comfortable clothes in which they could move freely (e.g., t-shirt, sweat pants, sneakers) because we were planning to measure their heart rate and breathing, possibly while they were moving, and it would be easier to attach sensors if they wore loose clothing. Furthermore, participants were asked to refrain from participating in exercise on the day of the study and to consume no more than their typical amount of caffeine 2 to 3 hours prior to their session. Of the 40 healthy control participants, 20 were randomly assigned to an "exercise" condition and 20 to a "no exercise" condition. Of the 41 women recovered from MDD, 22 were randomly assigned to an "exercise" condition and 19 to a "no-exercise" control condition.[1] Physiological sensors were placed on the participants, followed by a 5-min acclimation period. Next, participants rested quietly with eyes open while baseline heart rate was recorded for 5 minutes. Participants assigned to the exercise condition cycled on a stationary bicycle for 15 minutes (SensorMedics Ergometric 800 cycle ergometer, San Diego, CA) at a speed and resistance they found comfortable. A meta-analysis has shown that most individuals choose mild to moderate activity as comfortable, which is the intensity level that leads to the greatest improvement in affect (Reed & Ones, 2006). Participants assigned to the control condition were asked to rest quietly with eyes open for 15 minutes. A selection of magazines was provided. Following completion of the exercise or control condition, participants sat quietly for 2 minutes. Participants then viewed a sad film and completed a 2-min information-processing filler task to give them a short break. Next, they watched the second sad film. Affect was assessed throughout the session: 10 minutes after arriving at the laboratory, after the exercise/resting period (i.e., 17 minutes after the previous affect assessment), after the first sad film (i.e., approximately 6 minutes after the previous affect assessment), and after the second sad film (i.e., approximately 8 minutes after the previous affect assessment). Participants were debriefed before leaving the laboratory.

Results

Demographic and Clinical Characteristics of Participants

Participants who had recovered from MDD did not differ from healthy control participants in age, education, ethnicity, or physical activity level, but had significantly higher BDI-II scores than healthy control participants (see Table 1).

Manipulation Check 1: Effects of Exercise on Heart Rate and Affect

Recovered and control participants did not differ significantly in baseline heart rate during rest (see Table 1). Overall, participants cycled at a mean of 34% HRR (see Table 1), which constitutes mild exercise. Paired t tests indicated that in the no-exercise condition percent HRR was comparable in the baseline and resting periods, $t(36) = .22$, $p = .83$, $d = 0.04$; in contrast, in the exercise condition, percent HRR was significantly higher during the cycling period than during baseline, $t(41) = 10.47$, $p < .001$, $d = 1.61$.

Participants in the exercise condition reported a significant increase in PA between baseline and after the exercise bout, $t(40) = -3.53$, $p = .002$, $d = -0.59$, whereas participants in the rest condition did not, $t(38) = 1.55$, $p = .130$, $d = 0.32$. There was no change in NA between baseline and exercise bout in the exercise condition, $t(40) = -0.53$, $p = .599$, $d = -0.14$, or between baseline and resting period in the rest condition, $t(38) = 0.27$, $p = .786$, $d = 0.04$.

Manipulation Check 2: Effect of Sad Mood Inductions on Affect

Paired t tests showed that PA decreased, $t(79) = 10.05$, $p < .001$, $d = 1.09$, and NA increased, $t(79) = -9.01$, $p < .001$, $d = 1.46$, after the first sad mood induction. After the second sad mood induction, again, PA decreased, $t(79) = 10.60$, $p < .001$, $d = 2.04$, and NA increased, $t(79) = -8.65$, $p < .001$, $d = -1.56$.

Does Exercise Attenuate the Effect of Repeated Sad Mood Inductions?

Means and standard deviations of the affect assessments are presented in Table 2. Healthy control participants and participants recovered from MDD differed significantly in their levels of baseline negative affect, $F(1, 77) = 9.04$, $p = .004$, $\eta^2 = .15$. Despite the randomization, post hoc tests indicate a baseline difference in NA ($p = .024$) between recovered MDD participants in the exercise and control conditions; no baseline NA differences

[1] One participant did not complete the second affect assessment; her data from this assessment are not included in the analyses. Physiological data from four participants (two recovered, two controls) could not be collected because of technical equipment malfunctions and were not included in the analyses of heart rate.

Table 1
Demographic Characteristics of Participants Recovered From MDD and Control Participants

	Recovered from MDD; N = 41	Control; N = 40	Statistical difference values
Age; *M* (*SD*)	38.61 (11.28)	38.33 (11.17)	$t(79) = -0.11$
Ethnicity (% Caucasian)	66	63	$\chi^2(5) = 3.46$
Education (% college graduates)	80	73	$\chi^2(3) = 4.53$
BDI-II score; *M* (*SD*)	5.90 (5.74)	2.40 (3.22)	$t(79) = -3.37$*
Physical activity (kcal/kg; 7-Day PAR); *M* (*SD*)	94.1 (28.3)	105.0 (49.0)	$t(77) = 1.22$
HRR			
Baseline heart rate	76.21 (13.45)	73.24 (8.14)	$t(77) = -1.18$
% HRR cycling (Exercise group); *M* (*SD*)	.27 (.20)	.37 (.18)	$t(40) = 1.67$
% HRR rest (No exercise group); *M* (*SD*)	−.01 (.02)[4]	.01 (.06)	$t(35) = 1.19$

Note. MDD = Major Depressive Disorder; BDI−II = Beck Depression Inventory−II; 7-Day PAR = 7-Day Physical Activity Recall Interview; HRR = heart rate reserve.
* $p < .01$.

were observed between control participants in exercise and rest conditions ($p = .583$). Healthy control and recovered MDD participants differed in baseline PA, $F(1, 77) = 4.49$, $p = .037$; $\eta^2 = .06$; post hoc tests showed no differences in baseline PA between the experimental conditions within either diagnostic group ($ps > .2$). Because of the differences between experimental conditions, we control for baseline NA in our analyses.

To test whether the four groups of participants differed in levels of NA following the sad mood inductions, we conducted a 2 (diagnostic group; recovered, control) × 2 (experimental condition; exercise, no exercise) × 3 (time: presad mood induction, after first sad mood induction, after second sad mood induction) repeated measures analysis of covariance (ANCOVA) on NA ratings with baseline NA, BDI-II score, and physical activity as covariates. This analysis yielded significant main effects of time, $F(2, 142) = 3.51$, $p = .033$, $\eta^2 = .05$, and an interaction of diagnostic group and time, $F(2, 142) = 4.78$, $p = .010$, $\eta^2 = .06$. Although the omnibus three-way interaction was only marginally significant, $F(2, 142) = 2.00$, $p = .105$, $\eta^2 = .03$, within-subject contrasts showed that the linear component of the three-way interaction of diagnostic group, experimental condition, and time was significant, $F(1, 71) = 4.71$, $p = .033$, $\eta^2 = .06$ (the quadratic component of this interaction was not significant, $F(1, 71) = 0.06$, $p = .810$, $\eta^2 = .001$).[2] To explore the linear component of the three-way interaction, we conducted planned contrasts, still controlling for baseline NA, BDI-II score, and physical activity.[3] These planned contrasts indicated that this interaction was due to participants who had recovered from MDD in the no-exercise group reporting higher levels of NA after the second mood induction than did participants in the three other groups, $t(76) = 2.97$, $p = .004$, $d = 1.03$ (see Figure 1). Importantly, planned contrasts for levels of NA presad mood induction and after the first sad mood induction were not significant ($ps > .3$). Also supporting our hypothesis, within the recovered depressed group, those who exercised reported significantly lower levels of NA after the second sad mood induction than did those who did not exercise, $t(39) = 2.65$, $p = .012$, $d = 0.83$. There were no significant main effects or interactions between exercise condition, diagnostic group, and reported PA following the repeated sad mood inductions (all $ps > .1$).

Discussion

The present study was designed to assess experimentally the effects of exercise on responses to repeated stressors in individuals who have recovered from depression. As hypothesized, we found that both recovered depressed participants assigned to engage in acute exercise and healthy control participants (regardless of exercise condition) showed no increase in levels of NA in response to a repeated stressor (i.e., sad mood inductions), whereas participants recovered from MDD who did not exercise exhibited higher levels of NA after the second mood induction, suggesting sensitization. However, in contrast to our hypothesis, we did not find any interaction between exercise condition and diagnostic group in level of reported PA following the repeated sad mood inductions that would be consistent with the notion of sensitization or habituation.

The finding of higher NA after the repeated mood induction in recovered depressed individuals who did not exercise is consistent with results of previous studies. Caspi et al. (2003) found a higher risk of developing MDD for individuals with a short-allele in the 5-HTT genotype only if they had been exposed to repeated (two or more) stressful life events; there was no difference in the probability of an adverse outcome if only one stressful life event had occurred. Nyhuis et al. (2010) reported a similar pattern in rodents: there was no difference in cortisol levels in response to an initial stressor between rodents that exercised and rodents that did not. However, after being exposed to repeated stressors, rodents that

[2] Whereas the control group was free of current psychopathology, 11 participants recovered from depression met criteria for a different current psychopathology. Repeating our analyses excluding these participants yielded comparable results: Again, the omnibus three-way interaction was marginally significant, $F(2, 120) = 2.45$, $p = .090$, $\eta^2 = .04$; the linear component of the three-way interaction of diagnostic group, experimental condition, and time was significant, $F(1, 60) = 5.17$, $p = .027$, $\eta^2 = .08$.

[3] We conducted planned contrasts despite $p > .05$ for the overall omnibus three-way interaction following Jaccard and Guilamo-Ramos (2002), who argued that an omnibus-test only tests the null-hypothesis that all groups in the analysis share a common population mean. If the omnibus *F*-value is statistically significant, the null-hypothesis that all means are equal is rejected. In our study, however, where three groups are expected to be equal and one to be different, there is less power to detect this difference in an omnibus-test; indeed, there is a much higher probability that an existing effect would be overlooked. Therefore, conducting a set of contrasts of conceptual interest, independent of the significance level of the initial omnibus test, is recommended.

[4] The mean % HRR is negative because participants' resting HR during acclimation was slightly higher than during the following resting period. This does not affect the interpretation of the results.

Table 2
Affect Ratings

	Baseline *M* (*SD*)	After exercise/rest *M* (*SD*)	After 1st mood induction *M* (*SD*)	After 2nd mood induction *M* (*SD*)
Negative affect				
Control, no exercise	1.40 (0.60)	1.45 (0.60)	2.30 (1.49)	2.15 (1.23)
Control, exercise	1.25 (0.44)	1.25 (0.55)	2.50 (1.36)	2.25 (1.16)
Recovered, no exercise	2.21 (1.32)	2.11 (0.99)	3.79 (1.18)	4.16 (0.96)
Recovered, exercise	1.59 (0.85)	1.67 (0.80)	3.45 (1.82)	3.13 (1.42)
Positive affect				
Control, no exercise	5.20 (1.36)	4.90 (1.52)	3.85 (1.79)	3.60 (1.90)
Control, exercise	4.65 (1.23)	5.10 (1.29)	3.55 (1.39)	3.60 (1.43)
Recovered, no exercise	4.37 (1.30)	4.21 (1.47)	2.53 (1.26)	2.42 (1.22)
Recovered, exercise	4.14 (1.73)	4.57 (1.60)	3.00 (1.57)	2.91 (1.48)

exercised had a significantly lower cortisol response than rodents that did not.

Although participants in the exercise condition reported increased PA after the exercise bout as expected, our hypothesis concerning reported PA following the sad mood inductions was not supported. Power analyses suggest that the failure to support our hypothesis was not due to sample size. It is possible that the experimental manipulation of sad mood inductions, which explicitly targeted NA, led to a trajectory of PA that did not differentiate experimental conditions and diagnostic groups. This suggests that the interaction between exercise and response to repeated sad mood inductions works differently in PA and NA. Furthermore, the different findings for NA and PA support the conceptual difference between PA and NA (cf. Watson et al., 1988). Future research examining psychological and physiological mechanisms that underlie the relation between exercise and response to repeated stressors might clarify such a difference in PA and NA response.

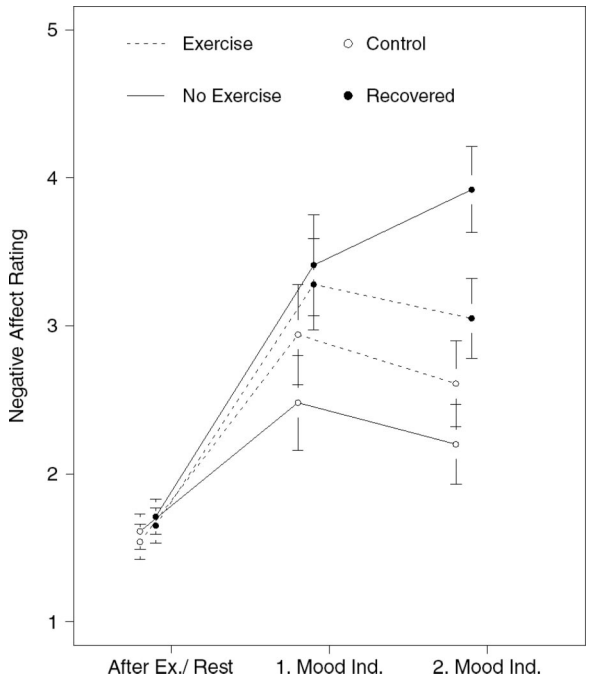

Figure 1. Negative affect ratings after exercise and sad mood inductions. *Note.* Error bars represent ±1 SEM. Negative affect ratings are estimated marginal means, controlling for baseline negative affect, BDI-II score, and physical activity. After Ex./Rest = After Exercise/Rest; Mood Ind. = Mood Induction.

This study has a number of theoretical and practical implications. This is the first study to test experimentally the attenuating effects of exercise on levels of NA in the face of exposure to repeated emotional stressors in individuals who have recovered from depression. Identifying such protective factors is an important contribution to research focused on understanding stress generation and stress sensitization in the context of the relation between stress and depression. This is particularly important in individuals recovered from depression who may be vulnerable to even mild stressors (Hammen, 2005). In her review article on stress and depression, Hammen (2005) concludes that exposure to repeated or elevated rates of stressors likely predicts recurrence of depression, leading to a "self-perpetuating cycle of depression and stress" (p. 303). Thus, a better understanding of factors associated with reduced stress responding, such as mild exercise, would not only contribute to a better understanding of vulnerability to recurrent depression, but may also lead to the development of more effective interventions to prevent such recurrences. Our findings highlight the effectiveness of low-intensity acute exercise to mitigate the effects of stress resulting from sad-mood inductions in participants who have recovered from MDD. Despite recommendations of regular moderate-intensity aerobic exercise to maintain general health (Haskell et al., 2007), the present finding is consistent with results of a recent meta-analysis indicating that mild-intensity acute aerobic exercise is optimal to improve positive affect (Reed & Ones, 2006). This can have important implications for adherence to an exercise regimen given that mild exercise, such as light cycling or going for a walk, is easier to integrate into everyday activities than is moderate or intense planned exercise.

It is important to acknowledge three limitations of this study. First, we assessed recovered depressed individuals in this study; future investigations should examine the effects of stress and exercise in participants with current MDD. Second, this is a single-session laboratory experiment; therefore, the extent to which the findings generalize to multiple sessions over longer periods of time, as well as to contexts outside the laboratory, must be determined in future research. Third, because positive and

negative affect were measured with one item each, reliability cannot be computed.

It will be important in future research to systematically examine psychological and physiological mechanisms that might underlie the effects obtained in this study. Nevertheless, the present study is important in indicating that exercise—even at a comfortable, mild level—can serve as a protective factor in the face of exposure to repeated emotional stressors, particularly concerning NA in individuals who have recovered from depression.

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Received January 31, 2012
Revision received July 18, 2012
Accepted July 24, 2012 ■