UNIVERSITY OF CALIFORNIA, SAN DIEGO

Reducing Cardiovascular Arousal to Psychological Stress with Brief Physical Exercise

A dissertation submitted in partial satisfaction of the requirements for the degree

Doctor of Philosophy

in

Psychology

by

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The dissertation of Sky Chafin is approved, and it is acceptable in quality and form for publication on microfilm:

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DEDICATION

To my mother, who will love me even if I get an F^{+}.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGEMENT

Chapter 2, in full, is as it appears in Chafin, S., Christenfeld, N., & Gerin, W. Improving cardiovascular recovery from stress with brief post-stress exercise. (in press). *Health Psychology.* The dissertation author was the primary investigator and author of this paper.

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ABSTRACT OF THE DISSERTATION

Reducing Cardiovascular Arousal to Psychological Stress with Brief Physical Exercise

by

Sky Chafin

Doctor of Philosophy in Psychology

University of California, San Diego, 2007

Professor Nicholas Christenfeld, Chair

We test if brief physical exercise can reduce cardiovascular arousal not just during a psychological stressor, as prior studies suggest, but both before it occurs, when one is anticipating the stressor, and after it occurs, when one is ruminating about it. Including both anticipatory and recovery responses may be more consistent with how individuals use exercise to cope with stress. It is also relevant to an expanded view of the cardiovascular reactivity hypothesis, which suggests that the

duration of the stress response, in addition to the magnitude of the initial peak reaction, may contribute to cardiovascular illness. In Study 1, following a mental arithmetic task, some participants did a brief exercise task while others sat still. We found that although exercising after the stressor adds to initial cardiovascular arousal, it goes on to improve recovery afterward. In both Study 2 and Study 3, subjects did a brief exercise task prior to a speech task. Some subjects were told of the speech before the exercise. Study 3 included a delay period after the exercise, so that residual arousal from exercise was no longer present when the speech began. Anticipating the stressor during the exercise did not produce any more, or less, arousal to the stressor than not anticipating it. However, taken together, Study 2 and Study 3 suggest an interaction of delay and exercise; relative to those who do not exercise, no delay between exercise and stress seems to prime the stress response, perhaps due to excitation transfer (Study 2), whereas a delay between exercise and stress attenuates the stress response (Study 3). We also test if the theory of misattribution of arousal can account for the stress attenuating effects of exercise; if an individual can credit some, if not all, of his arousal to positive invigoration from exercise rather than negative tension from a stressor, he may ruminate about the stressor less. However, in Study 4, other neutral tasks did not result in misattribution of arousal, which suggests that while exercise may simply be the best task at causing misattribution of arousal, exercise may instead be a unique activity.

Chapter I

INTRODUCTION

Participation in regular aerobic exercise has long been associated with good health, in particular a decreased risk of cardiovascular disease. Longitudinal studies show that individuals with the highest occupational or leisure time energy expenditure have the lowest incidence of myocardial infarction and other signs of heart disease, including sudden death (Paffenbarger & Hale, 1975; Paffenbarger, Wing, & Hyde, 1995; Morris, Pollard, Everitt, Chave, & Semmence, 1980;). Although the mechanisms responsible for this benefit are not fully understood, exercise is known to have favorable effects on many of the traditional risk factors for heart disease, including obesity (Blair, 1993; Wood, Stefanick, Williams, & Haskell, 1991), diabetes (Wasserman & Zinman, 1994), and high blood pressure (Paffenbarger, Wing, & Hyde, 1995).

Regular exercise also has favorable effects on many of the psychological risk factors for heart disease (for review see Rozanski, Blumenthal, & Kaplan, 1999). Individuals who participate in aerobic exercise training programs report less depression (McCann & Holmes, 1984; Roth & Holmes, 1987) and anxiety (Blumenthal, Williams, Needles, & Wallace, 1982) than control subjects who do not exercise. Runners also experience an anxiolytic effect of exercise (Dienstbier, Crabbe, Johnson, Thorland, Jorensen, Sadar, & LaVelle, 1981; Boutcher & Landers, 1988), as do swimmers (Berger & Owen, 1983). Fit individuals are also better at coping with chronic life stress (Roth & Holmes, 1987), and are less likely to experience illness following stressful life events (Roth & Holmes, 1985).

Exercise may also reduce cardiovascular risk by attenuating cardiovascular responses to stress. Exaggerated blood pressure and heart rate responses to

psychological stress are associated with damage to the cardiovascular system (Barnett, Spence, Manuck, & Jennings, 1997; Jennings et al., 2004; Kamarck et al., 1997; Matthews et al., 2004; Matthews, Woodall, & Allen, 1993; for review see Light, Sherwood, & Turner, 1992). People who exhibit large cardiovascular responses are at risk for the development of hypertension and cardiovascular disease, and situations that lead to large responses may put people at risk (Krantz & Manuck, 1984, 1986; Lovallo & Wilson, 1992;). Initial studies stemming from the cardiovascular reactivity hypothesis focused on acute cardiovascular responses in the immediate presence of the stressor (cardiovascular reactivity), but a number of more recent studies suggest that recovery, or the duration of blood pressure elevation, in addition to the magnitude of the initial peak reaction, may contribute to cardiovascular illness (Borghi, Costa, Boschi, Mussi, & Ambrosini, 1986; Gerin & Pickering, 1995; Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991; Steptoe & Marmot, 2005).

Since the cardiovascular adjustments to psychological stress mimic those associated with physical exertion (Dimsdale, Alpert, & Schnedierman, 1986), it was possible that fit individuals would have a more efficient response to psychological stress than less fit individuals (Surwit, 1986). While equivocal (de Geus, van Doornen, & Orlebeke 1993; Steptoe, Moses, Mathews, & Edwards, 1990; Sinyor, Golden, Steinert, & Serganian, 1986), fit individuals do show a reduction of cardiovascular reactivity to stressors in the lab (Anshel, 1996; Holmes & McGilley, 1987; Holmes & Roth, 1985; Light, Obrist, James, & Strogatz, 1987; Shulhan, Scher, & Furedy, 1986). Also, just as individuals with high aerobic fitness are able to recover from a physical stressor more quickly than low fitness individuals (Brown & Kenyon, 1968), fit individuals are able to recover more quickly from a psychological stressor

(Blumenthal, 1988; Cox, Evans, & Jamieson, 1979; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983).

While most of this work has been done with individuals who exercise regularly, more recent work has examined the role of a single exercise session. The rationale for this approach is both anecdotal, as individuals tend to report "feeling better" after exercising, and empirical, as the psychological benefits of aerobic exercise do not appear to depend on cardiovascular fitness, despite it being perhaps the most esteemed benefit of long-term exercise training (Moses, Steptoe, Mathews, & Edwards, 1989; Mondin, Morgan, Piering, Stegner, Stotesbery, Trine, & Wu, 1996). Mood improvements have been documented within the first few weeks of exercise training, before significant changes in fitness took place (Roth & Holmes, 1987), and after programs that provide minimal aerobic conditioning, such as yoga (Berger & Owen, 1992) and weight training (Doyne et al., 1987). This approach is also consistent with the finding that restricting habitual exercisers from their regular workouts can lead to an increase in mood disturbance with each successive day without exercise; with mood improving to baseline levels once exercise is resumed (Mondin, Morgan, Piering, Stegner, Stotesbery, Trine, & Wu, 1996).

Brief Exercise and Cardiovascular Reactivity

A single session of exercise can also attenuate cardiovascular reactivity to psychological stress. In general, following a single session of exercise, there is blood pressure (Ebbesen, Prkachin, Mills, & Green, 1991; Roy & Steptoe, 1991; Rejeski, Thompson, Brubaker, & Miller, 1992; Boone, Probst, Rogers, & Berger, 1993; West, Brownley, & Light; 1998), but not heart rate (Russell, Epstein, & Erickson, 1983; McGowan, Robertson, & Epstein, 1985; Duda, Sedlock, Melby, & Thaman, 1988) attenuation to a post-exercise stressor. While a review and meta-analysis of these

studies is available (Hamer, Taylor, & Steptoe, 2006), it is of interest here that although these studies vary in terms of the characteristics of the exercise (intensity, duration, and type), the stressor (intensity, duration, and type), the demographics of the sample (age, gender, ethnicity, activity and fitness level, history of hypertension), and the study design (between or within subjects design, time period between exercise and stressor, dependent measures) all put exercise before the stressor. That is, in all of these studies, subjects exercise before exposure to a laboratory stress task.

This arrangement is useful for determining whether exercise can limit the magnitude of stress responses. It does not, however, explore the use of exercise as a coping mechanism after the stressor, to limit the duration of the stress response. This is significant for two reasons. First, it is often the case that a stressor is quite short in duration, but the reaction to the stressor lasts far longer. It seems possible that the health impact of the event is not confined to the period when the stressor is present, but instead extends to the period after the stressor when the person is thinking about, and recovering from the stressor. Research on the benefits of regular exercise has shown that fit individuals are able to recover more quickly from a psychological stressor, but it is not known whether a single, brief session of exercise can have the same effect.

Second, the current approach has limited ecological value, as the investigators in these studies know the stressor is coming, but the subjects do not. Because stress can be, and often is, unforeseen in the real world, one cannot plan to exercise before being subject to it. Instead, it is more likely that people use exercise

as a coping mechanism after experiencing a stressor, such as going for a walk after an argument with one's spouse.

Anticipatory Stress and Brief Exercise

It also seems likely that individuals may use exercise as a coping mechanism in anticipation of a future stressor they are aware of, such as a job interview. Since anticipation of a stressor has effects that are similar to those associated with the stressor itself (Spacapan & Cohen, 1983), this is also relevant to the cardiovascular reactivity hypothesis. That is, it seems possible that the health impact of the event also extends to the period before the stressor when the person is thinking about, and anticipating the stressor. However, to date, no study has examined if exercise can attenuate cardiovascular stress responses to a stressor if the individual is aware of the stressor before the exercise begins.

Possible Mechanisms

Few authors have examined the mechanism(s) responsible for the attenuating effects of exercise on cardiovascular reactivity to stress. Nor do they suggest why exercise should promote recovery, although there are several possibilities.

Following a single session of exercise, there is a transient reduction in resting blood pressure (for review see Kenney & Seals, 1993). Postexercise hypotension (PEH) has been documented in both normotensive and hypertensive individuals, with the largest reductions in resting blood pressure, 18 to 20 mmHg SBP and 7 to 9 mmHg in DBP, in hypertensive subjects. PEH can persist as long as 16 hours after exercise, which could lower the blood pressure of hypertensive individuals into the normal range for much of the day following exercise, even in the presence of work-related stress (Brownley, West, Hinderliter, & Light, 1996) and mild exercise (MacDonald, Hogben, Tarnopolsky, & MacDougall, 2001). This finding has led some

investigators to suggest that the health benefits of regular exercise may not be due to chronic training adaptations but instead to individuals being more often in the post-exercise hypotensive period when stress occurs (Haskell, 1987). Nonetheless, in a recent meta-analysis and review (Hamer, Taylor, & Steptoe, 2006), among the studies to show attenuation in post-exercise cardiovascular stress responses, only three demonstrate a significant post-exercise hypotensive effect, which is at best only limited support for this mechanism.

Rather than through direct physiologic effects, exercise may modulate stress responses through cognitive appraisal processes (Bahrke & Morgan, 1978; Raglin & Morgan, 1987). That is, exercise may provide a diversion from distressing thoughts or activities, as subjects may find it difficult to ruminate while exercising. Or, subjects may not respond to an emotional stimulus following exercise because they are distracted by the residual arousal from exercise, and pay less attention to the provocation. However, while distraction has been found to be effective in reducing stress-associated elevations in blood pressure during rumination (Glynn, Christenfeld, & Gerin, 2002), it is not a sufficient explanation here. Roth, Bachtler, and Fillingim (1990) had subjects exercise or sit still for several minutes. During this period, half of the subjects in each condition performed a digits backward task. Scores on the *Profile of Mood States* (McNair, Lorr, & Droppelman, 1992) indicated that the exercising subjects, regardless of task exposure, reported less tension and more vigor than subjects who did not exercise. That is, subjects, who could not have been distracted from the stressor during exercise since the stressor was concurrent with the exercise, still reported less anxiety than subjects in the stressor-alone condition. Furthermore, in this study, the benefits of exercise on cardiovascular levels did not

occur during the exercise, when it should be distracting but blood pressure and heart rate are at their highest, but only after the exercise had ended.

A more subtle psychological process may be at work. Exercising after a stressor could have the effect of interfering with the arousal-anger-rumination process. Interfering with people's post-stress anger, or rumination, may be sufficient to break the feed-forward process in which anger, angry thoughts, and autonomic activation sustain each other long after the actual anger-provoking event, and thus may improve blood pressure recovery. Glynn, Christenfeld, and Gerin (2002) found that subjects who were distracted during a post-stress rest period exhibited faster recovery than subjects who were not, which suggests that ruminating about the stressful experience may contribute to its psychological and physiological sequelae. They also showed that stressors that produced an emotional response were associated with protracted cardiovascular recovery, independent of the blood pressure response evoked during the stressor, and that directed rumination of an emotional stressor was associated with greater cardiovascular reactivity.

Instead of simply distracting an individual from emotional thoughts, exercise may reduce the emotional nature of the thoughts, by providing an alternate attribution for the arousal produced in response to the anger-provoking stimulus. The misattribution of arousal hypothesis, a product of the two-factor theory of emotions, suggests that autonomic arousal results from a range of sources, including threatening environmental stimuli (i.e., a stressor) or interoceptive stimuli (such as may occur when one recalls a stressor). The theory further suggests that such arousal becomes the basis for emotion when and only when a cognitive attribution to an emotion-relevant stimulus is made (Schachter, 1964). Studies have shown that when a strong neutral attribution for arousal is present (i.e., have just exercised),

people can draw the wrong conclusions for why they feel the way they do, and may therefore inhibit an emotion from occurring even in the presence of an annoying stimulus (Schachter & Singer, 1962; Ross, Rodin, & Zimbardo, 1969; Storms & Nisbett, 1970).

The original misattribution of arousal formulation did not predict what would happen to sympathetic arousal after the misattribution was made, but research by Loftis and Ross (1974) has demonstrated that at least one index of sympathetic arousal, galvanic skin response (GSR), can be influenced by misattribution manipulations. Subjects were informed that their participation would involve exposure to a series of light presentations (CS), followed by a series of paired presentations of the light with a low level shock (UCS), followed by an extinction period in which the light would again be presented without the shock. The extinction period would also involve exposure to a loud white noise, ostensibly to test its effect on CS acquisition. During the experiment, subjects were told when the conditioning trials ended and, before the extinction trials began, were given a note card that informed them of the side effects of the white noise. Half of the subjects read symptoms that were similar to a fear response, such as palpitations, rapid breathing, and hand tremor. The remaining subjects read symptoms that were not associated with a fear response, such as headache and dizziness. The authors measured GSR during extinction. Their prediction, that the subjects who were able to misattribute their fear to the white noise would exhibit greater GSR resistance (less arousal) to the CS during the extinction trial than subjects who were given irrelevant symptoms of the white noise, was supported. In a second experiment, the misattribution manipulation preceded the conditioning trial, rather than the extinction trial. White noise was heard during the acquisition trial, when the CS and UCS were presented together, and

discontinued during the extinction trial. Subjects did not show diminished GSR during the conditioning trials, which is not surprising, given the presence of the UCS, a far more convincing source of the arousal than the noise. However, later during the extinction trials, subjects who had read fear-relevant side effects of the noise again evinced less arousal. Self-report measures, collected afterward, indicated that subjects given the misattribution manipulation felt less fear during both the acquisition and extinction trials. The authors suggest that misattribution effects were found during extinction rather than acquisition because the subjects changed their perceptions about the cause of their arousal "after the fact," even when the source of misattribution was no longer present. That is, it could be that, on reflection, the stressor did not seem so stressful.

The theory of misattribution of arousal is not inconsistent with the finding that fit individuals have a more efficient response to psychological stress than less fit individuals. That is, if exercise can decrease the magnitude and shorten the recovery time of physiological stress responses, including decreasing somatic signals of anxiety, individuals may interpret this reduction as an indication of being less anxious, and therefore may become less anxious (Blumenthal & McCubbin, 1987).

Insofar as an individual can credit some, if not all, of his arousal to positive invigoration from exercise rather than negative tension from a stressor, it is possible that exercise does not have to precede the stressor to reduce stress responses. And, if the anticipation of a stressor as well as the rumination of a stressor serve to prolong the physiological stress response, this may be relevant to the effects of cardiovascular reactivity on health. Thus, our first goal in these studies is to examine the effects of exercise on anticipatory responses to stress as well as recovery responses from stress. Our second goal is to determine if the theory of misattribution

of arousal can account for the stress attenuating effects of exercise. Considering that more than two-thirds of American adults are not active on a regular basis (US Department of Health and Human Services, 1996), and that half of all individuals who initiate an exercise program have quit participating within 6 months (Dishman, 1982), it is worth exploring further how a single session of exercise can have a positive impact on health.

Chapter 2

STUDY 1

Participation in regular aerobic exercise has long been associated with good health, and in particular with a decreased risk of cardiovascular disease. Longitudinal studies have found that individuals with the highest occupational or leisure time energy expenditure have the lowest incidence of infarction and other signs of heart disease, including sudden death (Morris, Everitt, Pollard, Chave, & Semmence, 1980; Paffenbarger & Hale, 1975; Paffenbarger, Wing, & Hyde, 1995). One explanation for this association is that regular aerobic exercise can buffer cardiovascular responses to psychological stress; investigators have demonstrated that fit individuals show a reduction in the magnitude (Anshel, 1996; Holmes & McGilley, 1987; Holmes & Roth, 1985; Light, Obrist, James, & Strogatz, 1987; Shulhan, Scher, & Furedy, 1986) and duration (Cox, Evans, & Jamieson, 1979; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983) of cardiovascular responses to stressors in the lab.

Exaggerated blood pressure and heart rate responses to stress are associated with damage to the cardiovascular system (Barnett, Spence, Manuck, & Jennings, 1997; Jennings et al., 2004; Kamarck et al., 1997; Matthews et al., 2004; Matthews, Woodall, & Allen, 1993; for review see Light, Sherwood, & Turner, 1992). People who exhibit large cardiovascular responses are at risk for the development of hypertension and cardiovascular disease, and situations that lead to large responses may put people at risk (Krantz & Manuck, 1984, 1986; Lovallo & Wilson, 1992). Initial studies stemming from the cardiovascular reactivity hypothesis focused on acute cardiovascular responses in the immediate presence of the stressor (cardiovascular reactivity), but a number of more recent studies suggest that recovery, or the duration of blood pressure elevation, in addition to the magnitude of the initial peak reaction,

may contribute to cardiovascular illness (Borghi, Costa, Boschi, Mussi, & Ambrosini, 1986; Gerin & Pickering, 1995; Haynes, Gannon, Orimoto, & O'Brien, 1991; Steptoe & Marmot, 2005).

While the association of exercise and health has been most often studied with chronic aerobic exercise, taking place several days a week for weeks or months, some studies have indicated that psychological benefits of exercise training can occur independent of changes in aerobic fitness (Mondin, Morgan, Piering, Stegner, Stotesbery, Trine, & Wu, 1996; Moses, Steptoe, Mathews, & Edwards, 1989; Roth & Holmes, 1987). This has led investigators to examine the role of a single session of aerobic exercise in reducing cardiovascular reactivity to a subsequent stressor. While not unanimous, in general, this line of research has found blood pressure (Boone, Probst, Rogers, & Berger, 1993; Ebbesen, Prkachin, Mills, & Green, 1992; Rejeski, Thompson, Brubaker, & Miller, 1992; Roy & Steptoe, 1991; Taylor & Katomeri, 2006; West, Brownley, & Light; 1998), but not heart rate (Duda, Sedlock, Melby, & Thaman, 1988; McGowan, Robertson, & Epstein, 1985; Roth, 1989) attenuation to a post-exercise stressor (see Hamer, Taylor, & Steptoe, 2006 for a review and meta-analysis). More recent work has focused on other measures of cardiovascular reactivity, including peripheral vascular resistance (Hamer, Jones, & Boutcher, 2006) and sympathetic activity (Brownley et al., 2003).

While these studies differ in their methodology, in terms of the characteristics of the exercise, the stressor, and the demographics of the sample, all put exercise before the stressor. This arrangement is useful for determining whether exercise can limit the magnitude of stress responses, but it does not explore the use of exercise as a coping mechanism after the stressor, to limit the duration of the stress response. This is significant for two reasons. First, it seems possible that the health impact of a

stressful event is not confined to the period when the stressor is present, but instead extends some time after when the person is thinking about, and recovering from the episode. Research on the benefits of regular aerobic exercise has examined cardiovascular recovery, and the acute aerobic exercise literature could benefit from this addition as well. Second, insofar as it is impossible to plan to exercise before an unforeseen stressor -- the investigators in these studies know the stressor is coming, but the subjects do not -- this approach has limited ecological value. Instead, it is more likely that people would use exercise as a coping mechanism after experiencing a stressor, such as going for a walk after an argument with one's spouse. Thus, both because cardiovascular recovery may be an important adjunct to reactivity in the health-relevant aspects of psychological stress, and because exercising after a stressor captures more of a real-world coping strategy, this study investigates the potential benefits of post-stress exercise on cardiovascular recovery.

The present investigation examined the effects of a single session of exercise, after a stressor, on cardiovascular recovery. The aim was to extend what is known about the benefits of exercise to a new domain, with important practical ramifications.

Method

Overview

Participants performed a serial subtraction task while being harassed by an experimenter. Participants were then randomly assigned, using a between-subjects design, into an experimental condition, in which the stressor was followed by three minutes of walking in place, or a control condition, in which participants sat still after the stressor. Another group of subjects walked in place without having done the math task. See Table 1.1 for a schematic of the study design. Blood pressure and heart rate were monitored during baseline, stressor, manipulation, and recovery periods.

Subjects

Undergraduates at the University of California, San Diego (*N* = 102) participated for course credit (72 females, 30 males, age *M* = 20.01 years, *SD* = 2.03 years). No instructions were given prior to participation, other than a brief description of the study (i.e., "Your blood pressure will be measured while you perform several tasks."). No subject reported being in poor health or on any medications that might influence cardiovascular readings. See Table 1.2 for baseline cardiovascular measures and demographic characteristics of the sample.

Procedure

Baseline. Upon the participant's arrival, the experimenter explained that the participant's blood pressure would be monitored during an arithmetic task and that some, but not all, subjects would participate in a moderate exercise task as well. (All subjects were informed about the prospect of exercising to control for any inflation in cardiovascular measures due to anticipation of the upcoming exercise task.) After giving informed consent, the subject was seated and fitted with the finger cuff of the blood pressure monitor. The experimenter explained that the subject would sit for a rest (baseline) period and then a different experimenter would administer a serial subtraction task, which would be followed by a brief activity period (possibly exercise), and a longer rest (recovery) period. In order to get a real time sample of what subjects were thinking about, the experimenter also instructed the participant to record his/her thoughts during the rest periods by jotting down a few words whenever a knock (at minutes 1, 2.5, 4, 7 and 11) was heard at the door. A knock was chosen as a signal because it did not require the experimenter to be in the same room as the subject. The participants were cautioned to write just enough to cue their memory so that later, at the end of the study, they'd be able to explain what they had been

thinking about to the experimenter. The experimenter also emphasized that she alone (and not the person administering the math task) would read the results of these thought reports. This was important because the role of the person administering the math task was to provoke anger; our past studies suggest that some of the thoughts reported during recovery related to the provoker, and we did not want the participant to feel unable to record such thoughts. After instructing the subject to sit still during the ten minutes of the rest period, the experimenter left the room.

Stressor task. After baseline, another experimenter, blind to the condition of the participant, entered the room and administered the mental arithmetic task. The subject was asked to count backward out loud by 13s from 2,397. Thirty seconds into the task, the experimenter informed the subject that his/her counting was too slow and that the task should be started again, at a faster pace. Similar interruptions informing the subject of deficient performance continued approximately every thirty seconds for three minutes. Each response was scheduled and standardized, so that each subject heard the same criticism at the same time. This task has been shown to be an effective stressor in several studies (Glynn, Christenfeld, & Gerin, 2002; Allen & Blascovich, 1994; Allen, Obrist, Sherwood, & Crowell, 1987), and has the potential to evoke more than one emotion, including anger and embarrassment. After the stressor ended, the experimenter who conducted the math task left the room and the original experimenter re-entered, to explain the instructions for the manipulation.

Stress / Exercise experimental condition. Following the stressor, subjects in the stress / exercise experimental condition performed a three-minute seated walking in place task, which involved raising the left and right leg alternately to a specified height (25 centimeters) in time to a metronome (120 beats per minute). This

procedure has been found to reliably elevate blood pressure in previous studies (Glynn, Christenfeld, and Gerin, 2002). Of more importance is that this task in isolation also leads to rapid blood pressure recovery, so that any sustained elevations in blood pressure during the recovery period, following the stressor and then the exercise task, are unlikely to be due to the exercise, but to the stressor. While actual walking or running is a more likely form of exercise in the real world, this task enabled the subject to maintain an erect posture, and to maintain the position of the arm, relative to the heart.

Stress / No Exercise control condition. Participants in the stress / no exercise control condition did not exercise. However, in order to determine whether any effects of the exercise could be due to the metronome used in that condition being distracting in itself, half of the subjects (stress / metronome task) tapped the index finger of their dominant hand to the same metronome beat for three minutes. The remaining subjects (stress / no task) sat still, in silence, for three minutes.

No Stress / Exercise control condition. A no stress / exercise control condition, added later, assessed the effect of exercise alone, without the stressor present. During the stressor period, subjects sat for three minutes in silence. While the subjects in this condition were not randomly assigned with the other subjects, they were drawn in the same way from the same subject pool, and were identical in basic demographic characteristics.

Recovery. In both the control and experimental conditions, at the end of the three-minute manipulation, the experimenter asked the subject to sit still for a final rest period, only jotting down a few words when a knock was heard at the door. The experimenter then left the room, knocking on the door at the five standardized times. After 15 minutes, the experimenter returned, removed the finger cuff and interviewed

the participant about the thought reports. The participant then completed several questionnaires and was debriefed.

Subjective Measures

At the end of the recovery period, all subjects rated, on 7-point Likert-type scales, “How difficult was the arithmetic task?” (1 = “not at all difficult” to 7 = “very difficult”), and “How stressful was the arithmetic task?” (1 = “not at all stressful” to 7 = “very stressful”). Subjects were also asked “How often did you think about the arithmetic task during the last 15 minutes?” (1 = "no time at all" to 7 = "the whole time") and “How often did you think about the other task (sitting in silence, finger tapping, or leg lifts) during the last 15 minutes?” (1 = "no time at all" to 7 = "the whole time").

Recording of Physiological Measures

Systolic and diastolic blood pressure and heart rate were recorded with an Ohmeda Finapres 2300 blood pressure monitor. Using the Peñaz method, this instrument measures beat-to-beat pressures from an inflatable finger cuff worn on the third finger of the non-dominant hand. The Finapres has proven to be a useful alternative to intra-arterial blood pressure measurement in laboratory testing (Imholtz, Settels, & Meiracker, 1990) and clinical practice (Gorback, Quill, & Lavine, 1991; Weiling, Harkel, & Lieshout, 1991). It is also able to track intra-arterial readings during abrupt changes of blood pressure (Parati, Casadei, & Groppelli, 1989). The Finapres enhances reliability by collecting a large number of readings (Gerin, Pieper, & Pickering, 1993).

Data Reduction and Analysis Procedures

The beat-to-beat pressures from the Finapres were combined into minute averages across the experimental session. The cardiovascular dependent measures

were change scores, computed using the difference between the minute averages for the period of interest and the mean of the pre-task baseline measurements. These means were computed using the pulse-based technique, in which equal weight is assigned to heart beats, rather than time intervals, resulting in greater weight given to the periods when the pulse is elevated (Glynn, Christenfeld, & Gerin, 1997). Raw change scores, rather than residualized change scores, were used (Llabre, Spitzer, & Saab, 1991).

We verified the initial equivalence of groups by comparing the mean of the pre-task baseline measurements with a separate one-way analysis of variance (ANOVA) for each of the cardiovascular measures. Manipulation and treatment effects were analyzed by comparing the mean change score for each period with a separate one-way ANOVA for each of the cardiovascular measures. An alpha level of .05 was used in the analysis.

Results

Baseline Measures

There was no significant difference between conditions during the baseline period for any of the cardiovascular measures (all Fs (2, 99) < 1.21, all ps > 0.30).

Stress Manipulation Check

The serial-subtraction task was effective as a stressor for all conditions, with average task increases of 22.8 mmHg systolic blood pressure, 14.5 mmHg diastolic blood pressure, and 13.7 bpm heart rate. There was no significant difference between conditions in the cardiovascular measures during the stress manipulation (all Fs (1, 76) < 0.45, all ps > 0.50).

Effect of Exercise on Blood Pressure and Heart Rate

In the stress / exercise condition, at the end of the exercise period blood pressure and heart rate were elevated compared to the end of the math stressor, and compared to the levels of the subjects in the stress / no exercise and no stress / exercise control conditions. Systolic blood pressure was 39.4 mmHg above baseline (11.9 mmHg higher than at the end of the math task, 29.9 mmHg higher than the stress / no exercise condition, and 8.5 mmHg higher than the no stress / exercise condition). Diastolic blood pressure was 21.7 mmHg above baseline (3.9 mmHg higher than at the end of the math task, 18.9 mmHg higher than the stress / no exercise condition, and 5.5 mmHg higher than the no stress / exercise condition). Heart rate was 37.0 bpm above baseline (22.3 bpm higher than at the end of the math task, 40.7 bpm higher than the stress / no exercise condition, and 5.3 bpm higher than the no stress / exercise condition).

Effect of Experimental Condition on Blood Pressure and Heart Rate Recovery

For ease of comparison, the last ten minutes of the recovery period is used in the analysis of cardiovascular recovery. This was a post hoc decision, as we were looking for the point at which the blood pressure measures for all conditions had leveled off, allowing a stable comparison across groups. This is also a conservative measure, as the stress / no exercise control condition has had three additional minutes to recover. Subjects in the stress / no exercise condition were sitting still for three minutes while those in the stress / exercise condition and no stress / exercise condition exercised; the manipulation period for these subjects was, in effect, the beginning of their recovery period.

Within the stress / no exercise control condition, there were no significant differences in any of the cardiovascular measures between subjects who finger-tapped and those who did not. Therefore, these two groups were combined for the

recovery analysis. It is worth noting that, while differences were not significant, those subjects who finger-tapped had slower recuperation, suggesting the metronome by itself did not enhance recovery, and was, if anything, actually more arousing than distracting.

There was a significant effect in improving recovery of having exercised on both systolic blood pressure (F (2, 99) = 4.35, $p < 0.05$, $\eta^2 = 0.08$; see Table 1.3 and Figure 1.1) and diastolic blood pressure (F (2, 99) = 6.38, $p < 0.005$, $\eta^2 = 0.11$; see Table 1.3 and Figure 1.2) during the last ten minutes of the recovery period. A *post hoc* Tukey HSD showed a significant difference in systolic blood pressure between the stress / exercise and stress / no exercise conditions, $p < 0.05$, with exercising after stress returning systolic blood pressure closer to baseline than not exercising after stress. Diastolic blood pressure followed the same pattern ($p < .01$).

Although subjects who exercised after the stressor (stress / exercise) had significantly ($p < .05$) higher systolic and diastolic blood pressure during the exercise manipulation than subjects who exercised without having first experienced the stressor (no stress / exercise), there were no significant differences in recovery between the two conditions ($p > .90$).

There were no significant differences in heart rate between conditions (F (2, 99) = 3.06, $p > 0.05$; see Table 1.3 and Figure 1.3). Slight spikes in the heart rate data during the recovery period, just visible on the figure, coincide with the thought sampling signals (at minutes 1, 2.5, 4, 7, and 11).

Self-Report

Stressor. There were no significant differences between conditions in how difficult subjects found the math task, F (1, 76) = 0.06, $p > 0.80$. The average response was 5.3 on a seven-point Likert-type scale in which “7” was most difficult.

Nor were there significant differences between conditions in how stressful subjects found it, F (1, 76) = 0.11, $p > 0.74$. The average response was 5.0 on a seven-point Likert-type scale in which "7" was most stressful.

Rumination. There was not a significant difference in how much subjects reported thinking, during the recovery period, about the stressor, F (1, 76) = 0.21, $p > 0.65$. The average response was 3.5, where "7" indicated that they were thinking about the stressor the whole time. There was a significant difference in how much subjects reported thinking about the manipulation (sitting in silence, finger tapping, or walking in place), F (1, 76) = 9.24, $p < 0.005$. Subjects who did exercise reported thinking more about the manipulation (M = 2.6) than subjects who did not exercise (M = 1.8), where "7" indicated that they were thinking about the manipulation the whole time.

Thought sampling. Subjects in the stress / exercise condition reported fewer emotional thoughts about the math task (for example, "I'm so bad at math," "I feel dumb," "I hate the number 13") than subjects in the stress / no exercise control condition, achieving marginal statistical significance (an average of .40 and .80 emotional thoughts per person, respectively, F (1, 76) = 2.88, $p < .10$, $\eta^2 = 0.04$). Over 25 percent of subjects in the stress / exercise condition wrote about going to the gym or the need to exercise more often, compared to three percent of subjects in the stress / no exercise condition (F (1, 76) = 9.97, $p < .005$, $\eta^2 = 0.12$), suggesting that exercise was successful in altering the nature of the subjects' thoughts. That is, since the thought sampling was a way of probing, in real time, what participants were thinking about during the recovery period, it follows that, at any given time, the participants who had exercised were more likely to be thinking about the exercise task, rather than the stressor. This does disagree, however, with our other measure

of rumination, in which subjects reported no difference in how often they thought about the math task. This disagreement may be due to the point in time that the measures were taken; the thought sampling was a real time measure, the self-report was retrospective.

Discussion

While exercising before a stressor has been shown to limit the magnitude of stress responses, we tested the use of exercise as a coping mechanism after the stressor, to limit the duration of the stress response. To our knowledge, this is the first study of its kind to examine the effects of post-stress exercise on cardiovascular recovery. While the blood pressure of the participants in the exercise condition was far higher, during the exercise, than the blood pressure of the participants who did not exercise, soon after the tasks were completed the subjects who had exercised had significantly lower systolic and diastolic blood pressure than subjects who had sat still. It is notable that, although exercising after a stressor adds to initial cardiovascular arousal, it goes on to facilitate cardiovascular recovery afterward.

The existing literature on acute aerobic exercise does not offer a clear explanation for why exercise should promote recovery, although there are several possibilities. Exercise may work by distracting subjects from thoughts of the stressor (Bahrke & Morgan, 1978; Raglin & Morgan, 1987). It is also possible that exercise could have produced a hypotensive aftereffect. Or, the exercise could have altered the way people thought about the stressor, by misattribution of arousal.

While distraction has been found to be effective in reducing stress-associated elevations in blood pressure during rumination (Glynn, Christenfeld, & Gerin, 2002), it is not a sufficient explanation for the efficacy of exercise in prior experiments. Roth, Bachtler, and Fillingim (1990) had female subjects bicycle or sit still for ten minutes.

During this period, half of the subjects in each condition performed a difficult mental arithmetic task. Scores on the *Profile of Mood States* indicated that the exercising subjects, regardless of task exposure, reported less tension and more vigor than subjects who did not exercise. That is, subjects, who could not have been distracted from the stressor during exercise since the stressor was concurrent with the exercise, still reported less anxiety than subjects in the stressor-alone condition. Furthermore, in our study, the benefits of exercise on cardiovascular levels do not occur during the exercise, when it should be distracting but blood pressure and heart rate are at their highest, but only after the exercise has ended.

Instead of distraction, a physiological mechanism may be at work. Some studies have found that, following a single session of exercise, there is a transient reduction in resting blood pressure (for review see Kenney & Seals, 1993). This post-exercise hypotension (PEH) has been found to persist for several hours after exercise, and can lower the blood pressure of hypertensive individuals into the normal range for much of the day following exercise, even in the presence of work-related stress (Brownley, West, Hinderliter, & Light, 1996). However, while PEH has been found within hours after exercise, it is not usually found minutes after, and more recent work has begun to examine sympathetic contributions to reduced reactivity, including reductions in catecholamine response (Brownley, Hinderliter, West, Girdler, Sherwood, & Light, 2003; Peronnet, Massicotte, Paquet, Brisson, & de Champlain, 1989). There is reason to think that, in our study, post-exercise hypotension is not responsible for the quick recovery. The exercise task by itself did not cause blood pressure to recover below baseline levels. Instead, blood pressure returned rapidly and precisely to pre-exercise levels. Glynn, Christenfeld, and Gerin (2002), using an exercise task identical to that used in this study, found the same effect. Moreover,

the participants who exercised in the absence of the stressor and participants who exercised after the stressor showed effectively identical recovery (despite the latter having significantly greater cardiovascular arousal at the start of the recovery period). If any post-exercise hypotension was simply masking the lingering effects of the psychological stressor, then these two groups should be very different. Instead it appears that the rapid recovery people show from exercise replaces the slow recovery they show from an emotional stressor when subjects do both.

A more subtle psychological process may also be at work. Exercising after a stressor could have the effect of interfering with the arousal-anger-rumination process. Interfering with people's post-stress anger, or rumination, may be sufficient to break the feed-forward process in which anger, angry thoughts, and autonomic activation sustain each other long after the actual anger-provoking event, and thus may improve blood pressure recovery (Glynn, Christenfeld, & Gerin, 2002). Instead of simply distracting people from emotional thoughts, exercise may reduce the emotional nature of the thoughts, by providing an alternative attribution for the arousal produced in response to the anger-provoking stimulus (Loftis & Ross, 1974; Nisbett & Schachter, 1966; Ross, Rodin, & Zimbardo, 1969; Schachter, 1964; Schachter & Singer, 1962; Storms & Nisbett, 1970). Insofar as individuals can credit some, if not all, of their arousal to positive invigoration from exercise rather than negative tension from a stressor, it is possible that exercise following a stressor can reduce stress responses.

Study Limitations

Because in this experiment the exercise started almost immediately after the stressor, it remains to be addressed how long after an event exercise can continue to be an effective form of stress management. This is an important question, since often

a lag is unavoidable. After an argument with one's spouse, one still needs time to put on a pair of sneakers before going for a walk, and one might need to wait hours to exercise after a crisis at work.

The current study suggests that even brief exercise can be effective, and it needs to be explored whether longer exercise works similarly, as well as whether other forms of exercise, differing in intensity and form, would also work. Nevertheless, while the exercise task used in this study was very brief and very light relative to other studies, these properties make it advantageous from a public health perspective. We chose to standardize the exercise task in terms of its form and duration, rather than its intensity. That is, one could adjust the exercise task for each individual so that it produces the same increases in heart rate or percentage of maximum VO_2. In this study, instead of holding the response constant, we held the stimulus constant. This method is more easily replicated and, as with the shorter duration of exercise, perhaps more easily adoptable by the public. However, future studies that employ real-world exercise, such as walking or running, would further contribute to the literature.

While the math task was an effective stressor, both in terms of self-report and cardiovascular measures, it is unknown if these results could generalized to other stressful situations (i.e., not just those that invoke anger or embarrassment, but perhaps grief or fear).

The present study was not designed to test the various potential mechanisms underlying the effects of exercise on stress recovery. Rather, it was designed to test whether exercise could improve stress recovery, as it has been shown to do with stress reactivity. Potential mechanisms are suggested and debated, but this was not

the focus of the study. Future work that is designed to test the various potential mechanisms underlying this effect is needed.

In conclusion, while research to date has looked at the impact of exercising before a stressor, this investigation demonstrates that exercising after a stressor can also limit the duration of cardiovascular arousal. Furthermore, within the framework of the cardiovascular reactivity hypothesis, this study has shown that interventions that follow a stressor, even those whose acute effect is to raise blood pressure, can still be effective in limiting the duration of cardiovascular responses, and thus have the potential to promote health.

Acknowledgement

Chapter 2, in full, is as it appears in Chafin, S., Christenfeld, N., & Gerin, W. Improving cardiovascular recovery from stress with brief post-stress exercise. (in press). *Health Psychology.* The dissertation author was the primary investigator and author of this paper.

Chapter 3

Study 2

There is accumulating evidence that even a single, brief session of exercise can contribute to mental and physical health. One benefit of acute aerobic exercise is that, following exercise, there is a period in which cardiovascular arousal to subsequent psychological stress is reduced (see recent review and meta-analysis by Hamer, Taylor, and Steptoe, 2006). This is significant within the framework of the cardiovascular reactivity hypothesis, which suggests that people who exhibit large cardiovascular responses to psychological stress are at risk for the development of hypertension and cardiovascular disease, and situations that lead to exaggerated responses put people at risk (Krantz & Manuck, 1984, 1986; Lovallo & Wilson, 1992).

All acute aerobic exercise studies to date have used the same basic procedure; all assess cardiovascular reactivity to a psychological stress task following acute aerobic exercise. It is also the case that, during exercise, subjects are unaware that a stress task will follow. This method has limited applied value. That is, because stress can be, and often is, unforeseen in the real world, one cannot plan to exercise before being subject to it. Rather, it seems likely that individuals use exercise as a coping mechanism in anticipation of a future stressor they are aware of, such as a job interview. Since anticipation of a stressor has effects that are similar to those associated with the stressor itself (Spacapan & Cohen, 1983), this is also relevant to the cardiovascular reactivity hypothesis, which suggests that the duration of the stress response, in addition to the magnitude of the initial peak reaction, may contribute to cardiovascular illness (Borghi, Costa, Boschi, Mussi, & Ambrosini, 1986; Gerin & Pickering, 1995; Haynes, Gannon, Orimoto, & O'Brien, 1997; Steptoe & Marmot, 2005).

Few studies (Rejeski, Thompson, Brubaker, & Miller, 1992; Hobson & Rejeski, 1993) have examined appraisal of threat in anticipation of a stressor. Following exercise or quiet rest, Rejeski, Thompson, Brubaker, & Miller (1992) gave participants instructions for a public speech. After preparing their comments, but before beginning the speech, subjects completed a thought listing procedure. Rejeski et al. found that subjects who had exercised reported fewer and less intense anxious thoughts in anticipation of the speech task. However here, as with other studies, instructions for the speech followed exercise; subjects were not anticipating the stressor while exercising.

One study has investigated the effects of exposing subjects to mental stressors while they are exercising. Roth, Bachtler, and Fillingim (1990) had subjects either exercise or not exercise, during which half of the subjects in each condition did a digits backward task. Scores on the *Profile of Mood States* (McNair, Lorr, & Droppelman, 1992) indicated that the exercising subjects, regardless of task exposure, reported less tension and more vigor than subjects who did not exercise. That is, subjects who were thinking about the stressor during the exercise still reported less anxiety than subjects in the stressor-alone condition. However, reported tension was higher in the exercise plus stressor condition than in the exercise-alone condition, suggesting the anxiolytic effect of exercise was weakened by the opposing effects of the stressor. Consistent with this, the stress task had an additive effect on the cardiovascular arousal experienced during exercise, such that heart rate was higher in subjects who were exercising while doing the digits backward task, than in subjects who exercised in the absence of the task. Cardiovascular reactivity to the same task administered after a period of recovery found exercise to neither exacerbate nor attenuate reactivity, after habituation to the task was taken

into account. Performing the same task twice, however, is not the same as anticipating the task and then doing it.

Anticipating the stressor during exercise may affect cardiovascular responses to the stressor for several reasons. For one, the psychological benefits of exercise may be moderated by preexisting psychological states. Rejeski, Gauvin, Hobson, and Norris (1995) found an interaction between subjective ratings of revitalization before and after exercise; only subjects with low to moderate ratings of revitalization before the exercise increased in this rating after the exercise. The authors suggest that there may not be a main effect of exercise, but rather the benefits of exercise may be dependent on prior mental health status. Similarly, O'Connor, Petruzello, and Robinson (1995) found that greater anxiety before exercise was correlated with greater reductions in anxiety after exercise. Here, if prior anxiety is a moderator of the stress attenuating effects of exercise, perhaps individuals who anticipate the stressor while exercising will benefit more from the exercise, and be less reactive to the stress task that follows, than those who are unaware of the stressor at the time of exercise.

By virtue of the opportunity to prepare more, anticipation may also increase various stress indicators before the stressor occurs, but decrease those same parameters during or after exposure. For example, in work done on anticipatory fear and recovery from surgery, patients given coping information, instruction, and encouragement recover better from surgery (Egbert, Battit, Welch, & Bartlett, 1964).

It is also possible that anticipating a stressor during exercise may work to reduce stress responses because of misattribution of arousal (Schachter, 1964); Schachter's theory posits that an emotion will occur when the person experiences autonomic arousal, and when the source of the arousal is ambiguous. Under these

conditions, the person makes a contextual attribution to some salient characteristic of the situation. For example, if an individual becomes nervous in anticipation of public speaking, it is possible that the side effects experienced as a result of feeling anxious (tremor, palpitations, rapid breathing) could be confused with similar manifestations of aerobic exercise. Since the stressor in this situation is not yet present, the most salient characteristic will be exercise. By misattributing any emotional arousal from anticipating the public speaking to arousal from a neutral source such as exercise, then perhaps the interpretation of the actual speaking will also be less emotional. As such, the magnitude and duration of the stress response could be reduced.

Therefore, to examine whether prior psychological arousal, from anticipating a stressor, will have an effect on subsequent physical arousal to exercise and the stressor itself, and because exercise while anticipating a stressor may capture more of an applied coping strategy, the purpose of this study was to assess the effect of exercising, while anticipating a stressor, on subsequent cardiovascular reactivity to that stressor. If the subject is informed of the stressor before the exercise begins, will the stress buffering effects of exercise, observed in prior studies, be replicated?

Method

Overview

Subjects briefly exercised before doing a speech task. Some subjects were informed of the speech, and anticipated it during the exercise, while other subjects were not informed. Other subjects exercised but did not do the speech, and the remaining did the speech without having exercised. See Table 2.1 for a schematic of the study design. Blood pressure and heart rate were monitored during each period of the study.

Subjects

Undergraduates at the University of California, San Diego (*N* = 86) participated for course credit (66 females, 20 males, age *M* = 21.13 years, *SD* = 4.97 years). No instructions were given prior to participation, other than a brief description of the study (i.e., "Your blood pressure will be measured while you perform several tasks."). No subject reported being in poor health or on any medications that might influence cardiovascular readings.

Procedure

Baseline. Upon the participant's arrival, the experimenter explained that the purpose of the study was to determine if mental and physical activities affect the cardiovascular system in different ways. Participants were told that they would do a mental task and that some, but not all, subjects would do a moderate exercise task as well. (All subjects were informed about the prospect of exercising to control for any inflation in cardiovascular measures during baseline due to anticipation of the upcoming exercise task.) After giving informed consent, the subject was seated and fitted with the finger cuff of the blood pressure monitor. The experimenter explained that the subject would sit for a rest (baseline) period, followed by a mental task, which would be followed by a brief activity period, and a longer rest (recovery) period. In order to get real-time information of what subjects were thinking about, the experimenter also instructed the participant to record his/her thoughts during the rest (i.e., baseline and recovery) periods by jotting down a few words whenever a knock (at minutes 1, 2.5, 4, and 7 during the baseline period and at minutes 1, 2.5, 4, 7, and 11 during the recovery period) was heard at the door. Using the knock as a signal enabled the experimenter to be absent from the room during the baseline and recovery periods, but still able to indicate when the thought samples were to be recorded. The participants were cautioned to write just enough to cue their memory

so that later, at the end of the study, they'd be able to explain in more detail what they had been thinking about to the experimenter. The experimenter also emphasized that she alone would read the results of these thought reports. This was important because the role of the person administering the math task was to provoke an emotional response; our past studies suggest that some of the thoughts reported during recovery related to anger at the provoker, and we did not want the participant to feel hesitant about reporting such thoughts. After instructing the subject to sit still during the ten minutes of the rest period, the experimenter left the room.

Exercise / Known Stress condition. After the baseline period, participants in the exercise / known stress condition were informed that, later in the study, they would give a speech on a controversial issue. Participants were given a list of pro and con arguments on the topic (see Appendix 1A) and instructed to choose a position to defend. Subjects were told to "focus both on the content of your speech (i.e., what you're going to say) and the delivery of your speech (i.e., how you're going to say it)". Note taking was not allowed. Subjects had five minutes to prepare the speech. Afterward, subjects were told they would have more time to prepare later, but first they were to participate in an exercise task, a three-minute seated walking in place task, which involved raising the left and right leg alternately to a specified height (25 centimeters) in time to a metronome (120 beats per minute). This procedure has been found to reliably elevate blood pressure in previous studies (Glynn, Christenfeld, and Gerin, 2002). Of more importance is that this task in isolation also leads to rapid blood pressure recovery, so that any sustained elevations in blood pressure during the recovery period, following the stressor and then the exercise task, are unlikely to be due to the exercise, but to the stressor. While this task is somewhat artificial, and is not actual walking or running, it does make it possible for the subject to maintain

the same posture as during all the other periods, and to maintain the position of the arm relative to the heart, enabling a direct comparison of cardiovascular scores across all periods, including the active exercise period.

Following the exercise task, subjects were given five more minutes to prepare for the speech, before the three-minute speech began. During the speech, if the participant stopped speaking, the experimenter asked that s/he continue, but did not comment on the speech or encourage the speaker in any way. We have used this procedure in a prior study (Glynn, Christenfeld, & Gerin, 1999), and found that it lead to reliably large increases in blood pressure and self-reported stress.

Exercise / Unknown Stress condition. The subjects in this condition were asked to evaluate, in their head, the same arguments that the subjects in the exercise / known stress condition were given, but the subjects in this condition were not informed they would be giving a speech. Subjects then participated in the same exercise task. Following the exercise task, subjects were told they would deliver a speech on the topic they had evaluated earlier and were given five minutes to prepare, before beginning the three-minute speech.

No Exercise / Known Stress condition. This condition was the same as the exercise / known stress condition, except that, instead of exercise, the subject tapped the index finger of their dominant hand to the same metronome beat used in the exercise task.

Exercise / No Stress condition. This condition, added later, assessed the effect of exercise alone, without the stressor present. Before the exercise, subjects were given the same instructions as the exercise / unknown stress condition, but following exercise, subjects did not prepare for or do the speech, but instead began the recovery period. While the subjects in this condition were not randomly assigned

with the other subjects, they were drawn in the same way from the same subject pool, and were identical in basic demographic characteristics.

Recovery. After the speech, the experimenter asked the subject to sit still for a final rest period, and to jot down a few words each time there was a knock at the door. The experimenter then left the room, and, at the five standardized times, knocked on the door. After 15 minutes, the experimenter returned, removed the finger cuff and interviewed the participant to have him or her expand on the thought reports. The participant then completed several questionnaires and was debriefed.

Subjective Measures

At the end of the recovery period, all subjects rated, on 7-point Likert-type scales, “How well did you do on the speech?” (1 = “very poor” to 7 = “very good”), “Did you feel prepared for the speech?” (1 = “not at all” to 7 = “very much so”), and “How stressful was the speech?” (1 = “not at all” to 7 = “very stressful”). Subjects were also asked “How often did you think about the speech during the last 15 minutes?” (1 = "not at all" to 7 = "the whole time").

Recording of Physiological Measures

Systolic and diastolic blood pressure and heart rate were recorded with an Ohmeda Finapres 2300 blood pressure monitor. Using the Peñaz method, this instrument measures beat-to-beat pressures with an inflatable finger cuff worn on the third finger of the non-dominant hand. The Finapres has proven to be a useful alternative to intra-arterial blood pressure measurement in laboratory testing (Imholtz, Settels, & Meiracker, 1990) and clinical practice (Gorback, Quill, & Lavine, 1991; Weiling, Harkel, & Lieshout, 1991). It is also able to track intra-arterial readings during abrupt changes of blood pressure (Parati, Casadei, & Groppelli, 1989). The

Finapres enhances reliability by collecting a large number of readings (Gerin, Pieper, & Pickering, 1993).

Data Reduction and Analysis Procedures

The beat-to-beat pressures from the Finapres were combined into minute averages across the experimental session. The cardiovascular dependent measures were change scores, computed using the difference between the minute averages for the period of interest and the mean of the pre-task baseline measurements. These means were computed using the pulse-based technique, in which equal weight is assigned to heart beats, rather than time intervals, resulting in greater weight given to the periods when the pulse is elevated (Glynn, Christenfeld, & Gerin, 1997). Raw change scores, rather than residualized change scores, were used (Llabre, Spitzer, & Saab, 1991).

We verified the initial equivalence of groups by comparing the mean of the pre-task baseline measurements with a separate one-way analysis of variance (ANOVA) for each of the cardiovascular measures. Manipulation and treatment effects were analyzed by comparing the mean change score for each period with a separate one-way ANOVA for each of the cardiovascular measures. An alpha level of .05 was used in the analyses.

Results

Baseline Measures

There was no significant difference between conditions during the baseline period for any of the cardiovascular measures (all Fs (3, 82) < 1.33, all ps > 0.27).

Effect of Being Informed (or Uninformed) of Upcoming Stressor

In the first preparation period, a *post hoc* Tukey HSD found those subjects who knew the arguments they were considering would be used in a later speech (i.e.,

the exercise / known stress and no exercise / known stress subjects) had significantly higher systolic blood pressure (F (3, 82) = 9.08, p < .001; see Figure 2.1), diastolic blood pressure (F (3, 82) = 10.08, p < .001; see Figure 2.2), and heart rate (F (3, 82) = 5.45, p < .005; see Figure 2.3) than subjects who were not informed.

Effect of Exercise on Blood Pressure and Heart Rate

There were significant differences between conditions during the exercise period for all of the cardiovascular measures. A *post hoc* Tukey HSD found subjects who did exercise had significantly higher systolic blood pressure (F (3, 82) = 19.81, p < .001), diastolic blood pressure (F (3, 82) = 12.88, p < .001), and heart rate (F (3, 82) = 57.64, p < .001) than those subjects who sat still.

Speech Preparation

To determine if prior exercise had an effect on cardiovascular measures during the period when subjects were preparing for the speech, we analyzed the difference between the exercise / known stress, exercise / unknown stress, and no exercise / known stress groups. The subjects who did not do the speech (exercise / no stress) were not considered in the analyses here, as the recovery period had begun for this group. Just before the speech began, subjects who did exercise had significantly higher systolic blood pressure (F (2, 66) = 7.60, p < .001) and heart rate (F (2, 66) = 11.15, p < .001) during the preparation period than subjects who did not exercise. The pattern was the same, but not significant for diastolic blood pressure (F (2, 66) = 0.82, p > .40).

Effect of Experimental Condition on Cardiovascular Reactivity to Stressor

If it were the case that prior exercise attenuates cardiovascular reactivity to a stressor, we'd expect that those subjects who did exercise before the speech had less arousal to the speech than those who did not exercise. However, the opposite

effect was found. During the speech, the subjects who did exercise prior to the speech (exercise / known stress and exercise / unknown stress) had higher systolic blood pressure during the speech than the subjects who did not exercise (F (2, 66) = 3.94, $p < .05$; see Table 2.2). A *post hoc* Tukey HSD showed a significant difference between those who exercised but did not know about the speech beforehand (exercise / unknown stress) and those who did not exercise but knew about the speech (no exercise / known stress), $p < 0.05$, and with marginal significance, between those who exercised and knew about the speech (exercise / known stress) and those who did not exercise but knew about the speech (no exercise / known stress), $p < 0.10$. The same trend was found for heart rate (F (2, 66) = 5.90, $p < .005$; see Table 2.2) and, although the difference was not significant, for diastolic blood pressure, (F (2, 66) = 1.18, $p > .05$; see Table 2.2).

Effect of Experimental Condition on Cardiovascular Recovery

During the recovery period, systolic blood pressure was significantly higher (F (3, 82) = 4.41, $p < .01$) in those subjects who had exercised and done the speech (exercise / known stress and exercise/ unknown stress) than in those subjects who had exercised and not done the speech (exercise / no stress). The trend is the same for diastolic blood pressure (F (3, 82) = 3.27, $p < .05$), although a *post hoc* Tukey HSD revealed a significant difference only between those who did exercise but did not know about the speech (exercise / unknown stress) and those who did exercise but did not do the speech (exercise / no stress), $p < .05$. Heart rate was significantly higher in the groups of subjects who did exercise, compared to subjects who gave a speech without prior exercise (F (3, 82) = 10.16, $p < .001$).

Self-Report

Stressor. There were no significant differences between conditions in how

well subjects felt they did on the speech, F (2, 66) = 2.31, p > 0.10. The average response was 3.0 on a seven-point Likert-type scale in which “7” was very good. Nor were there significant differences between conditions in how prepared subjects felt (F (2, 66) = 1.51, p > 0.20. It is worth noting that, while subjects who were told of the speech before the exercise (exercise / known stress) had 5 more minutes to prepare for it, they did not differ from subjects who were not told of the speech before the exercise (exercise / unknown stress) in how prepared they felt for it, t (45) = 0.03, p > 0.98. The average response was 3.0 on a seven-point Likert-type scale in which “7” was very prepared. Subjects did not differ in how stressful they found the speech, F (2, 66) = 0.01, p > 0.98. The average response was 3.8 on a seven-point Likert-type scale in which “7” was most stressful.

Rumination. There was not a significant difference in how much subjects reported thinking, during the recovery period, about the stressor, F (2, 66) = 1.44, p > 0.20. The average response was 2.7, where “7” indicated that they were thinking about the stressor the whole time.

Thought sampling. Subjects who did exercise and were told about the speech ahead of time (exercise / known stress) reported significantly more (F (2, 66) = 3.17, p < .05) emotional thoughts (an average of .83 emotional thoughts per person) about the speech (for example, “That was a very stupid speech,” “I hate giving speeches,” “That was so stressful and I didn’t mean a thing I said”) than subjects who were told about the speech ahead of time but did not exercise (no exercise / known stress) (an average of .20 emotional thoughts per person). The trend was the same, but not significant (p > .15, an average of .70 emotional thoughts per person) for the subjects who did exercise but were not told about the speech (exercise / unknown stress). This is inconsistent, however, with our other measure of rumination, in which subjects

reported no difference in how often they thought about the speech. This disagreement may be due to the point in time that the measures were taken; the thought sampling was a real time measure, the self-report was retrospective.

Discussion

The purpose of this study was to determine if the stress-buffering effects of exercise could be replicated when the subject was aware of the stressor before the exercise began. Our results suggest that anticipating the stressor during the exercise does not produce any more, or less, arousal to the stressor than not anticipating it. However, this conclusion must be taken with some caution, as exercise did not have a buffering effect in either group. Exercising before the stressor, whether the stressor was anticipated or not, actually increased arousal to the stressor, relative to subjects who did not exercise. Unlike in prior studies, instead of suppressing autonomic arousal to the stressor, having exercised seemed to have primed it. Furthermore, this was not carryover from the exercise, as the subjects who exercised without the stressor recovered back to baseline levels within minutes after the task.

The inconsistencies among studies may be due to a key methodological difference. In our study, subjects began the speech preparation period immediately after the exercise. In other studies, the attenuation of the stress response is only found in those studies that have a delay that allows for cardiovascular measures to return to, or below, baseline before the stressor begins. This delay has ranged from 5 minutes post-exercise (Russell, Epstein, & Erickson, 1983) to 24 hours post-exercise (Ebbesen, Prkachin, Mills, & Green, 1992). Hamer, Taylor, and Steptoe (2006) found consistent effect sizes when the stress task was administered up to half an hour after the exercise, with diminishing effects as the delay increased. It is unclear, however, whether the physiological indices of arousal from exercise must be

given adequate time to recover to the same level across groups before the stressor begins. That is, is there an optimal period of delay between the offset of the exercise and the onset of the stressor? And if residual arousal from exercise is present when the stressor begins, does this undermine the benefit of exercise?

Other studies, in the misattribution literature, have also found exercise to prime the emotional state. Zillmann's (1971) excitation transfer theory uses Schachter's (1962) misattribution of arousal paradigm, but instead of misleading subjects about the source of arousal, an interval is built in between the source of sympathetic arousal (most often exercise) and the emotion-provoking situation. Based on the idea that sympathetic arousal diminishes more slowly than the situation that caused it, subjects feel their arousal should no longer be due to the exercise, but instead to the emotion-provoking situation that follows. Physical exercise has been shown to increase aggressive retaliation to prior provocation (Zillmann & Bryant, 1974; Zillmann, Katcher, & Milavsky, 1972; Zillmann, Johnson, & Day, 1974), to enhance sexual excitement to erotic films (Cantor, Zillmann, & Bryant, 1975), to increase liking for an attractive female and disliking for an unattractive female (White, Fishbein, & Rutstein, 1981), and to increase negative mood induced by the reading of negative self-referenced statements (Reisenzein & Gattinger, 1982).

Here, the conditions for excitation transfer seem to be met. That is, there is an interval (the speech preparation period) between the source of sympathetic arousal (the exercise) and the source of emotional arousal (the speech), in which residual arousal (blood pressure and heart rate) from the source of sympathetic arousal is still present. During the speech preparation period, the subject has two possible interpretations for the arousal: the exercise or the speech. Since the speech is most salient to the subject, as it is to occur in just a few minutes, perhaps the

subject misattributes the residual arousal from the exercise to the speech task. Our measures of rumination support this explanation. That is, subjects who did exercise had significantly more emotional thoughts about the stressor than subjects who did not exercise.

In excitation transfer studies that find an exaggeration of the stress response, blood pressure is above baseline at the time of the stressor. In acute aerobic exercise studies that find an attenuation of the stress response, blood pressure at the time of the stressor is either at or below baseline. Therefore, Study 3 builds in a period of delay after the exercise, so that residual arousal from exercise is no longer present when the speech preparation period begins.

Study 3

Method

Overview

Subjects briefly exercised before doing a speech task. Some subjects were informed of the speech, and anticipated it during the exercise, while other subjects were not informed. Other subjects exercised but did not do the speech, or did the speech without having exercised. Unlike the prior study, following the exercise there was a delay of five minutes of rest before the speech preparation began. See Table 3.1 for a schematic of the study design. Blood pressure and heart rate were monitored during each period of the study.

Subjects

Undergraduates at the University of California, San Diego (N = 83) participated for course credit (61 females, 22 males, age M = 20.26 years, SD = 2.09 years). No instructions were given prior to participation, other than a brief description of the study (i.e., “Your blood pressure will be measured while you perform several

tasks.”). No subject reported being in poor health or on any medications that might influence cardiovascular readings.

Procedure

The procedure was the same as in the prior study, except that, following exercise, subjects sat in silence for five minutes. During this period, subjects in the exercise / unknown stress group remained uninformed about the upcoming stressor.

Results

Baseline Measures

There was no significant difference between conditions during the baseline period for any of the cardiovascular measures (all *F*s (3, 79) < .55, all *p*s > 0.65).

Effect of Being Informed (or Uninformed) of Upcoming Stressor

In the first preparation period, a *post hoc* Tukey HSD found those who knew the arguments they were considering would be used in a later speech (i.e., the exercise / known stress and no exercise / known stress subjects) had significantly higher systolic blood pressure (F (3, 79) = 6.47, p = 0.001; see Figure 3.1), diastolic blood pressure (F (3, 79) = 10.31, p = 0.000; see Figure 3.2), and heart rate (F (3, 79) = 11.69, p = 0.000; see Figure 3.3) than subjects who were not informed.

Effect of Exercise on Blood Pressure and Heart Rate

There were significant differences between conditions during the exercise period for all of the cardiovascular measures. A *post hoc* Tukey HSD found subjects who did exercise had significantly higher systolic blood pressure (F (3, 79) = 2.86, p < 0.05), diastolic blood pressure (F (3, 79) = 3.94, p < 0.01), and heart rate (F (3, 79) = 23.41, p < 0.000) than subjects who sat still.

Effect of Delay After Exercise on Blood Pressure and Heart Rate

There were no significant differences in systolic blood pressure (F (3, 79) = 1.10, $p > 0.35$) or heart rate (F (3, 79) = 1.37, $p > 0.25$) between conditions during the delay period after exercise. However, there was a significant difference in diastolic blood pressure (F (3, 79) = 4.35, $p < 0.01$). A *post hoc* Tukey HSD found exercise to return diastolic blood pressure closer to baseline than not exercising. Subjects who did not exercise but did anticipate the speech (no exercise / known stress) had significantly higher diastolic blood pressure during the delay period than both the subjects who did exercise but did not know about the speech (exercise / unknown stress) and the subjects who did exercise but did not do the speech (exercise / no stress), $p < 0.05$. The trend was the same, but with marginal significance, $p < 0.10$, for subjects who did exercise and did know about the speech (exercise / known stress). As we were looking for the point at which the blood pressure measures for all conditions had stabilized, we excluded, in these analyses, the first two minutes of the delay period in the analysis.

Speech Preparation

There were no significant differences in systolic blood pressure (F (2, 55) = 2.13, $p > 0.12$) or heart rate (F (2, 55) = 0.25, $p > 0.78$) between conditions during the preparation period just before the speech began. However, there was a difference of marginal significance in diastolic blood pressure (F (2, 55) = 2.64, $p < 0.09$). A *post hoc* Tukey HSD found that those subjects who had been told of the speech but did not exercise afterward (no exercise / known stress) had higher diastolic blood pressure, $p < 0.07$, during the preparation period than subjects who had had been told of the speech and did exercise afterward (exercise / known stress). The subjects who did not do the speech were not included in the analyses here, as the recovery period had begun for this group.

Effect of Experimental Condition on Cardiovascular Reactivity to Stressor

If it were the case that prior exercise attenuates cardiovascular reactivity to a stressor, we'd expect that those subjects who did exercise before the speech had less arousal to the speech than those who did not exercise. This is what we found. The subjects who did not exercise prior to the speech (no exercise / known stressor) had higher diastolic blood pressure during the speech than the subjects who did exercise (F (2, 55) = 3.64, $p < 0.05$; see Table 3.2). The pattern was the same, but not significant for systolic blood pressure (F (2, 55) = 1.44, $p > 0.24$; see Table 3.2) and heart rate (F (2, 55) = 0.85, $p > 0.43$; see Table 3.2).

Effect of Experimental Condition on Cardiovascular Recovery

During the recovery period, diastolic blood pressure was significantly higher in subjects who did do the speech (F (3, 79) = 6.18, $p < 0.001$) than in subjects who did not do the speech (exercise / no stress). Systolic blood pressure (F (3, 79) = 3.76, $p < 0.05$) and heart rate (F (3, 79) = 3.17, $p < 0.05$) were significantly higher as well, but only in the group of subjects who did the speech but did not exercise (no exercise / known stressor).

Self-Report

Stressor. There were no significant differences between conditions in how subjects felt they did on the speech, F (2, 53) = 1.27, $p > 0.28$. The average response was 3.3 on a seven-point Likert-type scale in which "7" was very good. Nor were there significant differences between conditions in how prepared subjects felt (F (2, 53) = .64, $p > 0.53$. The average response was 3.3 on a seven-point Likert-type scale in which "7" was very prepared. It is worth noting that, while subjects who were told of the speech before the exercise (exercise / known stress) had 5 more minutes to prepare for it, they did not differ from subjects who were not told of the speech

before the exercise (exercise / unknown stress) in how prepared they felt for it, t (41) = -0.94, $p > 0.35$. Subjects did not differ in how stressful they found the speech, F (2, 53) = 0.81, $p > 0.44$. The average response was 3.5 on a seven-point Likert-type scale in which "7" was most stressful.

Rumination. There was not a significant difference in how much subjects reported thinking, during the recovery period, about the stressor, F (2, 53) = .17, $p > 0.84$. The average response was 2.7, where "7" indicated that they were thinking about the stressor the whole time.

Thought sampling. There was not a significant difference in the number of emotional thoughts subjects reported about the speech (F (2, 53) = .06, $p > 0.94$).

Discussion

By including the delay period, so that residual arousal from exercise was no longer present when the speech preparation period began, subjects who exercised prior to the speech had less cardiovascular arousal to the speech than subjects who did not exercise. This is consistent with most studies in the exercise literature. Taken together, Study 2 and Study 3 suggest an interaction of delay and having exercised; relative to those who do not exercise, no delay between exercise and stress seems to prime the stress response (Study 2) whereas a delay between exercise and stress attenuates the stress response (Study 3).

While excitation transfer theory predicts that a delay will eliminate the emotional priming effects and the increase in arousal, it does not account for the delay having beneficial effects, such that arousal is lessened in subjects who did the exercise task before the speech, relative to those who did not. Excitation transfer fits as an explanation for Study 2 (participants felt their arousal was no longer due to the exercise, but instead to the emotion-provoking situation that followed). However, it

does not offer an explanation for why further delay makes subjects less reactive to the stressor. The theory of misattribution of arousal may suggest why adding in a delay reverses the excitation transfer effect, but only for those subjects who knew of the stressor in advance, by altering the anticipation of the stressor. That is, if subjects were able to misattribute their arousal from anticipating the speech task to the exercise task, then perhaps subjects going into the speech task felt less tense, the interpretation of the speech task was less emotional, and the magnitude of the stress response was reduced. However, the theory does not suggest why subjects who did not know of the stressor would have a similar response. The theory predicts that for these subjects, the "have just exercised" misattribution would be most salient without a delay. By adding in a delay, exercise provides some sort of protective effect that misattribution of arousal can't explain and suggests there may be some other factor at work. Similarly, it is unlikely that misattribution of arousal can explain findings from other studies in which stress-induced arousal is diminished hours after exercise (Ebbesen, Prkachin, Mills, & Green, 1992).

In both Study 2 and Study 3, knowing about the stressor beforehand did not affect later cardiovascular responses to the stressor. The manipulation was effective; subjects who knew of the speech had more arousal during the first preparation period, with the pattern reversing during the second preparation period. It is notable that, while subjects who knew about the speech had a greater duration of the stress response, both in the first preparation period and the second preparation period, subjects who did not know about the speech had a greater magnitude of the stress response, once they were told of the speech. However, during the speech the known and unknown stress groups did not differ. Nor were there differences in how stressful subjects found the task. Unfortunately, our self-reports were retrospective; subjects

may have reported more anxiety about the speech if we had asked them before it began, during the preparation period. Also, while subjects who were informed of the speech did have five more minutes to prepare for it, subjects who were not informed had the same amount of time with the material (albeit they did not know they would be speaking about it later). Perhaps if these subjects had had less time with the material, and felt less prepared for the speech, it would have better replicated the stressors used in prior studies, and we would have been more likely to find a difference in cardiovascular reactivity in those who knew the stressor was coming and those who did not know.

All the same, this study suggests that exercise is successful in attenuating cardiovascular arousal to a stressor even if the individual is informed of the stressor beforehand. It is also worth noting that this study provides a partial replication of Study 1, but with a different stressor. That is, being informed of an upcoming speech was in itself a stressor, as indicated by the significant increase in cardiovascular arousal for the known stress groups. For some subjects, exercise then followed this stressor, as it did in Study 1. During the delay period following the exercise period, subjects who exercised, whether they knew of the speech or not, had better recovery than subjects who knew of the upcoming speech, but did not exercise. Granted, this effect was not as strong in subjects who exercised after being informed of the speech, most likely because they continued to anticipate the speech during the delay period. However, the results suggest that, as with Study 1, exercising after a stressor aids in recovering from that stressor, even when another (related) stressor is known to be approaching.

Chapter 4

STUDY 4

According to Schachter (1964), the experience of an emotion is contingent on feeling sympathetic arousal and labeling that arousal as coming from an emotional source. If an individual experiences sympathetic arousal without explanation, he will search his environment for the cause. Depending on how he comes to label the source of his arousal, the individual can experience very different emotions or no emotion at all. On one end, he can come to imbue a source that would not otherwise produce an emotion with emotional properties, which will cause him to become more emotional (Schachter & Singer, 1962; Schachter & Wheeler, 1962). In the classic Schachter and Singer experiment (1962), state of arousal was manipulated with an injection of epinephrine. Subjects not informed of its sympathomimetic effects (tremor, palpitations, rapid breathing), later found a confederate's mood (either euphoria or anger) more contagious, than subjects who had been informed of the drug's effects or who had been injected with a placebo. On the other end, an individual can come to attribute emotional arousal to a nonemotional or neutral source, which will cause him to become less emotional (Nisbett & Schachter, 1966; Ross, Rodin, & Zimbardo, 1969; Storms & Nisbett, 1970; but see Reisenzein, 1983, for criticism).

While speculative, it is possible that this misattribution effect can explain why, following exercise, there is a period in which individuals are less responsive to emotional or stressful stimuli. In a study by Duda, Sedlock, Melby, and Thaman (1988), active individuals had lower heart rate responses to exercise, but higher ratings of anxiety to a subsequent stressor. Perhaps, low fitness individuals, still feeling the arousing effects of the exercise, blamed the exercise for their feelings of

arousal rather than the stressor, and felt less anxious as a result. That is, subjects may have labeled their arousal following the exercise as positive feelings of vigor, rather than negative feelings of tension from the stressor. Such an interpretation would be consistent with Roth's (1989) investigation that found fewer self-reported feelings of anxiousness, even with increased cardiovascular arousal, when exercise and stressor were presented together. Likewise, after exercising, subjects in an investigation by Rejeski, Thompson, Brubaker, & Miller (1992) reported fewer anxious thoughts in anticipation of a speech task than did subjects who had not exercised. Perhaps, if subjects became nervous at the thought of public speaking, the side effects experienced as a result of feeling anxious (tremor, palpitations, rapid breathing) were confused with similar manifestations of aerobic exercise. Similarly, in a study by Koltyn, Garvin, Gardiner, and Nelson (1996), after exercising, subjects had a higher pain threshold and lower pain ratings throughout a pain trial in which pressure was applied to a forefinger. While it is possible that this analgesia may be due to endogenous opioids (Haier, Quaid, & Mills, 1981), it is also possible that subjects were able to misattribute their pain from the finger weight to lingering discomfort from the exercise; Nisbett and Schachter (1966) found that subjects who were told to expect sympathomimetic effects after swallowing a placebo pill were able to tolerate more pain from electric shock, by misattributing their shock-induced arousal to the pill, than subjects who were led to expect irrelevant effects.

A wide variety of studies, covering a large range of emotional reactions, has demonstrated that if one is able to attribute arousal from an emotional source to a neutral source, the perception of the emotion is weakened. In Study 1, we found that although exercising after a stressor adds to initial cardiovascular arousal, it goes on to facilitate cardiovascular recovery afterward. It is possible that exercise has this

effect by providing an alternative attribution for the arousal produced in response to the stress task; this neutral attribution (i.e., “have just exercised”) may reduce anxiety about the task, and as a result, reduce arousal.

If exercise works because of misattribution of arousal, then other neutral tasks that result in misattribution of arousal ought to improve cardiovascular recovery as well. However, if exercise reduces arousal by some mechanism other than misattribution of arousal, then exercise will be a unique activity, and other neutral tasks will not improve cardiovascular recovery. The present study will test these competing hypotheses. We will expose subjects to an emotional source of arousal, in the form of a stressor task, and then have subjects engage in various other tasks that may lead to misattribution. Some subjects will perform the same exercise task as used in Study 1, with the hope of replicating the effect. Using an explicit misattribution manipulation, other subjects will be told to expect signs of sympathetic arousal during an emotionally neutral task. Such instructions have been used in prior studies and have been shown to produce misattribution effects (Loftis & Ross, 1974; Olson, 1988). For example, Ross, Rodin, and Zimbardo (1969) demonstrated that when subjects were led to misattribute their fear from an impending shock to background noise, they spent less time trying to avoid the shock. We will also test an implicit misattribution manipulation; instead of misleading subjects about sympathetic arousal, we hope to elicit these symptoms by having subjects perform a physically arousing, but emotionally neutral task. Still other subjects will do a distraction task, as exercise may provide a diversion from distressing thoughts or activities, making it difficult for subjects to ruminate while exercising (Bahrke & Morgan, 1978; Raglin & Morgan, 1987).

If our hypothesis is correct, recovery will be better in those subjects who exercise or do one of the misattribution tasks. If subjects are given an alternative attribution for the arousal they experience in response to the stress task, then subjects may ruminate about the stress task less, and recovery will be improved. However, it were as simple as thinking about the stressor task less, one would expect better recovery from the distracting task as well (Bahrke & Morgan, 1978; Raglin & Morgan, 1987). Our hypothesis predicts that the distracting task will work to decrease rumination only during the task itself (Glynn, Christenfeld, & Gerin, 2002); if it is spontaneous rumination after the stressor that maintains cardiovascular arousal, then preventing rumination with distraction will decrease that arousal. Yet once the source of distraction is removed, spontaneous rumination will reoccur. In contrast, we predict that the misattribution tasks will improve recovery not during the task, but after the task.

Method

Overview

Participants performed a serial subtraction task while being harassed by an experimenter. Participants were then randomly assigned to one of five conditions: exercise, explicit misattribution (white noise), implicit misattribution (video game), distraction, or a silence control. See Table 4.1 for a schematic of the study design. Blood pressure and heart rate were monitored during baseline, stressor, manipulation, and recovery periods.

Subjects

Undergraduates at the University of California, San Diego (N = 132) participated for course credit (91 females, 41 males, age M = 19.94 years, SD = 1.78 years). No instructions were given prior to participation, other than a brief description

of the study (i.e., “Your blood pressure will be measured while you perform several tasks.”). No subject reported being in poor health or on any medications that might influence cardiovascular readings.

Procedure

Baseline. Upon the participant’s arrival, the experimenter explained that the participant’s blood pressure would be monitored during an arithmetic task and that some, but not all, subjects would participate in a moderate exercise task as well. (All subjects were informed about the prospect of exercising to control for any inflation in cardiovascular measures due to anticipation of the upcoming exercise task.) After giving informed consent, the subject was seated and fitted with the finger cuff of the blood pressure monitor. The experimenter explained that the subject would sit for a rest (baseline) period and then a different experimenter would administer a serial subtraction task, which would be followed by a brief activity period (possibly exercise), and a longer rest (recovery) period. In order to get real-time information of what subjects were thinking about, the experimenter also instructed the participant to record his/her thoughts during the rest (i.e., baseline and recovery) periods by jotting down a few words whenever a knock (at minutes 1, 2.5, 4, and 7 during the baseline period and at minutes 1, 2.5, 4, 7, and 11 during the recovery period) was heard at the door. Using the knock as a signal enabled the experimenter to be absent from the room during the baseline and recovery periods, but still able to indicate when the thought samples were to be recorded. The participants were cautioned to write just enough to cue their memory so that later, at the end of the study, they’d be able to explain what they had been thinking about to the experimenter. The experimenter also emphasized that she alone would read the results of these thought reports. This was important because the role of the person administering the math task was to

provoke an emotional response; our past studies suggest that some of the thoughts reported during recovery related to anger at the provoker, and we did not want the participant to feel inhibited from recording such thoughts. After instructing the subject to sit still during the ten minutes of the rest period, the experimenter left the room.

Stressor task. After baseline, another experimenter, blind to the condition of the participant, entered the room and administered the mental arithmetic task. The subject was asked to count backward out loud by 13s from 2,397. Thirty seconds into the task, the experimenter informed the subject that his/her counting was too slow and that the task should be started again, at a faster pace. Similar interruptions informing the subject of deficient performance continued approximately every thirty seconds for three minutes. Each response was scheduled and standardized, so that each subject heard the same criticism at the same time. This task has been shown to be an effective stressor in several studies (Glynn, Christenfeld, & Gerin, 2002; Allen & Blascovich, 1994; Allen, Obrist, Sherwood, & Crowell, 1987), and has the potential to evoke more than one emotion, including anger and embarrassment. After the stressor ended, the experimenter who conducted the math task left the room and the original experimenter re-entered, to explain the instructions for the manipulation.

Exercise manipulation. Following the stressor, subjects performed a three-minute seated walking in place task, which involved raising the left and right leg alternately to a specified height (25 centimeters) in time to a metronome (120 beats per minute). This procedure has been found to reliably elevate blood pressure in previous studies (Glynn, Christenfeld, and Gerin, 2002). Of more importance is that this task in isolation also leads to rapid blood pressure recovery, so that any sustained elevations in blood pressure during the recovery period, following the stressor and then the exercise task, are unlikely to be due to the exercise, but to the

stressor. While actual walking or running is a more likely form of exercise in the real world, this task enabled the subject to maintain an erect posture, and to maintain the position of the arm, relative to the heart.

Implicit misattribution (video game) manipulation. Subjects played a well-known video game, Pac-Man, for three minutes. Subjects were encouraged to do the best they could on the game, but were told, in order to minimize evaluation apprehension, that their score would not be recorded.

Explicit misattribution (white noise) manipulation. Subjects were fitted with headphones and were exposed to loud (approximately 80 dB) white noise for three minutes. In an attempt to have the subject misattribute any autonomic arousal they were feeling to the noise and not the previous, stressful task, the subject was informed that we were required, by the Human Subjects office, to inform them of some of the typical physiological responses to noise bombardment including tremor, palpitation, increased breathing, and a sinking feeling in the pit of the stomach (see Appendix 1B). Such instructions have been used in prior studies and have been shown to produce misattribution effects (Loftis & Ross, 1974; Olson, 1988; Ross, Rodin, & Zimbardo, 1969).

Distraction manipulation. Subjects read a moral-dilemma scenario and then responded to questions about the scenario for three minutes (see Appendix 1C). The scenario was adapted from Colby and Kohlberg's classic moral judgment interview (Colby & Kholberg, 1987) and has been used as an effective distractor in prior research (Glynn, Christenfeld, & Gerin, 2002). This task was intended to be engaging but not stressful. In order to minimize evaluation apprehension, explicit instructions informed the participant, "this is about your opinion; there are no right or wrong answers and you won't be judged on your performance."

Silence manipulation. Subjects were instructed to sit quietly for three minutes. This condition served as the control group for the study.

Recovery. In both the control and experimental conditions, at the end of the manipulation, the experimenter asked the subject to sit still for a final rest period, only jotting down a few words when a knock was heard at the door. The experimenter then left the room, knocking on the door at the five standardized times. After 15 minutes, the experimenter returned, removed the finger cuff and interviewed the participant about the thought reports. The participant then completed several questionnaires and was debriefed.

Subjective Measures

At the end of the recovery period, all subjects rated, on 7-point Likert-type scales, “How difficult was the arithmetic task?” (1 = “not at all difficult” to 7 = “very difficult”), and “How stressful was the arithmetic task?” (1 = “not at all stressful” to 7 = “very stressful”). Subjects were also asked “How often did you think about the arithmetic task during the last 15 minutes?” (1 = "no time at all" to 7 = "the whole time") and “How often did you think about the other task (exercise, video game, noise bombardment, questionnaire) during the last 15 minutes?” (1 = "no time at all" to 7 = "the whole time").

Recording of Physiological Measures

Systolic and diastolic blood pressure and heart rate were recorded with an Ohmeda Finapres 2300 blood pressure monitor. Using the Peñaz method, this instrument measures beat-to-beat pressures from an inflatable finger cuff worn on the third finger of the non-dominant hand. The Finapres has proven to be a useful alternative to intra-arterial blood pressure measurement in laboratory testing (Imholtz, Settels, & Meiracker, 1990) and clinical practice (Gorback, Quill, & Lavine, 1991;

Weiling, Harkel, & Lieshout, 1991). It is also able to track intra-arterial readings during abrupt changes of blood pressure (Parati, Casadei, & Groppelli, 1989). The Finapres enhances reliability by collecting a large number of readings (Gerin, Pieper, & Pickering, 1993).

Data Reduction and Analysis Procedures

The beat-to-beat pressures from the Finapres were combined into minute averages across the experimental session. The cardiovascular dependent measures were change scores, computed using the difference between the minute averages for the period of interest and the mean of the pre-task baseline measurements. These means were computed using the pulse-based technique, in which equal weight is assigned to heart beats, rather than time intervals, resulting in greater weight given to the periods when the pulse is elevated (Glynn, Christenfeld, & Gerin, 1997). Raw change scores, rather than residualized change scores, were used (Llabre, Spitzer, & Saab, 1991).

We verified the initial equivalence of groups by comparing the mean of the pre-task baseline measurements with a separate one-way analysis of variance (ANOVA) for each of the cardiovascular measures. Manipulation and treatment effects were analyzed by comparing the mean change score for each period with a separate one-way ANOVA for each of the cardiovascular measures. An alpha level of .05 was used in the analyses.

Results

Baseline Measures

There was no significant difference between conditions during the baseline period for any of the cardiovascular measures (all Fs (4, 127) < 2.11, all ps > 0.08). There was a difference in systolic blood pressure of marginal significance, $p < .14$;

participants in the implicit (video game) misattribution condition had a higher baseline average than subjects in the control condition.

Stress Manipulation Check

The serial-subtraction task was effective as a stressor for all conditions, with average task increases of 26.0 mmHg systolic blood pressure, 16.5 mmHg diastolic blood pressure, and 11.0 bpm heart rate. There was no significant difference between conditions in the cardiovascular measures during the stress manipulation (all *F*s (4, 127) < 1.06, all *p*s > 0.37).

Effect of Manipulation on Blood Pressure and Heart Rate

There were significant differences between conditions during the manipulation period for all of the cardiovascular measures. A *post hoc* Tukey HSD found subjects who did exercise to have significantly higher systolic blood pressure (F (4, 127) = 31.80, *p* = .000; see Table 4.2 and Figure 4.1), diastolic blood pressure (F (4, 127) = 24.85, *p* = .000; see Figure 4.2) and heart rate (F (4, 127) = 117.94, *p* = .000; see Figure 4.3) than subjects who did not.

Effect of Experimental Condition on Blood Pressure and Heart Rate Recovery

There was a significant effect of having exercised in improving recovery on both systolic blood pressure (*F* (4, 127) =2.60, *p* < 0.05) and diastolic blood pressure (*F* (4, 127) = 4.91, *p* < .001) during the last ten minutes of the recovery period. A *post hoc* Tukey HSD showed a significant difference in systolic blood pressure between the exercise and video game conditions, *p* < 0.05, with exercising after stress returning systolic blood pressure and diastolic blood pressure closer to baseline. Diastolic blood pressure followed the same pattern, *p* < 0.001.

Heart rate was significantly higher (*F* (4, 127) = 5.06, *p* = 0.001) in the exercise group than in all other groups except for the video game group, possibly as

a result of heart rate compensating for blood pressure, in line with other studies (Rejeski, Thompson, Brubaker, & Miller, 1992). It is worth noting, however, that the heart rate difference diminishes toward the end of the recovery period, while the blood pressure difference does not.

As with Study 1, to allow for a stable comparison across groups, the last ten minutes of the recovery period is used in the analyses of cardiovascular recovery. This was a post hoc decision, as we were looking for the point at which the blood pressure measures for all conditions had leveled off.

Self-Report

Stressor. There were no significant differences between conditions in how difficult subjects found the math task, F (4, 127) = 0.31, $p > 0.87$. The average response was 5.4 on a seven-point Likert-type scale in which "7" was most difficult. Nor were there significant differences between conditions in how stressful subjects found it, F (4, 127) = 0.42, $p > 0.79$. The average response was 4.9 on a seven-point Likert-type scale in which "7" was most stressful.

Exercise manipulation check. When asked, "Did the exercise increase your level of physical arousal?" 100 percent of subjects wrote that it did. We also asked, "How much physical arousal (e.g., increased heart rate, breathing, etc.) did you experience during the exercise?" The average response was 6.2 on a Likert-type scale in which 1 was "none" and 7 was "a lot". We also asked subjects if their physical arousal remained high during the recovery period, and if so, what the arousal was due to. Of the 45 percent who reported arousal, 44 percent wrote it was due to the exercise.

Distraction manipulation check. When asked, "While you were answering the moral dilemma questionnaire, did you think about the arithmetic task?" 88 percent of

subjects wrote they did not. We also asked subjects if their physical arousal remained high during the recovery period, and if so, what the arousal was due to. Of the 57 percent who reported arousal, none wrote it was due to the questionnaire.

Video game manipulation check. When asked, “Did the video game increase your level of physical arousal?” 77 percent of subjects wrote that it did. We also asked, “How much physical arousal (e.g., increased heart rate, breathing, etc.) did you experience during the video game?” The average response was 3.5 on a Likert-type scale in which 1 was “none” and 7 was “a lot”. We also asked subjects if their physical arousal remained high during the recovery period, and if so, what the arousal was due to. Of the 63 percent who reported arousal, 29 percent wrote it was due to the video game.

White noise manipulation check. When asked, “Did you experience any of the noise bombardment symptoms that you were told to expect?” 24 percent of subjects wrote they did. We also asked, “How much physical arousal (e.g., increased heart rate, breathing, etc.) did you experience during the noise bombardment?” The average response was 2.3 on a Likert-type scale in which 1 was “none” and 7 was “a lot”. We also asked subjects if their physical arousal remained high during the recovery period, and if so, what the arousal was due to. Of the 50 percent of subject who reported arousal, 9 percent wrote it was due to the noise bombardment.

Rumination. There was not a significant difference in how much subjects reported thinking, during the recovery period, about the stressor, F (4, 127) = 0.55, p > 0.69. The average response was 3.4, where “7” indicated that they were thinking about the stressor the whole time. There was a significant difference in how much subjects reported thinking about the manipulation, F (4, 127) = 4.25, p < 0.01. Subjects who exercised reported thinking more about the manipulation (M = 2.6) than

subjects who listened to white noise (*M* = 1.3), where “7” indicated that they were thinking about the manipulation the whole time.

Thought sampling. There was not a significant difference in the number of emotional thoughts subjects reported about the speech (F (2, 53) = .06, $p > 0.94$). However, there was a significant difference in the number of thoughts subjects reported about the manipulation (F (4, 127) = 2.86, $p < .05$). Over 36 percent of subjects in the exercise condition wrote about going to the gym or the need to exercise more often, compared to four percent of subjects in the control condition, suggesting that exercise was successful in altering the nature of the subjects’ thoughts. That is, since the thought sampling was a way of probing, in real time, what participants were thinking about during the recovery period, it follows that, at any given time, the participants who had exercised were more likely to be thinking about the exercise task, rather than the stressor. This does disagree, however, with our other measure of rumination, in which subjects reported no difference in how often they thought about the math task. This disagreement may be due to the point in time that the measures were taken; the thought sampling was a real time measure, the self-report was retrospective.

Discussion

While this study was able to replicate the results of Study 1, in that exercising after a stressor lead to better cardiovascular recovery, our hypothesis that exercise has this effect by providing an alternative attribution for the arousal produced in response to the stress task did not receive support. Recovery was not better in subjects given the misattribution manipulations, and in the case of the implicit misattribution manipulation, recovery was worse.

Manipulation checks indicate that some of our manipulations were more effective than others. Few subjects given the explicit misattribution (white noise) manipulation reported feeling the symptoms they were told to expect and ratings of arousal during the manipulation were low. Several subjects reported that the noise was “soothing”, further suggesting that our manipulation did not work. It is possible that our symptoms, taken from Ross, Rodin and Zimbardo’s (1969) study, corresponded more to the physiological correlates of fear, rather than to the anger and embarrassment likely induced in the present study. Perhaps a misattribution effect would have been more likely had the noise bombardment been more believable or made more salient to the subject.

Unlike the explicit misattribution manipulation, the implicit misattribution (video game) manipulation was effective in increasing physical arousal during the task. However, as indicated by both self-report and cardiovascular measures, it did not lead to as much arousal as the exercise task. Nor does it appear to have lead to misattribution of arousal. On the contrary, the protracted recovery in this group suggests it generated more rumination rather than less. While we tried to prevent evaluation apprehension by not keeping a record of scores, perhaps playing the video game created feelings of competitiveness. This competitiveness may have compounded feelings of inadequacy produced by the math task, leading to more rumination.

Self-reports indicate the distraction task was effective in distracting subjects from thoughts of the math task while completing the questionnaire. However, it did not lead to better recovery. Cardiovascular arousal during the task was also higher than expected. Perhaps, despite our assurances to the contrary, subjects felt some pressure to complete the task.

It appears, then, that while it is possible that exercise is simply the best task at causing misattribution of arousal, it is perhaps more likely that exercise is a unique activity, as we did not find other neutral tasks to have similar effects. However, if exercise does not work because of misattribution of arousal, there may still be some cognitive or affective mechanism at work. As with Study 1, subjects were more likely to think of exercise during the recovery period than any other manipulation. This indicates that exercise, in some respect, may alter the nature of the subjects' thoughts. In turn, this may lead to less rumination about the stress task, and improve recovery. Also, while subjects from all groups tended to attribute their arousal more to the veridical source of arousal than to the neutral source, consistent with other misattribution of arousal studies (Nisbett & Wilson, 1977; see Cotton, 1981, for commentary), more subjects in the exercise group felt their residual arousal was due to the exercise than any other manipulation.

Chapter 5

DISCUSSION

The purpose of these studies was twofold. Our first goal was to determine if exercise can reduce arousal not just during a stressor, as prior studies indicate, but both before it occurs, when one is anticipating the stressor, and after it occurs, when one is ruminating about it. Including both anticipatory and recovery responses may be more consistent with how individuals use exercise to cope with stress. It is also relevant to an expanded view of the cardiovascular reactivity hypothesis, which suggests that the duration of the stress response, in addition to the magnitude of the initial peak reaction, may contribute to cardiovascular illness.

While prior studies have had subjects exercise before a stressor, and have found exercise to attenuate cardiovascular reactivity to that stressor, we had subjects exercise after a stressor, and found exercise to improve cardiovascular recovery from that stressor. In Study 1, while the blood pressure of the participants in the exercise condition was far higher, during the exercise, than the blood pressure of the participants who did not exercise, soon after the tasks were completed the subjects who did exercise had lower systolic and diastolic blood pressure than subjects who had sat still. Likewise, we were able to replicate this effect in Study 4 and, with a different stress task, in Study 3. It is notable that, although exercising after a stressor adds to initial cardiovascular arousal, it goes on to improve cardiovascular recovery afterward.

We also found that exercising before a stressor can attenuate cardiovascular reactivity to that stressor, even if an individual knows of the stressor at the time of the exercise. While prior studies had subjects exercise before a stressor, at the time of the exercise, subjects did not know that a stressor would follow. Our results indicate

that exercise can reduce cardiovascular arousal while subjects are anticipating the stressor as well. However, knowing about the stressor at the time of the exercise does not produce any more, or less, cardiovascular reactivity to that stressor than not knowing about it. In both Study 2 and Study 3, there were no significant differences in reactivity to the stress task between subjects who had known about the stress task at the time of the exercise and subjects who did not know.

One unexpected and, we think, important finding from Study 2 and Study 3 has to do with the effect of the delay between the offset of the exercise and the onset of the stressor. That is, while prior studies indicate that exercising before a stressor can attenuate later cardiovascular reactivity to that stressor, our studies suggest this effect will occur only if there is an interval between the exercise and the stressor such that residual arousal from exercise is no longer present once the stressor begins. Moreover, if such a delay is not present, exercise may in fact produce more cardiovascular arousal to the stressor than not exercising. Taken together, Study 2 and Study 3 suggest an interaction of delay and having exercised; relative to those who do not exercise, no delay between exercise and stress seems to prime the stress response, as seen both in cardiovascular measures and self-report measures (Study 2), whereas a delay between exercise and stress attenuates the stress response (Study 3).

Our second goal was to test if the theory of misattribution of arousal can account for the stress attenuating effects of exercise. When more than one source of arousal is present, such as is the case when an individual exercises before a stressor or exercises after a stressor, it may be difficult for the individual to identify how much of the arousal is due to one source or another. Furthermore, when a strong neutral attribution for arousal is present (i.e., have just exercised), an individual may draw the

wrong conclusions for why they feel the way they do, and may therefore inhibit an emotion from occurring even in the presence of a stressful stimulus. Our hypothesis was that exercise works by reducing the emotional nature of the stressor, by providing an alternate attribution for the arousal produced in response to the stressor. By reducing the emotional nature of the stressor, individuals may ruminate about the stressor less, thereby reducing cardiovascular arousal. Results from Study 1 are consistent with this. Results from Study 2 are consistent with the related theory of excitation transfer, in which prior arousal from a neutral source (i.e., exercise) can be carried over to an emotional source of arousal and thus intensify the emotion.

However, results from Study 3 are inconsistent with our hypothesis. Excitation transfer theory predicts that a delay between exercise and stress will eliminate the emotional priming effects and the increase in arousal found in Study 2, as residual arousal from exercise will no longer be present when the stressor begins. However, the theory does not account for the delay reducing arousal, as it did in Study 3. The theory of misattribution of arousal may suggest why adding in a delay reverses the excitation transfer effect, but only for those subjects who did know about the stressor beforehand, by altering the anticipation of the stressor. That is, if subjects were able to misattribute their arousal from anticipating the speech task to the exercise task, then perhaps subjects going into the speech task felt less tense, the interpretation of the speech task was less emotional, and the magnitude of the stress response was reduced. However, the theory does not suggest why subjects who did not know of the stressor would have a similar response. For these subjects, the theory predicts that the “have just exercised” misattribution would be most salient without a delay. By adding in a delay, exercise provides some sort of protective effect that misattribution of arousal can’t explain and suggests there may be some other factor at work. Then

too, in Study 4, when we gave subjects other possible neutral sources of arousal after the stressor, only the exercise task was effective in improving cardiovascular recovery.

While it is possible that exercise is simply the best task at causing misattribution of arousal, in that it produces some optimal level of arousal, or subjects expect that it will, it is also likely that there is something special about exercise. Unfortunately, our results do not clearly suggest what it is. Post-exercise hypotension does not seem to be at work, at least in any obvious way. In Study 1, following exercise, it is possible that subjects were still ruminating about the stressor, but the resulting stress-associated elevations in blood pressure were masked by the hypotension that is known to follow exercise (Kenney & Seals, 1993). In other words, vasolidation from exercise may have kept blood pressure low, even when subjects were ruminating about the stressor. This explanation would be of clinical significance, such that if subjects did ruminate during this period, it was less taxing to their cardiovascular system than for subjects who did not exercise. However, in each of our studies, the exercise task by itself did not cause blood pressure to recover below baseline levels. Instead, blood pressure returned rapidly and precisely to pre-exercise levels. Moreover, in Study 1, the participants who exercised in the absence of the stressor and participants who exercised after the stressor showed identical recovery (despite the latter having significantly greater cardiovascular arousal at the start of the recovery period). If any vasodilation was simply masking the lingering effects of the psychological stressor, then we would expect a corresponding dip in blood pressure for subjects who did the exercise task but did not do the stressor task. Instead, it appears that the rapid recovery people show from exercise replaces the slow recovery they show from a stressor when subjects do both.

In both Study 1 and Study 4, subjects were more likely to think of exercise during the recovery period than any other manipulation. This indicates that exercise, in some respect, may alter the nature of the subjects' thoughts. In turn, this may lead to less rumination about the stress task, and improve recovery. Furthermore, in Study 4, while subjects from all groups tended to attribute their arousal more to the veridical source of arousal than to the neutral source, consistent with other misattribution of arousal studies (Nisbett & Wilson, 1977; see Cotton, 1981, for commentary), more subjects in the exercise group felt their residual arousal was due to the exercise than any other manipulation. This suggests that a cognitive or affective mechanism may still be at work, although our results from Study 4 indicate this is not due to simple distraction.

One factor that we did not explore was the impact of the subjects' expectations about what exercise would do. That is, some individuals may expect exercise to reduce stress, and this expectation may help them to recover from the stressor. Other individuals may dread exercise and expect it to be punitive rather than therapeutic (Dimsdale, Alpert, & Schneiderman, 1986). Or perhaps subjects have no expectation at all. As an individual's expectations about the efficacy of a treatment may, in some measure, influence the effectiveness of the treatment, future work may want to look at the impact of such expectations about exercise. Somewhat related to this, exercise can improve an individual's sense of self-efficacy, and thereby improve mood, including reducing anxiety (McAuley, Talbot, & Martinez, 1999). Individuals who feel more competent have also been found to have better immune system functioning (Bandura, Cioffi, Taylor, & Brouillard, 1988).

Limitations

Dependent Measures

The determination of a mechanism may depend on the dependent measures used. Perhaps blood pressure and heart rate are not sufficiently descriptive of the processes taking place. The first few studies in this area (Duda, Sedlock, Melby, & Thaman, 1988; McGowan, Robertson, & Epstein, 1985; Roth, 1989) used heart rate as the main index of cardiovascular responsiveness, but few found prior exercise to reduce heart rate reactivity to a stressor, perhaps due to the presence of residual arousal "carried over" from exercise (Rejeski, Thompson, Brubaker, & Miller, 1992). More recent work has focused on other measures of cardiovascular reactivity, including peripheral vascular resistance (Hamer, Jones, & Boutcher, 2006) and sympathetic activity (Brownley, Hinderliter, West, Girdler, Sherwood, & Light, 2003). That is, several of the mechanisms that are associated with PEH could also be responsible for mediating cardiovascular stress responses, including a decrease in sympathetic drive, as measured by a decrease in norepinephrine levels, and an increase in beta-adrenergic receptor responsiveness (Brownley et al., 2003). An opioid-mediated reduction of catecholamines is also possible (Boone, Flynn, Andres, Pizza, & Kubitz, 1992, as cited in Boone, Probst, Rogers, & Berger, 1992) as is a decrease in alpha-adrenergic receptor responsiveness (see discussion in West, Brownley, & Light, 1998).

Stressor

Choice of stressor. Few authors in this field give justification for their choice of stressor, although all require minimal movement and produce large, modifiable psychophysiological responses that are mediated by the sympathetic nervous system (Sherwood & Turner, 1992; Fillingim & Blumenthal, 1992). Stressors can be either

active, demanding a behavioral response with a clear objective for successful performance on the task, or passive, involving vigilance, but requiring no action. While active and passive stressors affect cardiovascular indices of arousal in different ways, exercise has been found to attenuate cardiovascular reactivity to both types of stressors in prior studies (Boone, Probst, Rogers, and Berger, 1993; Ebbesen, Prkachin, Mills, & Green, 1992; Probst, Bulbulian, & Knapp, 1997; Rejeski, Gregg, Thompson, & Berry, 1991; West, Brownley, & Light, 1998). In our studies, the stressors we used (mental arithmetic and public speaking) were both active stressors, but it is notable that exercise was able to improve cardiovascular recovery from more than one type of active stressor task. In the future, it may be worth examining this finding in the context of other stressors (i.e., not just those that invoke anger or embarrassment, but perhaps grief or fear). For example, can exercise work to reduce cardiovascular arousal when one is anticipating a fearful task, such as an electric shock (cf Ross, Rodin, & Zimbardo, 1969)?

Timing of stressor. Questions remain about the delay between the offset of the exercise and the onset of the stressor. In Study 2 we found exercise may be harmful if there is not a delay between the exercise and the stress, such that arousal from the exercise may prime the emotional state. However, by adding in a delay in Study 3, such that the arousal from the exercise has faded before the stressor begins, exercise is beneficial.

Similarly, questions remain about the delay between the offset of the stressor and the onset of the exercise; because in Study 1 and Study 4 the exercise began almost immediately after the stressor, it remains to be addressed how long after an event exercise can continue to be an effective form of stress management. This is an important question, since often a lag is unavoidable. After an argument with one's

spouse, one still needs time to put on a pair of sneakers before going for a walk, and one might need to wait hours to exercise after a crisis at work.

Exercise

Type of exercise. Most of the studies in this field use a bicycle ergometer. Although this device allows experimenters strict control over the workout, and facilitates comparison between studies, as with our exercise task, it does not lend itself well to generalizing to different forms of exercise. That is, although the affect literature would suggest that other forms of exercise have beneficial mood effects, even competitive contact sports like rugby (Steptoe, Kimbell, & Basford, 1998), it is not known whether these different modes of exercise would extend to psychophysiological indices of arousal as well.

If the stress attenuating effects of exercise are driven by physiology, it follows that any form of exercise that leads to some threshold of arousal will lead to an attenuation of stress responses. In contrast, if exercise works because of some sort of cognitive or affective mechanism, then some forms of exercise may be more effective than others. Unfortunately, defining a “pure” form of exercise in this field may be difficult. For example, in these studies, the choice of a nonsocial form of exercise removes the confounding factor of social support, as the presence of a supportive other can reduce cardiovascular reactivity to a psychosocial stressor (Gerin, Pieper, Levy, & Pickering, 1992). As at least some of the positive effects of chronic exercise on psychological well-being may come from social factors (Hughes, 1984), it is notable that a nonsocial form of exercise is associated with psychological well-being as well. However, even solitary forms of exercise may have a cognitive or affective component to them. For example, rock climbing is an individual sport that has some element of fear in it. Likewise, while some social forms of exercise may

foster camaraderie, others, like boxing, may encourage competition and aggression. Future work is needed, as the results of our studies, and prior studies, may not apply to all forms of exercise.

Intensity and duration of exercise. While one review in the affect literature has concluded that, with intensity kept constant, duration does not appear to have an effect on pre- to post-exercise affective changes (Ekkekakis & Petruzzello, 1999), a recent review and meta-analysis by Hamer, Taylor, and Steptoe (2006) found greater effects on cardiovascular reactivity for longer sessions of exercise. Also somewhat inconsistent with the affect literature, which suggests moderate intensity exercise is most effective in improving mood (Blanchard, Rodgers, Spence, & Courneya, 2001; Steptoe & Cox, 1988) there is an indication that high intensity exercise is most effective in reducing cardiovascular reactivity (Rejeski, Gregg, Thompson, & Berry, 1991; Roy & Steptoe, 1991; Steptoe, Kearsley, & Walters, 1993). It may be that the affect effects of brief exercise are not the same as the cardiovascular effects, and that there may be more than one reason why exercise works to reduce stress.

The exercise task used in our studies was very brief and very light relative to other studies, but these properties make it advantageous from a public health perspective. We chose to standardize the exercise task in terms of its form and duration, rather than its intensity. That is, one could adjust the exercise task for each individual so that it produces the same increases in heart rate or percentage of maximum VO_2. In our studies, instead of holding the response constant, we held the stimulus constant. This method is more easily replicated and, as with the shorter duration of exercise, perhaps more easily adoptable by the public.

In conclusion, while research to date has looked at the impact of exercising before a stressor, the present work demonstrates that both exercising while

anticipating a stressor and exercising after a stressor can also limit the duration of cardiovascular arousal. Furthermore, within the framework of the cardiovascular reactivity hypothesis, this work has shown that interventions that follow a stressor, even those whose acute effect is to raise blood pressure, can still be effective in limiting the duration of cardiovascular responses, even when another stressor is approaching, and thus have the potential to promote health.

APPENDIX 1

Experimental Material

A

Drug Legalization

Pro: All drugs (including cocaine and heroin) should be legal and readily available.

- reduces criminal activity
- drug rules don't work anyway
- existentialistic thought
- drug use is a victimless crime
- drugs improve consciousness
- people on drugs don't commit crimes
- the illegality of drugs actually makes people use more drugs
- legalization would save money now wasted on drug enforcement
- profits could go to homeless, not mafia
- alcohol and tobacco are legal, as are the methylxanthines
- drugs prevent violence by sedating people
- drugs don't cause hangovers
- incentive salience

Con: All drugs that are now illegal should remain illegal and the use or sale of any amount should be a felony.

- drugs reduce workplace efficiency
- Code of Hammurabi
- legalization will increase drug use
- drug establishments will be criminal hangouts
- "gateway" drugs
- drugs increase traffic accidents
- the matching law
- metabolic tolerance
- alcohol and tobacco already cause enough trouble, we don't need to make it worse
- drugs cause birth defects
- drugs lead to crime
- drugs can kill
- drugs are addictive

B

Noise Bombardment Side Effects

We are required to inform you of some of the typical physiological responses to noise bombardment. These symptoms are not dangerous, but may be uncomfortable.

- You may have some tremor, that is, you hands will start to shake.
- You may have some palpitation, that is, your heart will start to pound.
- Your rate of breathing may increase.
- You may get a sinking feeling in the pit of your stomach, like butterflies.

C

Two young men, brothers, had got into serious trouble. They were secretly leaving town in a hurry and needed money. Karl, the older one, broke into a store and stole a thousand dollars. Bob, the younger one, went to a retired old man who was known to help people in town. He told the man that he was very sick and that he needed a thousand dollars to pay for an operation. Bob asked the old man to lend him the money and promised that he would pay him back when he recovered. Really Bob wasn't sick at all, and he had no intention of paying the man back. Although the old man didn't know Bob very well, he lent him the money. So Bob and Karl skipped town, each with a thousand dollars.

1a. Which is worse, stealing like Karl or cheating like Bob?

1b. Why is that worse?

2. What do you think is the worst thing about cheating the old man?

2a. Why is that the worst thing?

3. In general, why should a promise be kept?

4. Is it important to keep a promise to someone you don't know well or will never see again?

4a. Why or why not?

5. Why shouldn't someone steal from a store?

6. What is the value or importance of property rights?

7. Should people do everything they can to obey the law?

7a. Why or why not?

8. Was the old man being irresponsible by lending Bob the money?

8a. Why or why not?

APPENDIX 2
Tables

Table 1.1. Schematic of Study 1 design.

Time: Condition:	Baseline 10 min	Stressor 3 min	Manipulation 3 min	Recovery 15 min
Stress / Exercise	√	Math	Exercise	√
No Stress / Exercise	√	X	Exercise	√
Stress / No Exercise				
Stress / Metronome Task	√	Math	Finger Tapping	√
Stress / No Task	√	Math	X	√

Table 1.2. Baseline and demographic characteristics of participants in Study 1.

	Stress / Exercise (n = 26)	No Stress / Exercise (n = 24)	Stress / Metronome Task (n = 26)	Stress / No Task (n = 26)
Baseline				
SBP	122.54 (12.44)	120.04 (21.49)	119.54 (15.31)	117.24 (12.44)
DBP	77.24 (15.40)	72.09 (15.04)	74.93 (9.38)	71.98 (8.80)
HR	77.58 (12.97)	79.49 (8.67)	78.45 (11.36)	73.53 (11.90)
Age	19.62 (1.44)	19.67 (1.17)	20.46 (2.58)	20.27 (2.46)
Gender				
Females	21	19	14	18
Males	5	5	12	8
Ethnicity				
Caucasian	11	7	11	10
Asian	11	15	9	10
Hispanic	3	1	3	3
African American				1
Other	1	1	3	2

Note. Standard deviations are listed in parentheses.

Table 1.3. Study 1 mean recovery period cardiovascular scores (change from baseline).

Condition	Systolic BP	Diastolic BP	Heart Rate
Stress / Exercise (n = 26)	3.5* (9.7)	0.3** (6.6)	2.1 (3.5)
No Stress / Exercise (n = 24)	3.6 (11.7)	1.1 (7.0)	1.4 (4.4)
Stress / No Exercise (n = 52)	8.8* (7.8)	4.8** (4.9)	0.1 (3.2)
Stress / Metronome Task	9.9 (8.3)	5.4 (5.6)	0.8 (2.8)
Stress / No Task	7.6 (7.2)	4.2 (4.2)	-0.6 (3.4)

Note. Standard deviations are listed in parentheses. * $p < .05$ ** $p < .01$

Table 2.1. Schematic of Study 2 design.

Period:	Baseline	Preparation I	Exercise	Preparation II	Stressor	Recovery
Time:	10 min	5 min	3 min	5 min	3 min	15 min
Condition:						
Exercise / Known Stress	√	Speech Prep	Exercise	Speech Prep	Speech	√
Exercise / Unknown Stress	√	X	Exercise	Speech Prep	Speech	√
Exercise / No Stress	√	X	Exercise	X	X	√
No Exercise / Known Stress	√	Speech Prep	X	Speech Prep	Speech	√

Table 2.2. Study 2 mean cardiovascular scores (change from baseline) for all periods.

SYSTOLIC BLOOD PRESSURE

Period: Condition:	Preparation I	Exercise	Preparation II	Stressor	Recovery
Exercise / Known Stress (n = 24)	12.1 (11.5)	37.3 (16.4)	24.0 (13.5)	39.2 (16.3)	14.5 (10.8)
Exercise / Unknown Stress (n = 23)	2.6 (5.4)	33.2 (15.6)	25.6 (13.7)	42.0 (21.5)	16.7 (12.6)
Exercise / No Stress (n = 17)	0.3 (7.8)	26.3 (16.5)			4.9 (12.1)
No Exercise / Known Stress (n = 22)	8.5 (6.3)	7.2 (5.5)	12.1 (10.5)	28.3 (12.4)	10.3 (8.0)

DIASTOLIC BLOOD PRESSURE

Period: Condition:	Preparation I	Exercise	Preparation II	Stressor	Recovery
Exercise / Known Stress	5.9 (5.5)	16.2 (8.9)	7.3 (7.0)	21.9 (10.2)	6.3 (6.3)
Exercise / Unknown Stress	0.3 (2.3)	14.1 (8.9)	7.9 (7.0)	22.5 (14.5)	8.5 (7.4)
Exercise / No Stress	0.4 (4.5)	17.6 (13.0)			1.6 (9.0)
No Exercise / Known Stress	3.9 (3.0)	2.3 (3.6)	5.4 (5.5)	17.9 (6.5)	5.3 (5.2)

HEART RATE

Period: Condition:	Preparation I	Exercise	Preparation II	Stressor	Recovery
Exercise / Known Stress	4.6 (5.1)	30.3 (9.6)	11.5 (6.6)	16.4 (8.3)	2.5 (4.5)
Exercise / Unknown Stress	1.0 (3.0)	32.0 (9.4)	15.3 (9.5)	21.6 (13.1)	3.4 (4.0)
Exercise / No Stress	0.2 (3.1)	31.1 (14.7)			6.1 (4.3)
No Exercise / Known Stress	4.5 (6.0)	-0.5 (3.8)	5.0 (5.5)	10.6 (10.2)	-1.4 (4.6)

Note. Standard deviations are listed in parentheses.

Table 3.1. Schematic of Study 3 design.

Period:	Baseline	Preparation I	Exercise	Delay	Preparation II	Stressor	Recovery
Time:	10 min	5 min	3 min	5 min	5 min	3 min	15 min
Condition:							
Exercise / Known Stress	√	Speech Prep	Exercise	√	Speech Prep	Speech	√
Exercise / Unknown Stress	√	X	Exercise	√	Speech Prep	Speech	√
Exercise / No Stress	√	X	Exercise	√	X	X	√
No Exercise / Known Stress	√	Speech Prep	X	√	Speech Prep	Speech	√

Table 3.2. Study 3 mean cardiovascular scores (change from baseline) for all periods.

SYSTOLIC BLOOD PRESSURE

Period: Condition:	Preparation I	Exercise	Delay	Preparation II	Stressor	Recovery
Exercise / Known Stress (n = 21)	7.4 (9.7)	27.7 (13.1)	10.2 (13.9)	10.7 (13.1)	38.8 (17.7)	14.7 (13.1)
Exercise / Unknown Stress (n = 22)	3.0 (9.6)	31.6 (22.2)	6.9 (15.8)	17.1 (14.1)	38.1 (15.4)	14.0 (15.4)
Exercise / No Stress (n = 25)	0.9 (7.2)	30.6 (20.2)	4.5 (15.0)			5.0 (11.0)
No Exercise / Known Stress (n = 15)	12.5 (8.1)	15.5 (11.8)	12.0 (12.4)	9.5 (14.4)	47.4 (15.1)	17.6 (12.9)

DIASTOLIC BLOOD PRESSURE

Period: Condition:	Preparation I	Exercise	Delay	Preparation II	Stressor	Recovery
Exercise / Known Stress	4.5 (4.4)	14.3 (7.6)	1.9 (7.1)	5.1 (9.9)	25.8 (13.8)	11.1 (12.6)
Exercise / Unknown Stress	-0.4 (5.7)	17.9 (15.4)	-1.2 (11.0)	7.9 (11.5)	26.5 (12.8)	10.8 (12.3)
Exercise / No Stress	-0.6 (5.5)	17.2 (10.3)	0.1 (7.7)			0.7 (6.3)
No Exercise / Known Stress	7.1 (4.6)	6.8 (4.3)	8.5 (7.7)	13.2 (10.1)	37.7 (16.6)	12.8 (10.5)

HEART RATE

Period: Condition:	Preparation I	Exercise	Delay	Preparation II	Stressor	Recovery
Exercise / Known Stress	2.1 (4.7)	30.6 (11.7)	6.5 (8.9)	6.9 (7.5)	15.0 (8.9)	1.1 (6.4)
Exercise / Unknown Stress	-0.7 (2.9)	31.2 (16.0)	2.8 (4.9)	7.4 (6.6)	13.7 (17.7)	1.6 (5.3)
Exercise / No Stress	-0.3 (2.8)	28.4 (9.5)	4.1 (5.1)			4.3 (4.3)
No Exercise / Known Stress	5.4 (3.0)	2.5 (5.3)	3.8 (4.3)	5.9 (5.1)	18.1 (11.1)	-0.4 (3.0)

Note. Standard deviations are listed in parentheses.

Table 4.1. Schematic of Study 4 design.

Period: Time: Condition:	Baseline 10 min	Stressor 3 min	Manipulation 3 min	Recovery 15 min
Control	√	Math	X	√
Distraction	√	Math	Questionnaire	√
Exercise	√	Math	Exercise	√
Implicit Misattribution	√	Math	Video Game	√
Explicit Misattribution	√	Math	White Noise	√

Table 4.2. Study 4 mean recovery period cardiovascular scores (change from baseline).

Condition	Systolic BP	Diastolic BP	Heart Rate
Control (n = 26)	11.2 (10.0)	5.3 (5.3)	-1.6 (4.3)
Distraction (n = 23)	12.8 (10.9)	7.3 (7.7)	-0.5 (5.2)
Exercise (n = 36)	5.9 (9.9)	2.0 (6.9)	3.6 (5.7)
Implicit Misattribution (n = 24)	14.5 (13.6)	10.7 (10.5)	0.5 (3.4)
Explicit Misattribution (n = 23)	11.3 (11.8)	5.9 (7.0)	-0.2 (3.5)

Note. Standard deviations are listed in parentheses.

APPENDIX 3
Figures

Figure 1.1 Average systolic blood pressure change from baseline score (per minute) across the experimental session of Study 1.

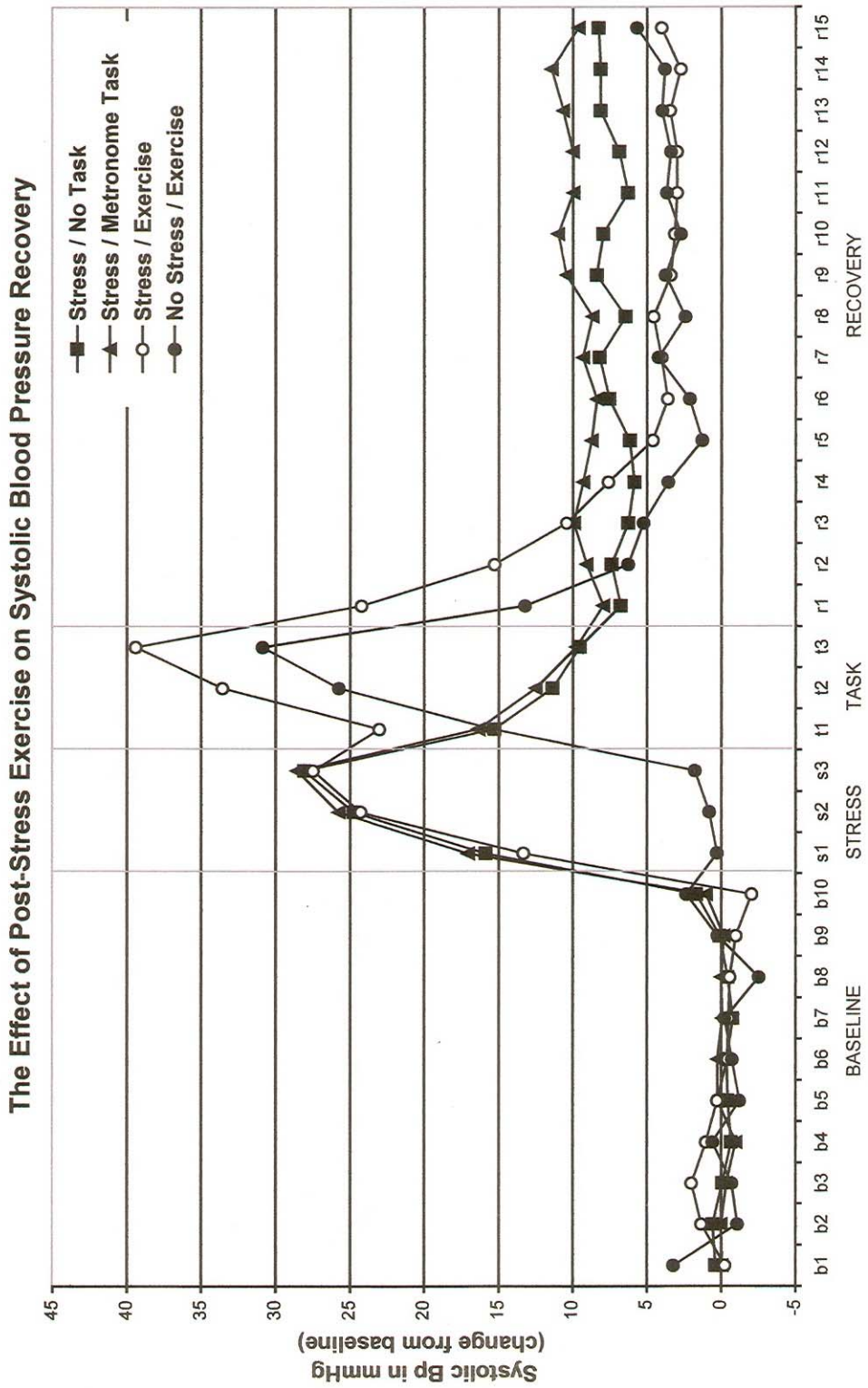

The Effect of Post-Stress Exercise on Systolic Blood Pressure Recovery
Stress / No Task
Stress / Metronome Task
Stress / Exercise
No Stress / Exercise
Systolic Bp in mmHg
(change from baseline)
45
40
35
30
25
20
15
10
5
0
-5
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 s1 s2 s3 t1 t2 t3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
BASELINE
STRESS
TASK
RECOVERY
Minute Averages
(continuous monitoring with Finapres)

Figure 1.2 Average diastolic blood pressure change from baseline score (per minute) across the experimental session of Study 1.

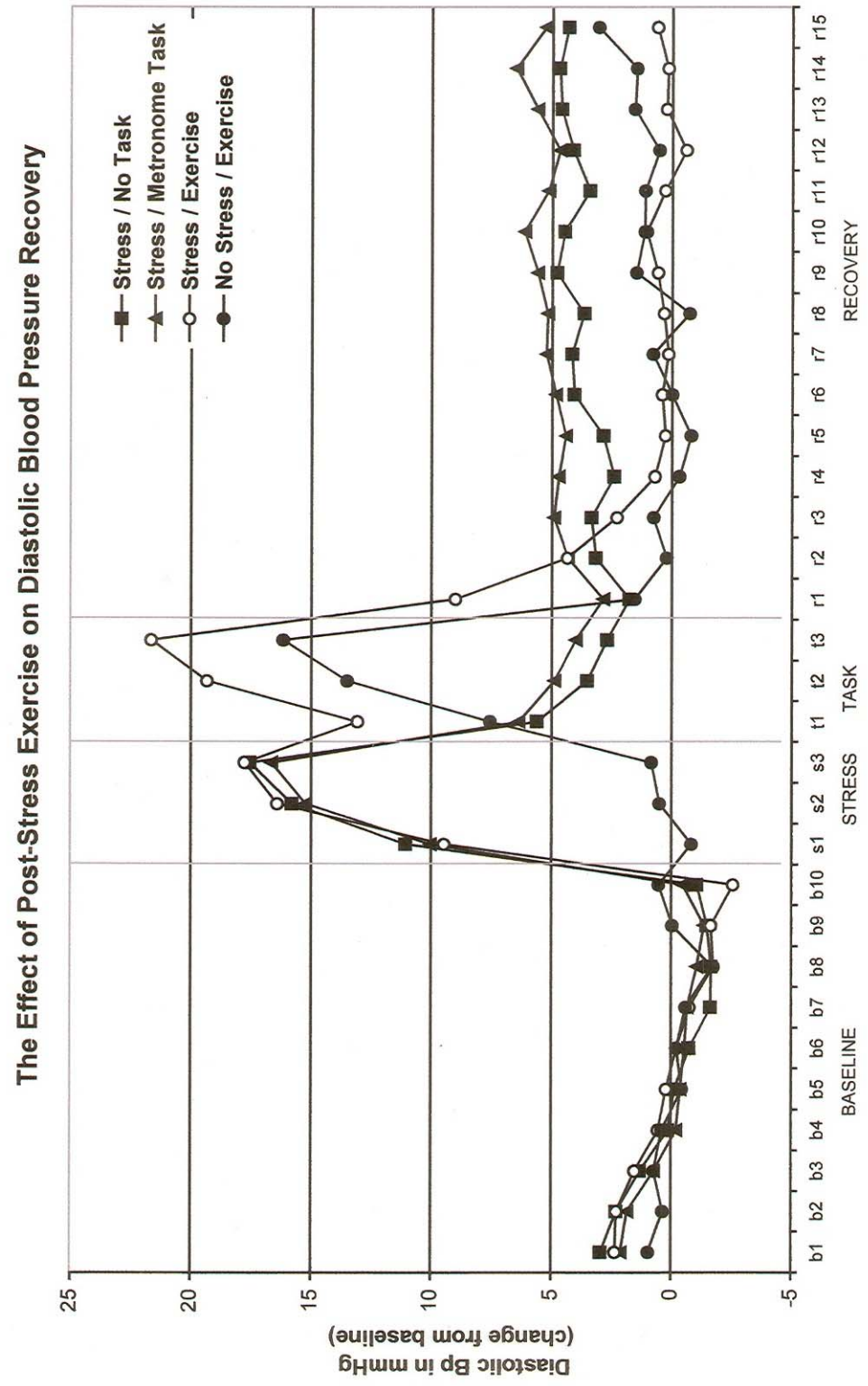

The Effect of Post-Stress Exercise on Diastolic Blood Pressure Recovery
Stress / No Task
Stress / Metronome Task
Stress / Exercise
No Stress / Exercise
Diastolic Bp in mmHg
(change from baseline)
25
20
15
10
5
0
-5
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 s1 s2 s3 t1 t2 t3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
BASELINE
STRESS TASK
RECOVERY
Minute Averages
(continuous monitoring with Finapres)

Figure 1.3 Average heart rate change from baseline score (per minute) across the experimental session of Study 1.

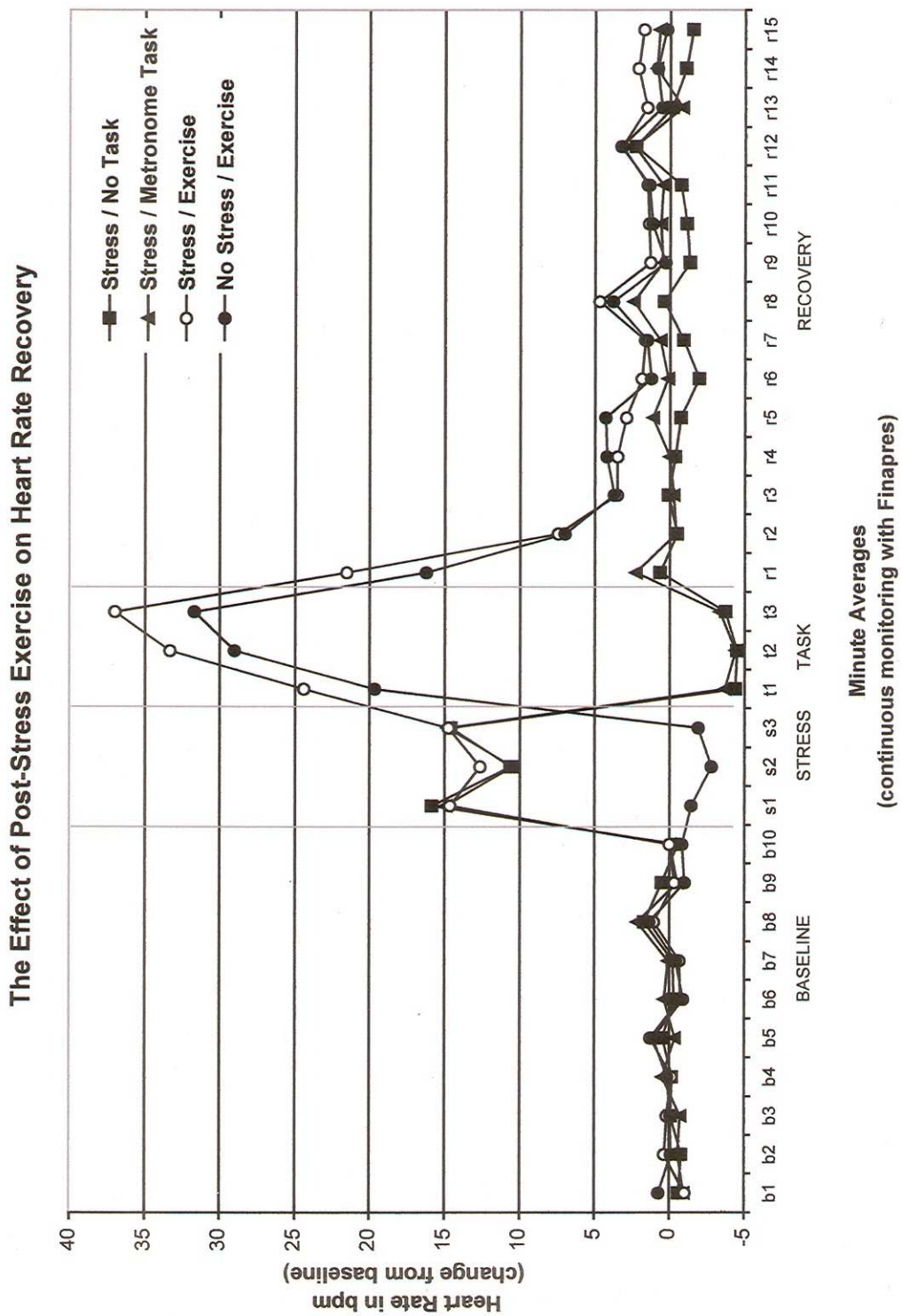

The Effect of Post-Stress Exercise on Heart Rate Recovery
Stress / No Task
Stress / Metronome Task
Stress / Exercise
No Stress / Exercise
Heart Rate in bpm
(change from baseline)
40
35
30
25
20
15
10
5
0
-5
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 s1 s2 s3 t1 t2 t3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
BASELINE
STRESS TASK
RECOVERY
Minute Averages
(continuous monitoring with Finapres)

Figure 2.1 Average systolic blood pressure change from baseline score (per minute) across the experimental session of Study 2.

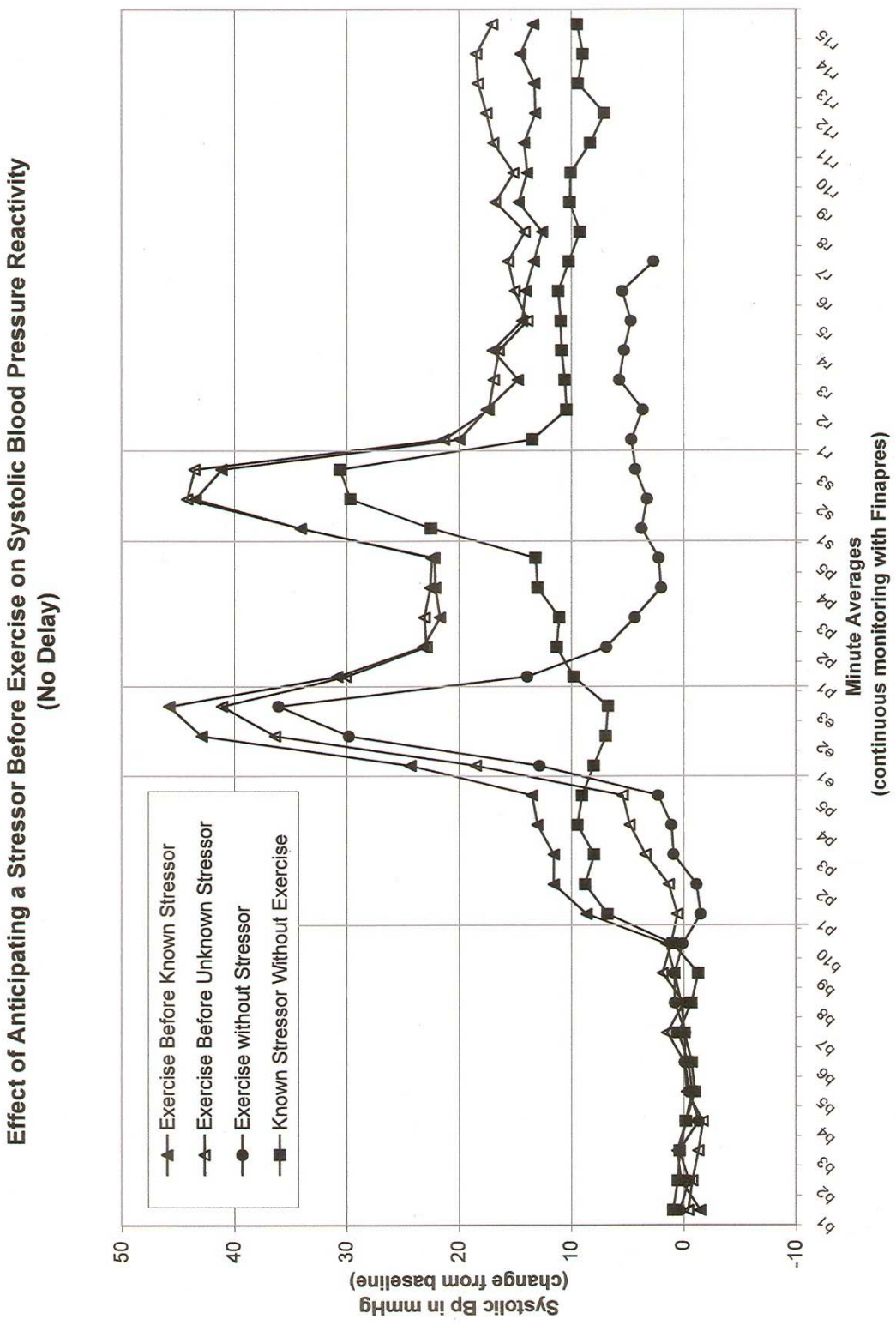

Effect of Anticipating a Stressor Before Exercise on Systolic Blood Pressure Reactivity
(No Delay)
Exercise Before Known Stressor
Exercise Before Unknown Stressor
Exercise without Stressor
Known Stressor Without Exercise
Systolic Bp in mmHg
(change from baseline)
50
40
30
20
10
0
-10
Minute Averages
(continuous monitoring with Finapres)

Figure 2.2 Average diastolic blood pressure change from baseline score (per minute) across the experimental session of Study 2.

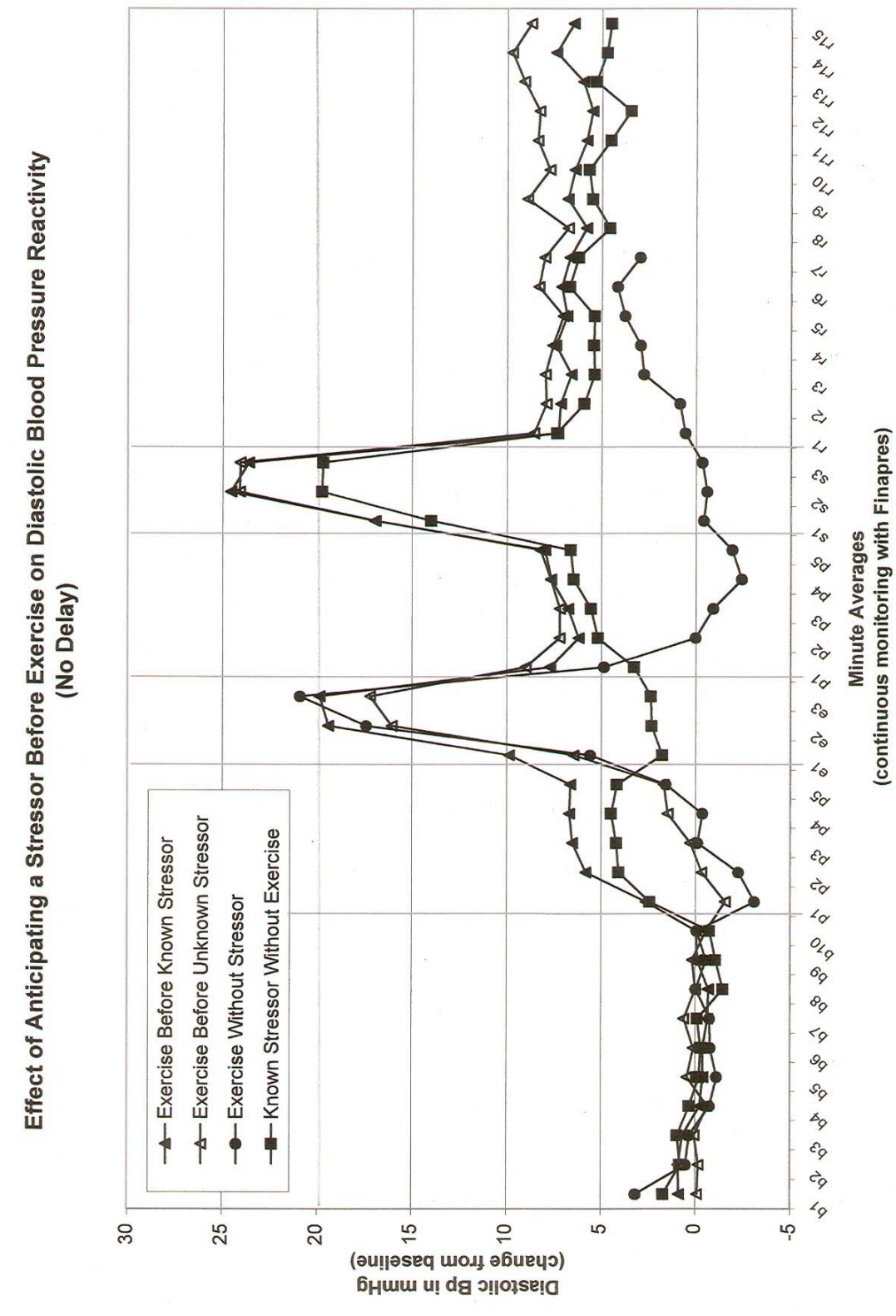

Effect of Anticipating a Stressor Before Exercise on Diastolic Blood Pressure Reactivity
(No Delay)
Exercise Before Known Stressor
Exercise Before Unknown Stressor
Exercise Without Stressor
Known Stressor Without Exercise
Diastolic Bp in mmHg
(change from baseline)
30
25
20
15
10
5
0
-5
Minute Averages
(continuous monitoring with Finapres)

Figure 2.3 Average heart rate change from baseline score (per minute) across the experimental session of Study 2.

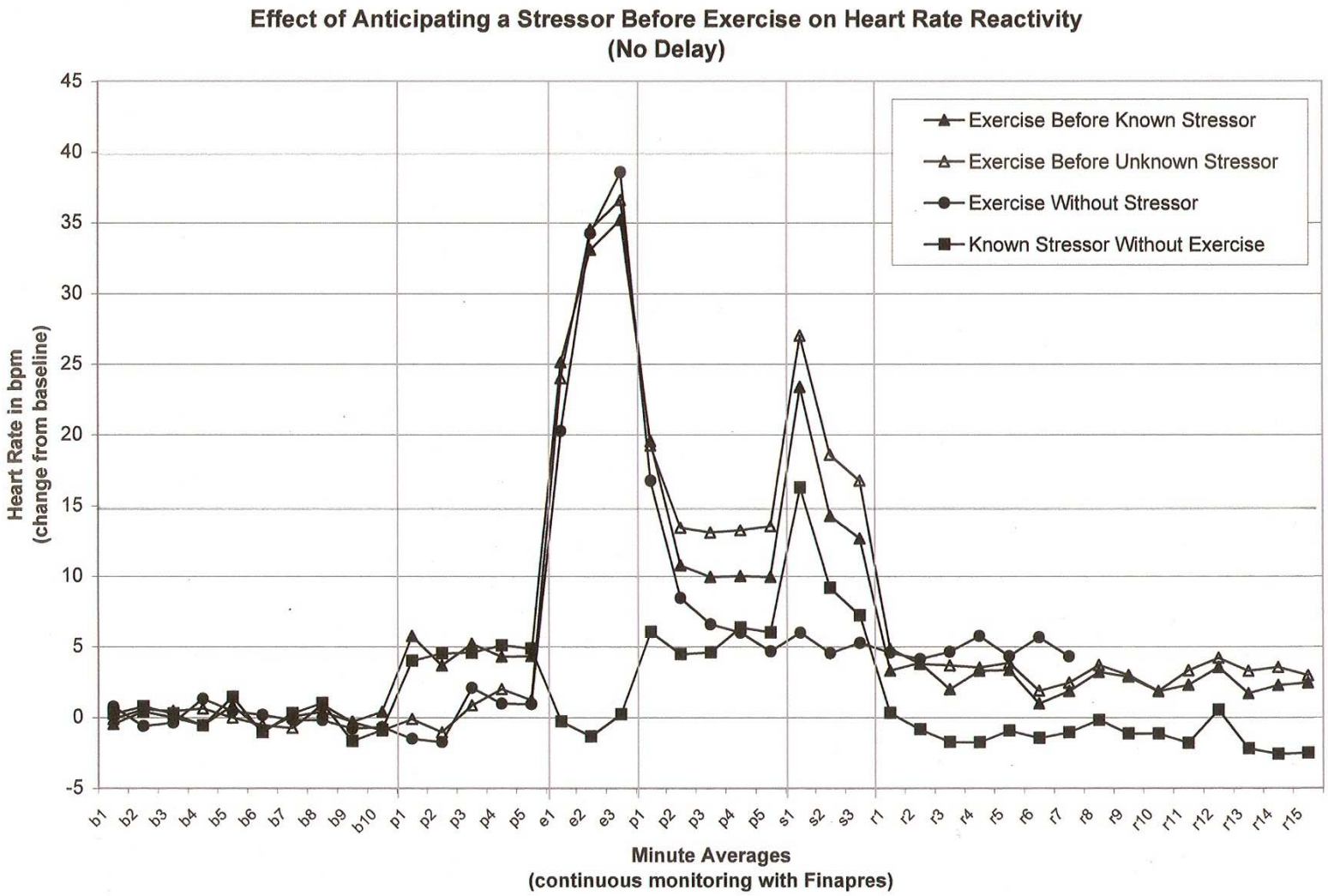

Effect of Anticipating a Stressor Before Exercise on Heart Rate Reactivity
(No Delay)
Heart Rate in bpm
(change from baseline)
45
40
35
30
25
20
15
10
5
0
-5
Exercise Before Known Stressor
Exercise Before Unknown Stressor
Exercise Without Stressor
Known Stressor Without Exercise
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 p1 p2 p3 p4 p5 e1 e2 e3 p1 p2 p3 p4 p5 s1 s2 s3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
Minute Averages
(continuous monitoring with Finapres)

Figure 3.1 Average systolic blood pressure change from baseline score (per minute) across the experimental session of Study 3.

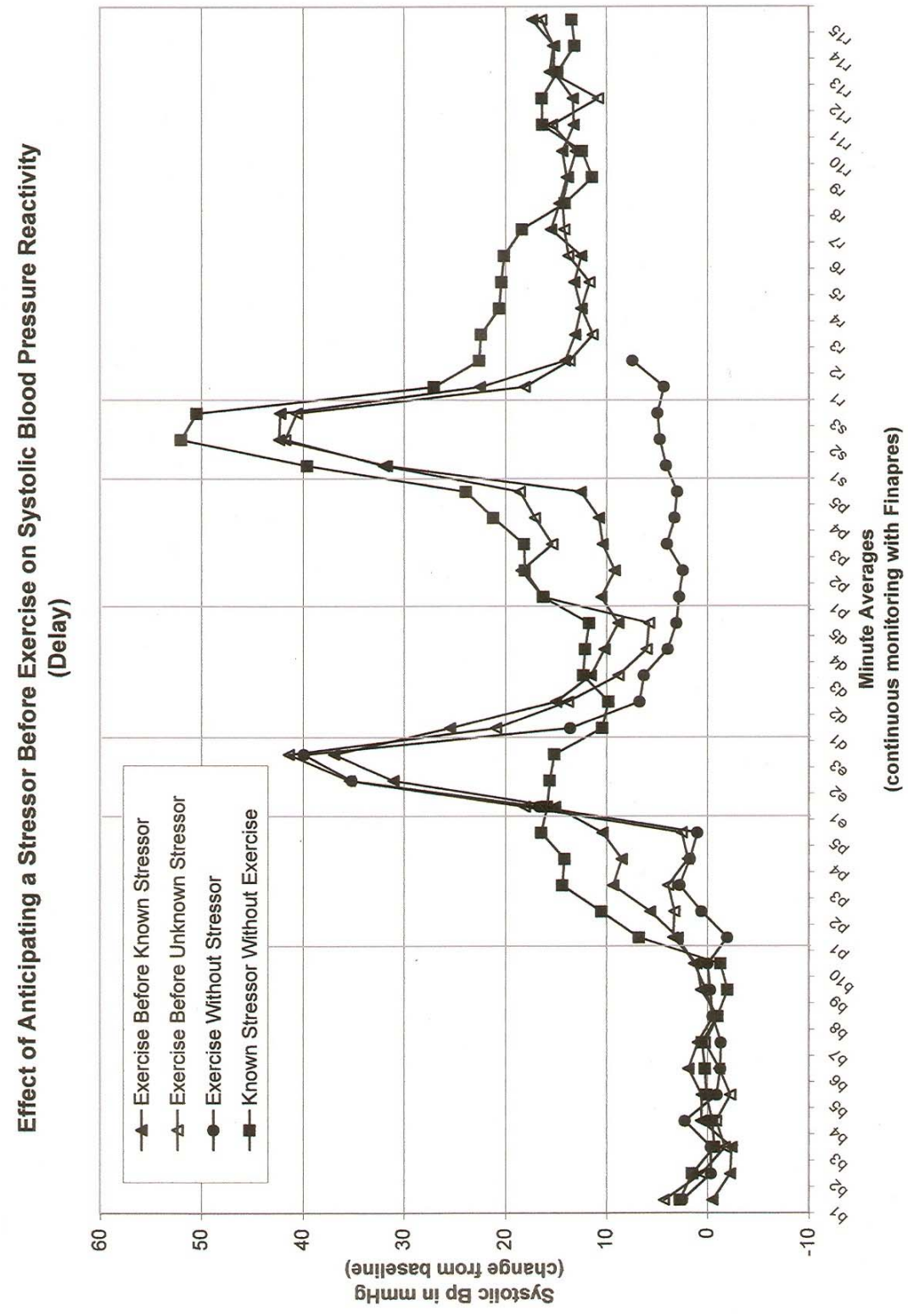

Effect of Anticipating a Stressor Before Exercise on Systolic Blood Pressure Reactivity
(Delay)
Exercise Before Known Stressor
Exercise Before Unknown Stressor
Exercise Without Stressor
Known Stressor Without Exercise
Systolic Bp in mmHg
(change from baseline)
60
50
40
30
20
10
0
-10
Minute Averages
(continuous monitoring with Finapres)

Figure 3.2 Average diastolic blood pressure change from baseline score (per minute) across the experimental session of Study 3.

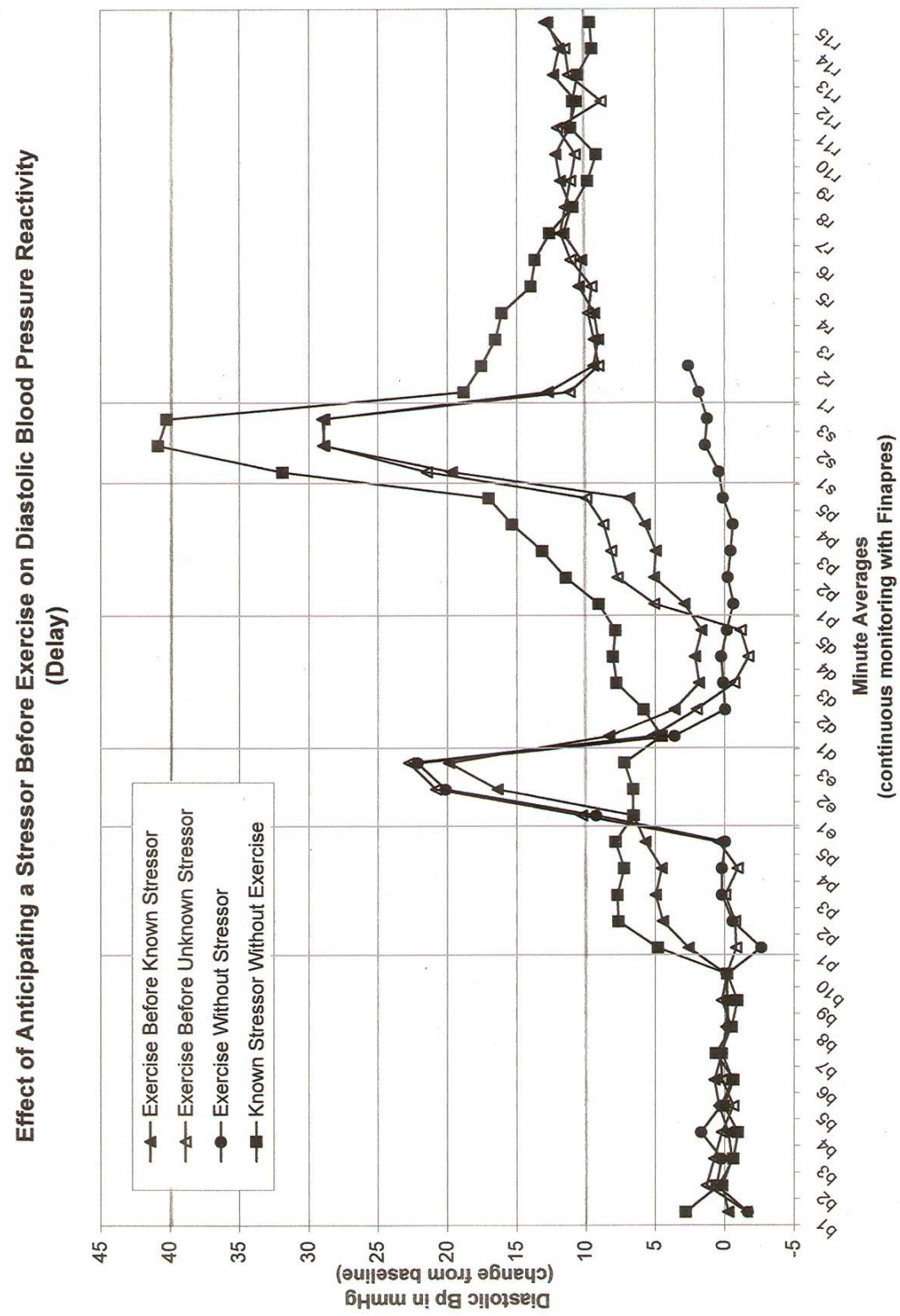

Effect of Anticipating a Stressor Before Exercise on Diastolic Blood Pressure Reactivity
(Delay)
Exercise Before Known Stressor
Exercise Before Unknown Stressor
Exercise Without Stressor
Known Stressor Without Exercise
Diastolic Bp in mmHg
(change from baseline)
45
40
35
30
25
20
15
10
5
0
-5
Minute Averages
(continuous monitoring with Finapres)

Figure 3.3 Average heart rate change from baseline score (per minute) across the experimental session of Study 3.

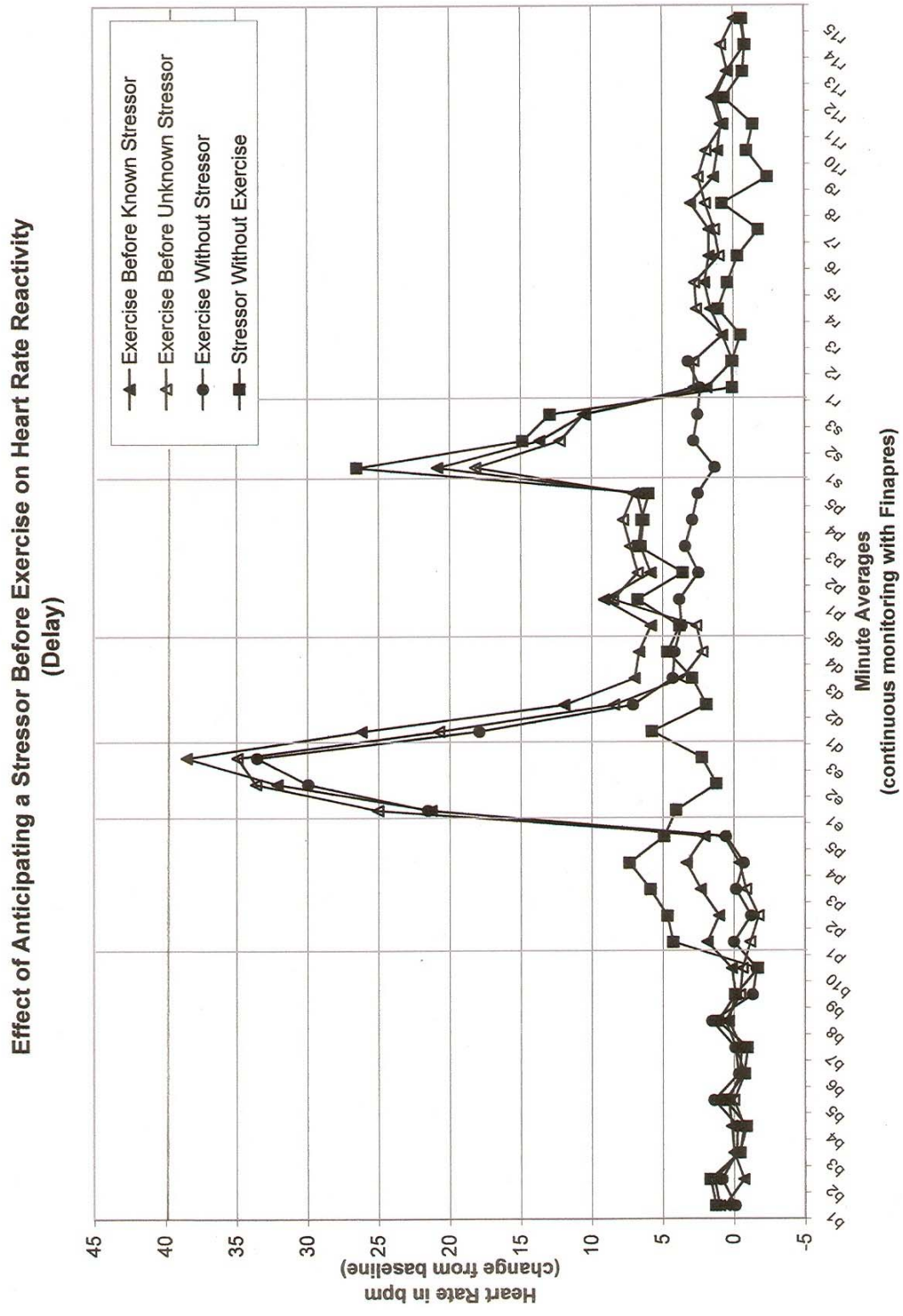

Effect of Anticipating a Stressor Before Exercise on Heart Rate Reactivity
(Delay)
Exercise Before Known Stressor
Exercise Before Unknown Stressor
Exercise Without Stressor
Stressor Without Exercise
Heart Rate in bpm
(change from baseline)
45
40
35
30
25
20
15
10
5
0
-5
Minute Averages
(continuous monitoring with Finapres)

Figure 4.1 Average systolic blood pressure change from baseline score (per minute) across the experimental session of Study 4.

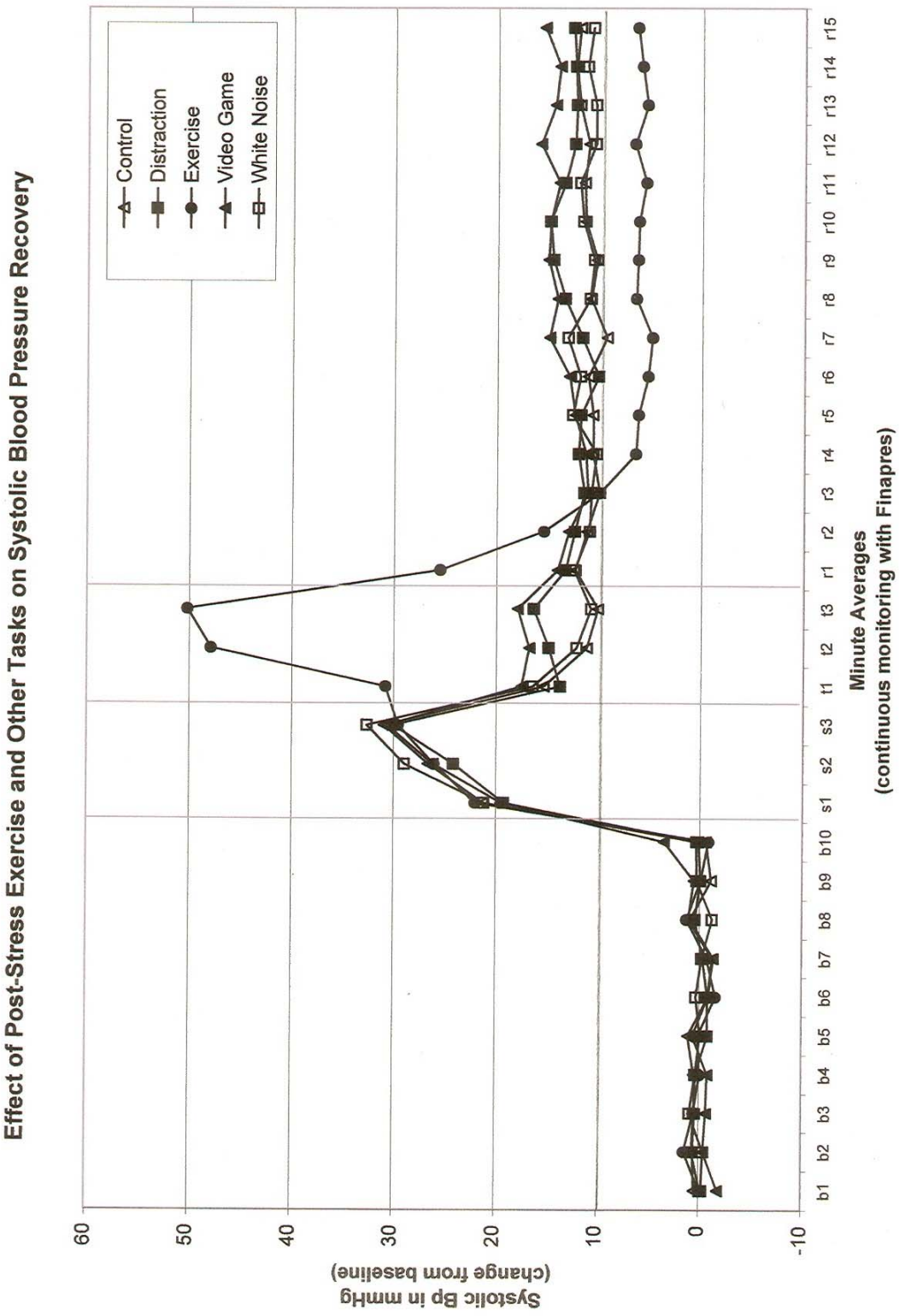

Effect of Post-Stress Exercise and Other Tasks on Systolic Blood Pressure Recovery
Control
Distraction
Exercise
Video Game
White Noise
Systolic Bp in mmHg
(change from baseline)
60
50
40
30
20
10
0
-10
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 s1 s2 s3 t1 t2 t3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
Minute Averages
(continuous monitoring with Finapres)

Figure 4.2 Average diastolic blood pressure change from baseline score (per minute) across the experimental session of Study 4.

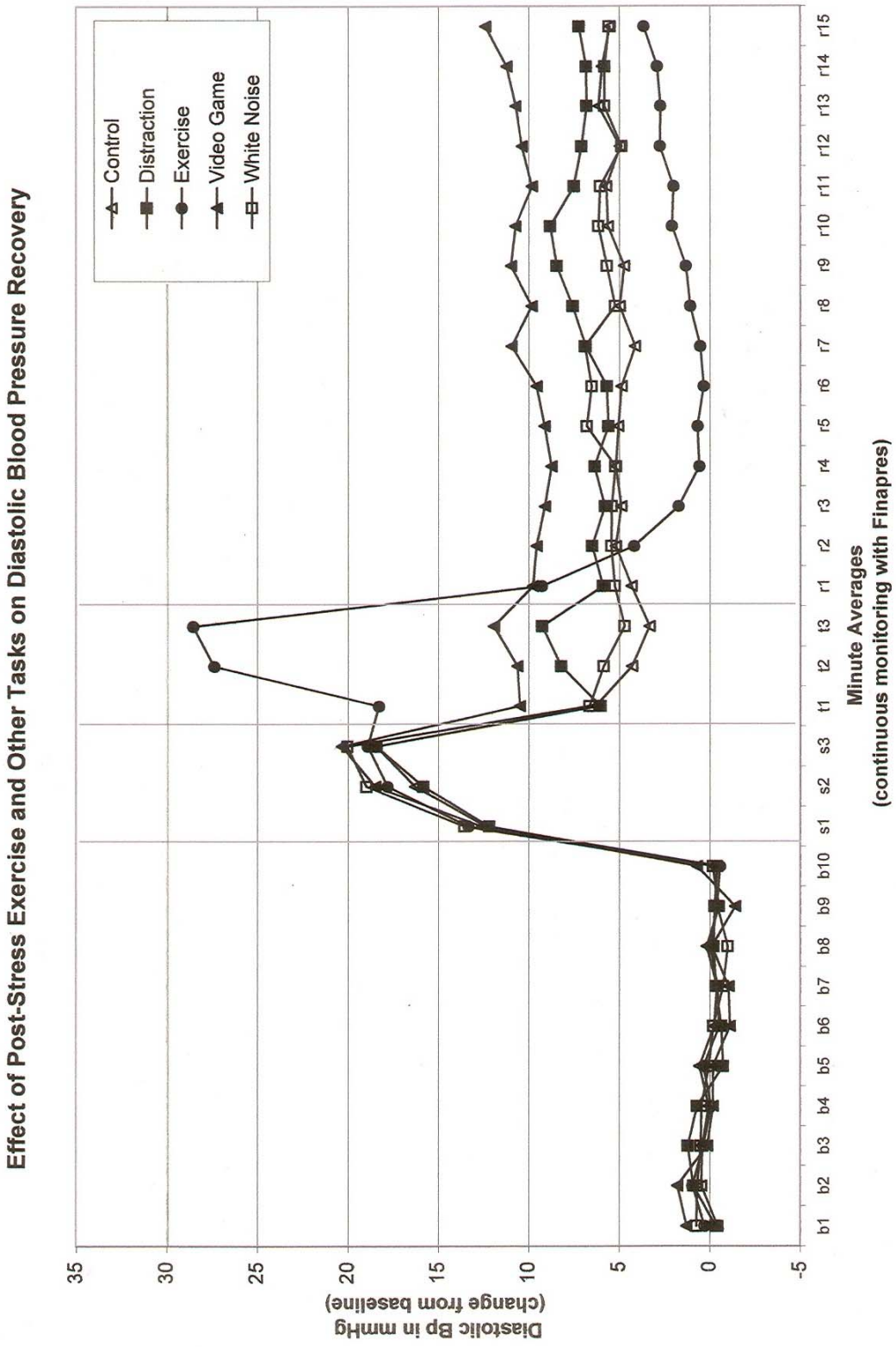
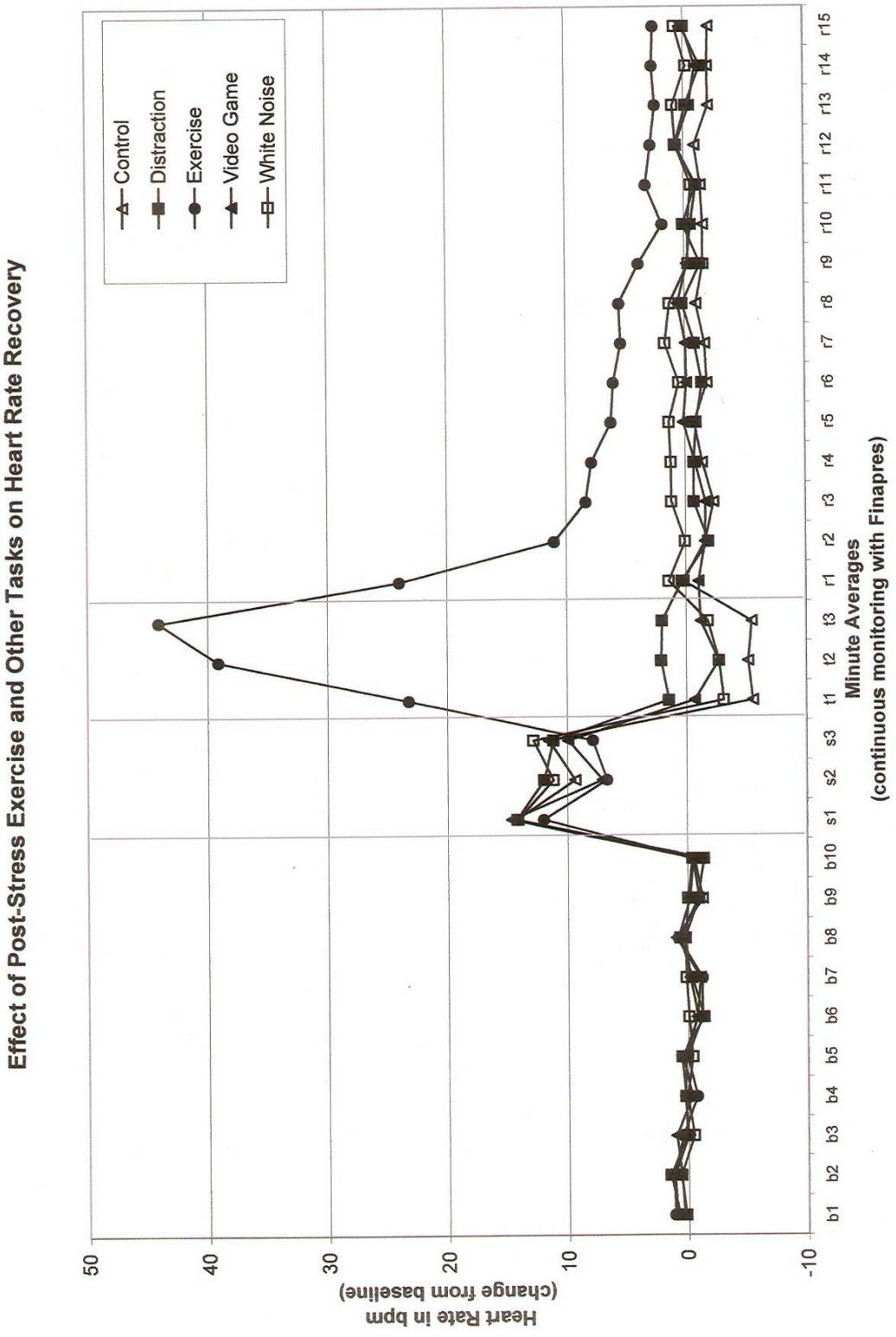

Effect of Post-Stress Exercise and Other Tasks on Diastolic Blood Pressure Recovery
Control
Distraction
Exercise
Video Game
White Noise
Diastolic Bp in mmHg
(change from baseline)
35
30
25
20
15
10
5
0
-5
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 s1 s2 s3 t1 t2 t3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
Minute Averages
(continuous monitoring with Finapres)

Figure 4.3 Average heart rate change from baseline score (per minute) across the experimental session of Study 4.


Effect of Post-Stress Exercise and Other Tasks on Heart Rate Recovery
Control
Distraction
Exercise
Video Game
White Noise
Heart Rate in bpm
(change from baseline)
50
40
30
20
10
0
-10
b1 b2 b3 b4 b5 b6 b7 b8 b9 b10 s1 s2 s3 t1 t2 t3 r1 r2 r3 r4 r5 r6 r7 r8 r9 r10 r11 r12 r13 r14 r15
Minute Averages
(continuous monitoring with Finapres)

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