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**LA THÈSE A ÉTÉ
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**Methodological Factors Influencing the Effect of Aerobic
Fitness on Reactivity to Psychosocial Stress**

Thomas G. Brown

**A Thesis
in
The Department
of
Psychology**

**Presented in Partial Fulfillment of the Requirements
for the Degree of Master of Arts at
Concordia University
Montréal, Québec, Canada**

September 1986

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ABSTRACT

Methodological Factors Influencing the
Effect of Aerobic Fitness on Reactivity
To Psychosocial Stress.

Thomas B. Brown

Two experiments examined the impact of aerobic fitness upon reactivity in a context where the influence of subject sex, stressor intensity, and the effort of verbalization were systematically explored. In Experiment 1, 17 males and 20 females were selected based on aerobic fitness, with 8 males and 9 females assigned to the high fit group, and 9 males and 9 females assigned to the low fit group. Subjects were exposed to a high and a low intensity laboratory psychosocial stressor. Heart rate was monitored during stressors and in recovery, while cognitive ratings were collected after both stressors. High fit males had lower heart rates in recovery than low fit males ($p < .05$), while stressors were differentiated by cognitive report ($p < .001$). In Experiment 2, 9 high fit and 12 low fit males were exposed to a high intensity stressor and a control, no-stress verbalization task. Significant differences were found in heart rate during the stressor between tasks, while differences in blood pressure appeared in recovery. Cognitive assessment differentiated the tasks ($p < .001$). These experiments support the hypothesis that aerobic fitness attenuates

reactivity during recovery from psychosocial stress.

Cardiovascular reactivity was found to result from the cognitive challenge of the psychosocial stressors. However, the cognitive appraisal of high or low intensity stressors was not reflected by corresponding levels of cardiac response. Future work on the psychological effect of fitness might explore the influence of other stressor-related factors, for example stressor modality, as well as further clarifying the impact of subject sex on reactivity.

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General Introduction

The behavioral community has become interested in aerobic exercise for its possible health and life-enhancing benefits (Martin & Dubbert, 1982). Aerobic exercise is characterized by rhythmic contractions of large muscle groups that increase heart rate to between 60 and 90% of an individual's maximal heart rate. With the proper intensity, duration and frequency of training, improvements in cardiovascular efficiency, such as reduced heart rate and lowered blood pressure at rest and during exercise, may occur in as little as six weeks (Nadel, 1985). Individuals exposed to psychological or behavioral challenge (ie., psychosocial stress) experience changes on a variety of physiologic parameters, including heart rate and blood pressure (Manuck & Krantz, 1984). The physiological adaptations with aerobic fitness have led to the exploration of whether improved fitness might also attenuate the cardiovascular responses to psychosocial stress. However, attempts to determine whether fitness dampens the responses to psychosocial stress have raised as many questions as have been answered.

To date, support for the reactivity-moderating properties of aerobic fitness falls into two distinct categories: studies where aerobic fitness attenuates cardiac reactivity during a stressor, and those where aerobic fitness has been found to hasten cardiac recovery following termination of a stressor. Support for the former finding

comes from two correlational studies and one employing a repeated measures design. Holmes and Roth (1984) found that trained subjects, as compared to untrained, had smaller heart rate responses during laboratory stressors, but no differences at 45 seconds into recovery. Hull, Young, and Zeigler (1984) reported reduced diastolic responses to stressors in older fit subjects, but no differences in recovery over a 10-minute period. Holmes and McBilly (1985) found that previously untrained subjects, when exposed to a 13-week aerobic exercise program, experienced reduced heart rate responses during stressors as compared to their pretest response. However, no recovery data was reported. Evidence in support of the second relationship, namely that fitness alters recovery, comes from three correlational studies. Cox, Evans and Jamieson (1979) found that heart rate recovered significantly faster from laboratory stressors five minutes following stressor termination in trained subjects than in untrained controls. Sinyor et al. (1983) reported significant differences between trained and untrained subjects at one minute into recovery, while Hollander and Seraganian (1984) found faster cardiac recovery with greater fitness in the 3 minutes following stressor termination. In these studies, no fitness effects were found during stressor presentation. Although both sets of findings support the hypothesis that aerobic fitness moderates reactivity to psychosocial stress, the precise nature in which this relationship is expressed is unclear.

The lack of consensus in the literature has raised a number of methodological issues. First, differences in the intensity of stressor across studies may influence when the effects of fitness upon reactivity appear. Cox et al., (1979), Hollander and Seraganian (1984), and Sinyor et al., (1983) all reported heart rate increases of approximately 25 beats per minute above baseline during stressor presentation. In contrast, Holmes & Roth (1984) reported increases of approximately 15 beats per minute. They suggest that the use of a more intense stressor could result in an 'arousal ceiling' in all subjects during exposure to the stressor. This ceiling could effectively mask differences in the magnitude of reactivity between treatment groups, but allow differences in cardiac reactivity to appear in recovery. In contrast, stressors yielding smaller increases in heart rate might result in reactivity differences from the influence of fitness appearing during stressors presentation only. Therefore, aerobic fitness may attenuate cardiac responses to stress, but the intensity of the stressors used in these studies could influence the precise temporal manifestation of this effect. However, no study to date has sought to test this hypothesis by systematically exploring the influence of stressor intensity upon reactivity.

A second methodological concern is whether sex differences influence reactivity. In related psychophysiological literature, sex differences have been seen to consistently influence the cardiovascular and

subjective responses to psychosocial stressors

(Frankenhauser, et al., 1978; McDougall & Dembroski, 1981; Lawler & Schmied, 1986).

However, in the fitness literature, the findings are unclear. The work of Cox, Evans and Jamieson (1979) revealed that sex did not influence heart rate response. Hull et al. (1984), though including both male and female subjects, did not systematically address sex effects. Nevertheless, it is suggestive that research yielding reduced reactivity during stressors were conducted using only female subjects (Roth & Holmes, 1984; Roth & McGilley, 1985), while much of the work reporting attenuation in recovery used males (Sinyor et al., 1983; Sinyor et al., 1986). Overall, the impact of sex differences upon reactivity does not seem to have been adequately addressed.

A third methodological issue concerns the degree to which the reactivity in response to stressors is due to physiological rather than psychological demands (Mathews et al. 1984). For example, speech patterns have been investigated as a source of increased cardiovascular activity (Friedmann, et al., 1982). The rapid and significant increase in blood pressure with normal verbalization has led these authors to speculate that aerobically fit people may experience relatively less cardiovascular reactivity as a result of the physical effort of talking than less fit people. This would parallel the more efficient cardiovascular responses of fit over unfit individuals to any

given intensity of physical activity (Nadel, 1983; Mathews and Fox, 1976). As much of the work has relied on stressor tasks requiring some physical activity along with cognitive challenge, a tautology of measurement may exist; that is, to what degree are fitness-related differences in reactivity simply a reflection of the cardiovascular efficiency in work that determines fitness in the first place? To date, clarification of the degree to which reactivity reflects both physiological and psychophysiological processes remains to be systematically addressed.

Several authors (Mathews et al. 1984; Manuck & Krantz, 1984) have suggested that progress in psychophysiological reactivity research may be achieved from greater attention to differences in methodology between studies, as well as improved experimental control. The present two-part study represents an attempt to clarify the influence of a number of methodological variables in the study of fitness. The first experiment explored the influence of fitness upon psychosocial reactivity in a context where the impact of stressor intensity and sex differences could be systematically addressed. The second experiment tried to parcel out the physical versus psychological demands of a laboratory psychosocial stressor.

Experiment 1

Introduction

The first experiment explored whether stressor intensity might influence the manner in which fitness level moderated cardiac reactivity. Employing a within-subject design, subjects determined to be either of high or low aerobic fitness were presented both a high and low intensity laboratory psychosocial stressor. Both consisted of arithmetic problems, with those in the high intensity stressor being more difficult, and therefore more cognitively challenging, than the problems in the low intensity stressor. Stressors were counterbalanced across subjects in their order of appearance. Heart rate responses prior to, during and in recovery from these stressors were obtained. As well, subjective ratings of task difficulty were collected in order to confirm that the stressors were perceived differently. By manipulating stressor intensity, clarification of its influence on the manner in which fitness might moderate reactivity was attempted. For example, if the rationale provided by Holmes and Roth (1984) was supported, exposure to a high intensity stressor might yield a ceiling effect in cardiac response, resulting in a fitness effect emerging in recovery. With a low-intensity stressor, however, the effect of training might emerge during the stressor itself rather than during recovery.

The second issue explored was whether a sex effect might be present in the impact of fitness on reactivity.

Consequently, both high fit and low fit groups consisted of similar numbers of male and female subjects. This arrangement permitted systematic, within-study comparison of the influence of fitness in males and females. If this effect was found to be critical, the interaction between fitness level and sex on reactivity might be clarified.

Method

Subjects

Males and females attending Concordia University, or their friends, were initially screened for age (between 18 and 40 years) and the absence of major health problems. Eligible subjects were further screened for aerobic fitness level, as expressed by three criteria: verbal reports of the level of participation in aerobic training; resting heart rate; and through $\dot{V}O_2$ max assessment from bicycle ergometry. Thirty-seven subjects (mean age=25.8 years), divided into four groups, were retained for participation: eight males and eleven females were assigned to the low-fit group (ie., $\dot{V}O_2$ max. < 40 ml O₂/kg/min, little regular participation in aerobic training, and resting heart rate >70 beats/min), and nine males and nine females were assigned to the high-fit group (ie., $\dot{V}O_2$ max >50 ml O₂/kg/min, regular participation in aerobic training, and a resting heart rate of <70 beats/min). These fitness criteria follow those established by Cooper (1981), whereby 50 ml O₂/kg/min or more is considered excellent or better for subjects aged 20 years or older, and 40 ml O₂ or less indicates a fitness

level of fair or worse. Subjects were told that they would undergo a series of mildly stressful psychological tasks for which they would receive a free physical fitness assessment.

Apparatus and Stimuli

A standard Hartz sphygmomanometer was used to measure blood pressure for the initial health status screening, while a Bodyguard bicycle ergometer (model 990), coupled with a Quinton 6-point ECG Monitor (model 622A-MS) and Medi-Trace silver chloride disposable electrodes to record heart rate, was employed for the initial fitness screening procedure. The experimental room consisted of a temperature and humidity-controlled, electronically shielded chamber (305 cm x 335 cm, Spectrashield). A 4-channel Beckman 511A Dynograph polygraph and 3 Medi-Trace silver chloride disposable electrodes were used to record heart rate during the experimental sessions, with the signal being processed through a Beckman (Type 9857) cardiometer coupler and fed directly into an IBM-PC computer (Model 5100) for beat-by-beat data-logging. Subjective appraisal of the level of task difficulty was obtained by a printed 10-point rating scale, which subjects employed to rate task difficulty from 1 (easy) to 10 (very difficult). A Sony stereo taperecorder (Model TC-630) and Sony external speakers were employed to provide instructions and psychosocial stimuli.

Procedure

Upon arrival to the laboratory, subjects were first required to sign a consent form which also included a brief

description of the study as well as a health status questionnaire. Blood pressure was then taken.

Psychosocial Stressor Task. The subject was seated in an armchair in a curtained-off portion of the enclosed chamber. Three electrodes were attached to the subject's chest for heart rate monitoring. Taped instructions were presented which instructed subjects that they were to undertake two tasks that would test their ability to answer swiftly and correctly mathematical questions. A 10-minute baseline period ensued with continuous heart rate monitoring employing the Beckman polygraph. Actual digital computer-logging of the heart rate occurred during the final 75 seconds of this period, although only the last 30 seconds were used for baseline determination. The first task was then presented. Briefly, the tasks consisted of a series of 10 arithmetic problems (see Appendix 1), which the subject was required to answer. For example, subjects were asked to count backwards from 100 by 3's, or were required to multiply a number by another, to which they would then add a third. A brief period after each problem, the solution was provided. The tasks were either of low (eg. $2 \times 3 + 4$) or high difficulty (eg. $17 \times 9 - 13$), with each subject exposed to both. Order of presentation was counterbalanced within all groups. Heart rate was recorded continuously during the task, as well as during the 30-second recovery period following the task. A 10-minute inter-trial waiting period ensued, with heart rate recorded during the final 75 seconds providing a

baseline for the second task. The second task was then presented, with the same format and data-collecting procedure as in the first trial, except that after the 30-second recovery period, the session ended with the subjective rating of task difficulty of both trials (see Appendix 2).

Aerobic Fitness Test. Following the psychosocial stress session and a 20-minute rest period, the subject was weighed, seated on the bicycle ergometer and was then attached to the ECG apparatus. Using the Astrand-Rhyming (1954) submaximal test for estimated maximal oxygen uptake ($\dot{V}O_2$ max), heart rates attained while pedalling at progressively higher workloads (until a criterion 130 beats/min was attained) were entered into a nomogram. The resulting estimated $\dot{V}O_2$ max expressed in liters/min, when divided by bodyweight, gives an estimated oxygen uptake expressed in relative units of ml/kg/min.

Results

Table 1 contains the mean heart rates for all groups for baseline (i.e., the average of each 30-second baseline period prior to each task), during the high and low intensity stressors, and in period for each task. Computer-assisted digitization of heart rate allowed these means to be generated in 10 second intervals from cardiac responses during the entire duration of monitoring.

As shown in Figure 1, changes in heart rate from baseline of about 12 beats per minute were observed for all subjects in response to both high and low intensity stressors.

TABLE 1

Mean heart rate (\pm standard error) at baseline, during, and in recovery for high and low intensity stressors.

	Males		Females	
	Fit	Unfit	Fit	Unfit
Baseline	51.3 \pm 4.6	75.1 \pm 13.1	61.0 \pm 7.6	82.1 \pm 9.5
	During Stressor			
High Intensity	64.4 \pm 10.5	87.7 \pm 10.5	75.4 \pm 4.9	91.6 \pm 11.9
Low Intensity	67.5 \pm 10.2	86.5 \pm 9.3	72.1 \pm 6.3	90.5 \pm 11.4
	During Recovery			
High Intensity	50.8 \pm 6.3	80.4 \pm 12.7	61.3 \pm 8.1	80.1 \pm 12.6
Low Intensity	52.4 \pm 7.1	77.0 \pm 10.2	63.2 \pm 7.4	80.3 \pm 10.1

The heart rate data was analyzed employing a repeated measures analysis of variance (Biomedical Data Programs, University of California, 1983). A one-way ANOVA on baseline heart rate yielded reliable differences by fitness [$F(1,33) = 55.0$, $p < 0.001$]. During stressors, a three-way ANOVA (ie., fitness x sex x intensity) rendered a significant main fitness effect [$F(1,33) = 42.6$, $p < 0.001$]. In recovery, significant fitness [$F(1,33) = 57.1$, $p < 0.001$] and sex [$F(1,33) = 4.5$, $p < 0.05$] main effects were observed. The differences in heart rate at baseline between high fit and low fit subjects replicated well-established differences between these groups (Mathews & Fox, 1976). In order to evaluate cardiac response while accounting for significant group differences at baseline, two approaches were employed: an analysis of variance on change scores (ie., baseline heart rate subtracted from stressor and recovery heart rate); and an analysis of covariance which statistically parcels out the variability in heart rate due to initial differences in baselines. This allowed a comparative analysis of the data.

Employing change scores, no reliable differences between high fit and low fit subjects or between males and females were found. Furthermore, differences between subjects' cardiac change scores on the high and low intensity stressors were not significant. In recovery, no differences in change scores were uncovered between high fit and low fit subjects, between males and females, or in recovery between high and low intensity stressors. However, a significant

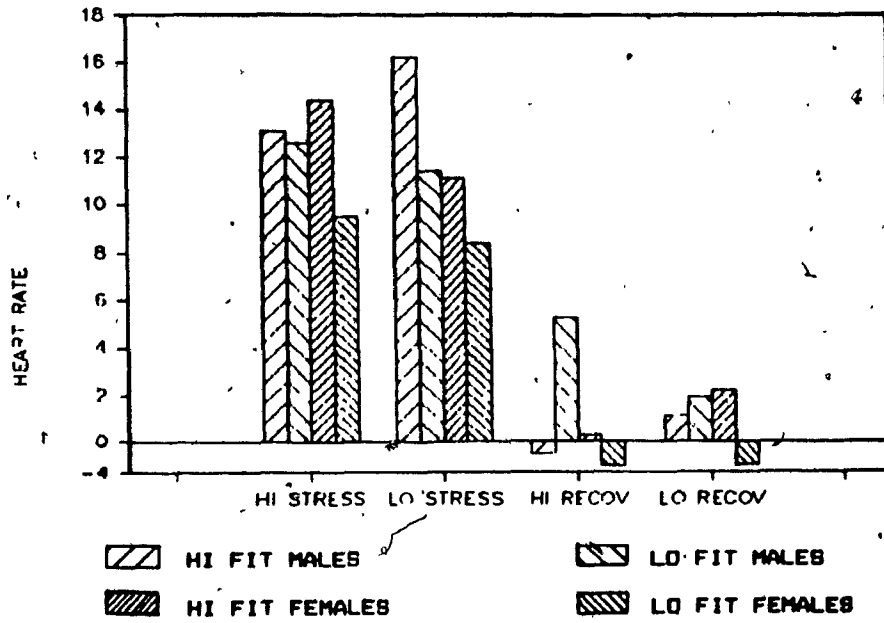


Figure 1. Mean heart rate change (BPM) from baseline during stressors and in recovery.

fitness x sex interaction emerged [$F(1,33) = 4.46, p < 0.05$]. According to a Scheffé post hoc analysis, employed due to its comparison flexibility and its capacity to accommodate uneven cell sizes, low fit females had a significantly higher recovery change score compared to the other groups combined ($p < 0.05$).

Assessment of the contribution of fitness and sex to heart rate during stressors and in recovery, with the influence of baseline statistically removed (i.e., ANACOVA with baselines as covariate), yielded a significant sex x fitness interaction [$F(1,32) = 5.0, p < 0.05$], with no other significant main effects or interactions. Employing a Scheffé post hoc analysis ($p < 0.05$), the source of the interaction was due to high fit males having significantly lower heart rate during recovery than low fit males. No differences in heart rate with stress intensity was found.

Cognitive ratings on the 10-point scale reliably differentiated the high intensity task from the low intensity task [$F(1,33) = 190.96, p < 0.001$], with a mean rating for the former of 7.4, and 2.5 for the latter. Other significant main effects or interactions were not significant.

Discussion

Physical fitness and sex both played a role in the cardiac responses to psychosocial stressors. The heart rate of fit males returned towards baseline more rapidly than unfit males. Cardiac response to stress in females did not reveal any dampening of response with fitness. During the

stressors, the groups were indistinguishable. Stressors of high or low intensity did not elicit differences in cardiac response.

The faster cardiac recovery from psychosocial stress in high fit males, as opposed to response attenuation during stress, replicates previous findings of this laboratory (Hollander & Seraganian, 1984; Sinyor et al., 1983). Highly fit subjects may respond equally to a given stressor as low fit subjects, but their greater aerobic capacity is correlated with more rapid recovery from stressors. In a model of reactivity, referred to as the "recurrent activation model", Manuck and Krantz (1984) have described the physiological changes due to psychological challenges as transient deviations from a stable baseline level of activation. While they propose that individual differences may be expressed by the magnitude of the acute physiological arousal to psychosocial stress, our results suggest that individual differences exist in the duration of the aroused state.

The subjective ratings of the stressors indicated that subjects perceived the stressors as being of significantly different difficulties. However, the failure to observe differences in cardiac reactivity as a function of perceived task difficulty suggests that cardiac reactivity may be influenced by characteristics other than the subjective intensity of the stressor.

This failure also made difficult a within-study examination of the impact of fitness in the context of

Cardiac response magnitude. However, changes in heart rate above baseline for both stressors are similar to those reported by Holmes and Roth (1984). Following their rationale, this degree of elicited reactivity should not have been affected by their hypothesized ceiling effect and should have yielded a significant fitness effect during stressor presentation. Furthermore, these heart rate increases are only about half the magnitude of those reported by Sinyor et al., (1983) that had also coincided with attenuation during recovery. The present observation of a fitness effect in recovery therefore seems to weaken the importance of the response magnitude as an explanatory construct with regards to whether reactivity attenuation will occur during stress or in recovery.

While high fit and low fit males were distinguishable in cardiac response in recovery from stressors in the analysis of covariance, low fit females were different from all other groups when change scores were used. This discrepancy between analyses suggests that other factors may have obscured the effects of fitness in the female subjects. It is suggestive that the recovery heart rate following both stressors in low fit females was lower than baseline levels. It is possible that the baseline of low fit females was artificially high. Thus, while the contribution of baseline heart rates on reactivity was averaged between groups when statistically partialled out in ANACOVA, change scores reduced significantly the absolute reactivity in unfit females as

compared to all other groups. Therefore, conclusions about sex-based differences in the effects of fitness at this point must be made with caution.

Differences in response attenuation with fitness between males and females have been observed in related work (MacDougall & Dembroski, 1981). It has been argued that certain situations may evoke different responses in females than in males. Specifically, situations with a high degree of interpersonal challenge elicit greater responses in females than males as compared to psychomotor tasks. The magnitude of responses in the present study was approximately the same for both males and females, even though the stressors employed did not have a high interpersonal component.

Experiment 2

Introduction

The second experiment focused on the degree to which cardiovascular responses to a psychosocial stressor in high fit and low fit subjects might be attributable to the physical demands of the stressor. Male subjects, categorized as either of high or low aerobic fitness, participated in two experimental tasks. One involved exposure to a high-intensity psychosocial stressor, the same task that was employed in Experiment 1. The second task, was essentially a control procedure. Rather than generating their own verbal responses to authentic questions, subjects simply repeated recorded numbers that mimicked those required by the high-

intensity stressor. This task required little cognitive effort and consequently, appreciably less psychosocial challenge than that of the high-intensity stressor. However, the physical effort required for both tasks was similar. The order of these tasks was counterbalanced across subjects. In addition to heart rate, blood pressure measures were collected. Subjective ratings of the tasks were gathered in order to corroborate their impact. In this manner, direct comparison of reactivity between the groups due to verbalization and psychophysiological reactivity could be undertaken. Furthermore, by statistically partialing out the physiological demands of the stressor, a more valid picture of the effect of fitness on psychophysiological reactivity might be achieved.

Method

The procedures employed were the same as those of Experiment 1, except for the following:

Subjects

Nine high fit and 12 low fit males (mean age = 24.6) were recruited using the same selection criteria described previously.

Apparatus

A Vita-Stat Automated Blood Pressure Monitor (Model 900-S), was added in order to increase the sensitivity of the measurement of cardiovascular reactivity.

Procedure

The low-intensity stressor was replaced by a task that required simple repetition of a series of 10 numbers or number sequences (eg., "repeat after me: 1, 4, 7.....") and blood pressure recordings were taken each minute during the full duration of the psychosocial stressor phase of the experiment, starting immediately after termination of the initial instructions.

Results

The heart rate and blood pressure means and standard errors at baseline and in response to the stressors are presented in Table 2. Average increases in heart rate with stressors over baseline, illustrated in Figure 2, were approximately 10 beats per minute. As in Experiment 1, a repeated measures ANOVA of heart rate and blood pressure was undertaken on change from baseline, as well as a repeated measures ANACOVA, with the differences in baselines between groups statistically removed.

The two-way analysis of change scores during presentation of the stressors uncovered a significant within main effect between the stressors employed [$F(1,20) = 48.7, p < 0.000$], but no significant fitness main effect or fitness x stressor intensity interaction. In recovery, no significant main effects or interactions were revealed.

The two-way ANACOVA performed on heart rate during stressor presentation produced a significant within factor stressor intensity main effect [$F(1,20) = 48.7, p < 0.000$], but

TABLE 2

Mean heart rate and blood pressure (\pm standard error) at baseline, during, and in recovery for high and low intensity stressors.

		Fit	Unfit
Heart Rate			
Baseline		61.2 \pm 10.5	78.4 \pm 10.9
During High Intensity Stressor		77.3 \pm 12.2	89.8 \pm 14.6
During Control Stressor		68.8 \pm 12.5	84.0 \pm 14.4
Recovery High Intensity Stressor		62.2 \pm 11.7	80.4 \pm 13.3
Recovery Control Stressor		64.1 \pm 12.0	77.9 \pm 12.2
Blood Pressure			
Baseline	Systolic	121.2 \pm 6.5	119.9 \pm 10.9
	Diastolic	65.9 \pm 8.1	72.8 \pm 11.0
During High Intensity Stressor	Systolic	126.7 \pm 17.9	129.3 \pm 13.1
	Diastolic	71.6 \pm 16.7	76.2 \pm 14.9
During Control Stressor	Systolic	126.9 \pm 11.7	121.7 \pm 14.6
	Diastolic	71.9 \pm 10.3	75.0 \pm 14.6
Recovery High Intensity Stressor	Systolic	127.6 \pm 11.6	125.5 \pm 12.4
	Diastolic	68.8 \pm 9.9	74.2 \pm 11.1
Recovery Control Stressor	Systolic	120.1 \pm 12.1	118.5 \pm 14.1
	Diastolic	66.7 \pm 13.4	75.2 \pm 13.1

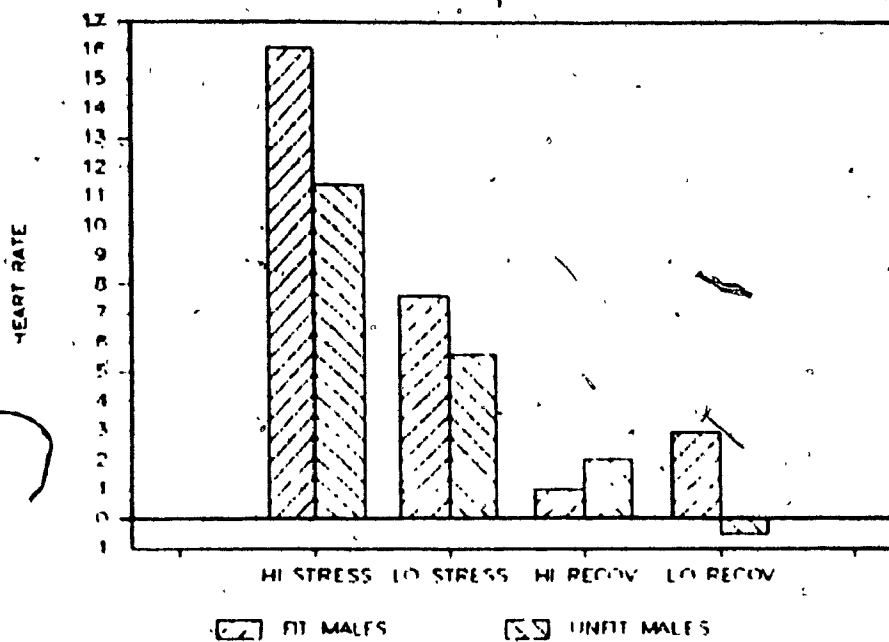


FIGURE 2. Mean heart rate change (BPM) from baseline during stressors and in recovery.

as with change scores, no significant fitness main effect or fitness x stressor intensity interaction. In recovery, no significant main effects or interaction were found.

Blood pressure at baseline, during the stressors and in recovery are presented in Table 2, while changes from baseline are illustrated in Figures 3 and 4. Analysis of blood pressure change scores during stressor presentation revealed no significant main effects or interaction for either systolic or diastolic measures. In recovery from stressors, a significant within main effect of stressor intensity was revealed for systolic blood pressure [$F(1,20) = 13.7, p < 0.005$]. No other significant fitness main effects or fitness x stressor intensity interaction were uncovered for systolic blood pressure. For diastolic blood pressure, no significant main effects or interaction were found.

Employing ANACOVA on systolic and diastolic blood pressure during stressor presentation, as in the analysis of change scores, no significant main effects or interactions emerged. In recovery, significant systolic difference appeared for the within measure of stressor intensity [$F(1,20), p < 0.005$]. However, no other significant effects were uncovered.

Subjective ratings, with a mean of 1.1 and 6.2 for low and high intensity stressors respectively, produced a significant two-way ANOVA on the within measure of stressor intensity [$F(1,20) = 102.7, p < 0.000$], with no other fitness main effect or interaction being revealed.

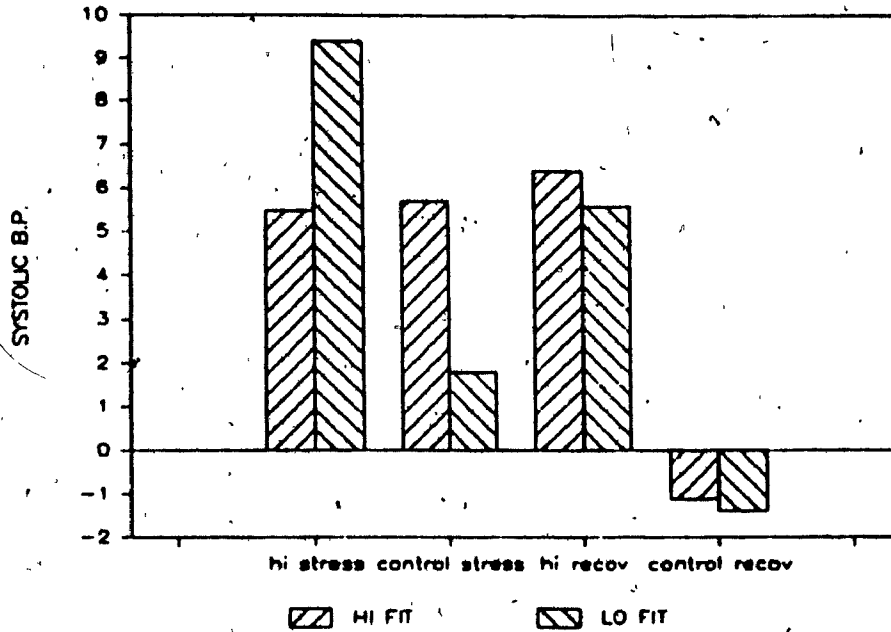


Figure 3. Mean systolic blood pressure change (mm Hg) from baseline during stressors and in recovery.

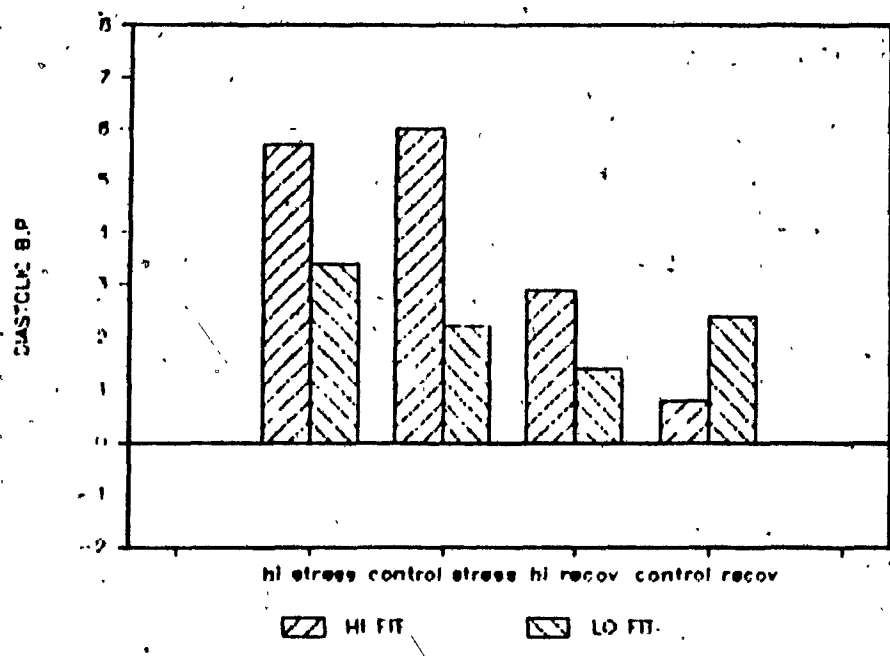


Figure 4. Mean diastolic blood pressure change (mm Hg) from baseline during stressors and in recovery.

Discussion

Differences in reactivity in Experiment 2 emerged between the high intensity stressor and the control verbalization task during and after stressors. As well, there was a lack of any significant change in cardiovascular activity from baseline during the control task in all groups. Coupled with the reliable subjective differentiation between the tasks, increases in cardiovascular reactivity appear to be a function of the cognitive challenge of the task and not simply the physical effort of vocalization. However, the lack of a significant fitness effect rendered inappropriate further scrutiny of the residual reactivity, with vocalization partialled out. The finding that heart rate provided reliable differences between the task intensities during stress, while blood pressure revealed differences in recovery, suggests that a time lag may mark the sensitivity of blood pressure measurement to psychosocial stress.

General Discussion

The present attempt to address the methodological variables that might have contributed to the inconsistencies in past work clarified some issues, while at the same time raising further questions.

The present work supported past findings in which attenuation of reactivity was found during recovery from psychosocial stress with greater aerobic fitness. This observation occurred in Experiment 1, but was not replicated in Experiment 2. Differences in the experimental protocol between the two phases of the present work may have resulted in this lack of consistency. In Experiment 1, two stressors were employed that, in spite of their different cognitive impact, resulted in fairly consistent levels of cardiac reactivity. Experiment 2, on the other hand, employed two quite different tasks: a stressor that elicited significant levels of cardiovascular reactivity and similar cognitive ratings as in Experiment 1; and a control, no-stress verbalization task that resulted in insignificant reactivity and little cognitive impact. It is possible that the influence of aerobic fitness emerged on the strength of the two effectively equivalent repeated measures in Experiment 1, while the fitness effect was not robust enough in Experiment 2 to occur with only one actual stressor.

Stressor intensity appears not to have a significant impact on the magnitude of cardiovascular reactivity. Although stressor intensity was reflected in the cognitive

appraisals of the stressors in Experiment 1, it did not result in differences in cardiac reactivity. In Experiment 2, the high intensity stressor again elicited significant and comparable levels of cardiac reactivity and cognitive ratings as in Experiment 1. However, significant cardiovascular change above baseline was not observed from a control task with little cognitive impact (i.e., with a mean cognitive rating of 1.1 compared to 2.5 of the low intensity stressor of Experiment 1). These observations suggest two important characteristics of these psychosocial stressors employed in the laboratory. First, it is the stressor possessing some degree of cognitive challenge, and not simply the experimental situation, that provokes significant levels of cardiovascular reactivity; and second, there seems to be no simple relationship between cognitive appraisals of stressor intensity and the coinciding magnitude of cardiac reactivity.

The present work also did not provide support for the importance of stressor intensity or magnitude of reactivity in determining whether the influence of fitness might occur during stress or in recovery. Therefore, different observations of the impact of fitness on cardiovascular reactivity between laboratories may reflect qualitative differences of the stressors employed. As well, these or other qualitative factors may determine the magnitude of cardiovascular reactivity. For example, response stereotypy, or the tendency for individuals to respond in a unique manner specific to the provoking stimulus (Steptoe, 1984) may

overshadow stressor intensity in determining the resulting magnitude of reactivity as well as when fitness might influence reactivity. Future inquiry might attempt to systematically study the importance of stressor modality in psychophysiological research.

The influence of subject sex, based on the present work, remains unclear. It is suggested that future work continue to recruit subjects counterbalanced for sex. When possible, counterbalancing for experimenter sex and for stressor modality might result in a clearer picture of the influence of sex differences on reactivity.

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Appendix 1

General Instructions

We are asking you to perform a pair of one minute tasks that test your ability to respond accurately and quickly to mathematical questions. Each task consists of a set of questions you must answer verbally. Do as well as you can, but if you make errors, just continue on. There will be a several minute rest interval after these preliminary instructions as well as a several minute rest period between the two tasks.

High Intensity Stressor

1. Subtract 7 continuously from 100.....10 sec....stop!
2. $9 \times 7 + 15$5 sec.....78!
3. $8 \times 6 - 9$5 sec.....39!
4. Subtract 13 continuously from 425.....10 sec....stop!
5. $6 \times 9 - 12$5 sec.....42!
6. $27 / 3 + 8$5 sec.....17!
7. $27 + 62 - 12$5 sec.....17!
8. Subtract 6 continuously from 200.....10 sec....stop!

Low Intensity Stressor

1. Add 5 continuously to 25.....10 sec....stop!
2. $3 \times 2 + 4$5 sec.....10!
3. $4 \times 3 - 2$5 sec.....10!
4. Subtract 2 continuously from 100.....10 sec....stop!
5. $5 \times 5 + 5$5 sec.....30!

6. $20 / 2 + 10$5 sec.....20!

7. $20 + 12 + 3$5 sec.....35!

8. Add 4 continuously to 4.....10 sec.....stop!

Appendix 2

Cognitive Rating

We would like to get some indication of how difficult you found the two tasks on a scale from 1 to 10, where 1 is not challenging and 10 is very challenging. Please circle one of the numbers for each task.

Task 1

1 2 3 4 5 6 7 8 9 10
very very
easy difficult

Task 2

1 2 3 4 5 6 7 8 9 10
very very
easy difficult