The effect of acute aerobic exercise on stress related blood pressure responses: A systematic review and meta-analysis

Mark Hamer a,*, Adrian Taylor b, Andrew Steptoe a

a Department of Epidemiology and Public Health, Psychobiology Group, University College London, 1-19 Torrington Place, London WC1E 6BT, UK
b School of Sport & Health Sciences, University of Exeter, UK

Received 2 February 2005; accepted 4 April 2005

Abstract

The beneficial impact of regular exercise on cardiovascular health is partly mediated by psychobiological mechanisms. However, the effect of acute exercise on psychobiological responses is unclear. Thus, we performed a systematic review of randomised controlled trials (RCTs) that examined the effect of acute aerobic exercise on blood pressure (BP) responses (the change from baseline to stress) to psychosocial laboratory tasks. Fifteen RCTs met inclusion criteria of which ten demonstrated significant reductions in post-exercise stress related BP responses compared with control (mean effect sizes for systolic and diastolic BP, 0.38 and 0.40). Studies involving greater exercise doses tended to show larger effects, with the minimum dose to show a significant effect being 30 min at 50% $\dot{V}O_2max$. No other moderators emerged from the examination of participant characteristics, research designs and stressor characteristics. In conclusion, an acute bout of aerobic exercise appears to have a significant impact on the BP response to a psychosocial stressor.

#2005 Elsevier B.V. All rights reserved.

Keywords: Cardiovascular health; Aerobic exercise; Blood pressure response; Mental stress; Meta-analysis

1. Introduction

Emerging and recently published scientific evidence has demonstrated a prospective relationship between heightened blood pressure (BP) response to psychosocial stress and hypertension development (Carroll et al., 2001, 2003; Matthews et al., 2004) and progression of carotid atherosclerosis (Barnett et al., 1997; Kamarck et al., 1997; Jennings et al., 2004). Cardiovascular reactivity tests have therefore been proposed as a possible clinical tool for detecting the likelihood of hypertension (Turner, 1994) and provide a useful research paradigm for understanding psycho-physiological mechanisms. The potentially detrimental role of stress highlights the importance of examining and implementing healthcare interventions that control the impact of stress on health. A variety of practical interventions for stress management (to control physiological stress-reactivity) have been proposed including aerobic exercise.

Exercise has been proposed to reduce sympathetic responses to stress (Crews and Landers, 1987) thereby limiting exposure to repeated pathophysiological hypersympathetic arousal. Although there is a general consensus that chronic aerobic exercise (a physiological adaptation to exercise training) exerts a significant anti-hypertensive effect (Whelton et al., 2002) and acute exercise (the physiological response to one bout of exercise) results in post-exercise hypotension (Pescatello and Kulikovich, 2001), the relationship between chronic/acute exercise and stress related BP responses is less clear. Investigators have generally given much more attention to the effects of chronic exercise on stress related cardiovascular responses (Crews and Landers, 1987) due to the cross-stressor adaptation hypothesis. However, repeated exposure to acute bouts of exercise with resulting multiple episodes of reduced cardiovascular reactivity may cumulatively result in cardiovascular health benefits. Earlier studies that examined the effects of chronic exercise on stress related BP responses generally used cross-sectional designs which showed rather small and inconsistent effects (Crews and Landers, 1987).
Cross-sectional studies are potentially confounded by a number of variables, such as psychological and genetic factors, therefore, further studies have used randomised controlled trials (RCTs) to examine chronic exercise effects. However, more recent findings from RCTs have also provided rather mixed and disappointing results. For example, King et al. (2002) demonstrated a significant reduction in the BP response to a speech task after a 12-month-exercise intervention in caregivers, although Georgiades et al. (2000) did not see any changes in stress related BP responses after a 6-month intervention in hypertensive participants, despite observing reductions in absolute BP. Steffen et al. (2001) have also demonstrated an absolute reduction in BP during daily life stressors after a 6-month-exercise intervention, although stress related BP responsiveness was not reported. One potentially confounding factor is that the benefits of exercise for reducing stress related BP responses are confined to a post-exercise window. Thus, if mental stress testing is performed in chronically trained individuals outside this window it is possible that no effects will be observed. Therefore, the main benefits of chronic exercise may be due to the fact regular exercisers are more often in the post-exercise window when they encounter stressors, in addition to demonstrating lower absolute BP levels. Given the potentially confounding impact of acute exercise it is important to study chronic and acute effects separately and also how they may relate to one another. The present review considers the less well-explored issue of acute exercise as a modulator of stress related BP responses. All of the reviewed studies were RCTs and involved an acute bout of aerobic exercise followed by a cardiovascular stress–reactivity test that was compared with a non-exercise control condition.

2. Methods

2.1. Search strategy and inclusion criteria

The present review expanded on a recent review of literature (Taylor, 2000) that examined the effects of acute and chronic exercise on anxiety and stress. The systematic review included a search of MEDLINE and scrutiny of reference lists from relevant reviews and articles. Eligibility for inclusion was independently determined by two authors. Criteria for inclusion were full peer reviewed journal articles in English language, in which the BP response to a psychosocial stress task was determined following an acute bout of aerobic exercise. Also, only RCTs of parallel or within-subject design, where the order of the condition (exercise or control) was counterbalanced between subjects were selected for inclusion.

2.2. Meta-analysis

Stress related BP responses were calculated from the change in BP from baseline to stress task. Where possible an effect size (ES) was then calculated via a t-test using the difference in the BP stress change score from exercise and control conditions and the standard deviation of the change score from the control condition (for within-subject designs) (Clark-Carter, 1997). This was then transformed into a correlation coefficient (r). When using r as a measure of ES it must be transformed into a Fisher’s Z-transformation and then weighted for sample size in order to calculate the combined ES (Clark-Carter, 1997). If a t-score could not be calculated then the F-ratio for the condition × time interaction was used for conversion to r instead. However, in some cases when the findings were not statistically significant there was insufficient data reported to calculate an ES: the results were then treated as having zero effect and the sample size was simply added to the total sample size when calculating the combined ES. An inferential statistic or ES was used to calculate a z-score for each study. Sample size was then used to calculate a combined z-score for all studies from which its probability was found by consulting the standard z-table (Clark-Carter, 1997).

The file-drawer problem represents the number of unpublished, non-significant studies that would have to exist in order to render the meta-analysis non-significant. The file-drawer problem only becomes an issue if the critical number of studies is equal to or greater than the fail-safe N. The fail-safe N was calculated using the following formula (Clark-Carter, 1997): \( k \times (k \times z^2 - 2.706)/2.706 \) and the critical number of studies was calculated as follows: \( (5 \times k) + 10 \). In these calculations, \( z \) denotes the combined standard z-value for the meta-analysis and \( k \) is the number of studies in the meta-analysis.

2.3. Qualitative examination of moderators

Further information was also examined that included participant characteristics (i.e., age, gender, ethnicity, control and post-exercise baseline BP and fitness/physical activity status), experimental design (i.e., between or within subjects design, time period between exercise and stressor, method for assessing BP), exercise characteristics (i.e., intensity, duration and type) and stressor characteristics (i.e., length, type and intensity of challenge). The decision to perform a qualitative examination of moderator variables instead of a statistical analysis was made because of the limited number of available studies.

3. Results

3.1. Post-exercise stress related BP response

The 15 studies that met the inclusion criteria are presented in Table 1. Ten studies demonstrated significant reductions in post-exercise stress related BP responses in comparison with control. The meta-analysis is summarised in Table 2. The combined ES for diastolic BP (DBP) across
## Table 1

Effects of acute aerobic exercise on stress related blood pressure responses

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Design</th>
<th>Stressor</th>
<th>Exercise</th>
<th>Main effects and non-weighted ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brownley et al. (2003)</td>
<td>12 m/12 f (Black and White) mod reactors. (24.5 yrs; 24.4 kg m⁻²)</td>
<td>Within subjects (pre–post-ex)</td>
<td>5 min math task + 3-min speech before and 25-min post-ex</td>
<td>25-min cycle erg @ 70% p VO₂max</td>
<td>Post-ex ↓ DBPR and SBPR during speech (7.7 and 8.8 mmHg) ES = 0.64 and 0.78b</td>
</tr>
<tr>
<td>Bartholomew (2000)</td>
<td>40 T athletes (17 m/23 f) (21.2 yrs; 21.7 kg m⁻²)</td>
<td>Parallel r.a. to ex or contrl</td>
<td>2 min MA and Stroop task + speech task 40 min post-ex</td>
<td>Graded VO₂max treadmill test</td>
<td>Post-ex ↓ MAPR during speech (4 mmHg) ES = 0.85 (given)</td>
</tr>
<tr>
<td>West et al. (1998)</td>
<td>14 m/18 f HT + NT (Black and White) (34 yrs)</td>
<td>Within subjects (counterbalanced conds; ex/contrl)</td>
<td>2 min CP, 6 min MA, 20-min post-ex</td>
<td>20 min cycle erg @ 60–70% age-pred max HR</td>
<td>Post-ex ↓ DBPR during MA (4 mmHg) ES = 0.17b</td>
</tr>
<tr>
<td>Probst et al. (1997)</td>
<td>12 m healthy (10 White, 2 Asian) (19–26 yrs; 24.2 kg m⁻²)</td>
<td>Within subjects (counterbalanced conds; ex/contrl)</td>
<td>4 min Stroop, 1 min CP, pre-ex and 15 min post-ex</td>
<td>30 min cycle erg @ 60% VO₂max</td>
<td>ES = 0.17</td>
</tr>
<tr>
<td>Boone et al. (1993)</td>
<td>8 borderline HT m (41 yrs; 28.8% BF)</td>
<td>Within subjects (counterbalanced conds; ex/contrl)</td>
<td>5 min Stroop, 10 min post-ex</td>
<td>60 min treadmill ex @ 60% VO₂max</td>
<td>Post-ex ↓ DBPR and SBPR (7 and 6 mmHg) ES = 0.99 and 0.99b</td>
</tr>
<tr>
<td>Hobson and Rejeski (1993)</td>
<td>80 f healthy collegiate (18.3 yrs; 21.5 kg m⁻²; 25.9% BF)</td>
<td>Parallel r.a. to 4 grps (10, 25, 40 min ex, contrl)</td>
<td>3 min Stroop, 20 min post-ex</td>
<td>Cycle erg @ 70% HRR for 10, 25 or 40 min</td>
<td>Post-ex ↓ DBPR (3.5 mmHg) in 40 min ex-grp. ES = 0.22a</td>
</tr>
<tr>
<td>Steptoe et al. (1993)</td>
<td>72 m healthy (36 T, 36 U/T) 20.2% BF in (20–35 yrs; 13 and T and U/T)</td>
<td>Parallel r.a. to 3 grps (hard and light ex, contrl)</td>
<td>5 min MA, 3 min public speech 30 min post-ex</td>
<td>20 min cycle erg @ 70% VO₂max (hard ex) or 50% VO₂max (light ex)</td>
<td>Post-ex ↓ DBPR and SBPR during MA in hard ex grp (3 and 7 mmHg) ES = 0.21 and 0.23b</td>
</tr>
<tr>
<td>Szabo et al. (1993)</td>
<td>9 m healthy (31.6 yrs)</td>
<td>Within subjects (counterbalanced conds; ex/contrl)</td>
<td>3 min stroop, 3 min MA,</td>
<td>30 min cycle erg @ 60% VO₂max</td>
<td>NS effects ES = 0c</td>
</tr>
<tr>
<td>Ebbesen et al. (1992)</td>
<td>24 m healthy (18–35 yrs; 24.5 kg m⁻²)</td>
<td>Mixed design [3 conds: 1 h ex, 2 h ex, light ex (3 min)]</td>
<td>2 min stroop, CP, public speech 1, 3, and 24 h post-ex</td>
<td>Cycle erg @ 50–55% VO₂max for 1 or 2 h</td>
<td>NS effects ES = 0c</td>
</tr>
<tr>
<td>Rejeski et al. (1992)</td>
<td>48 f healthy (White and Black) (23–40 yrs; 24.0 kg m⁻²; 26.6% BF)</td>
<td>Within subjects (counterbalanced conds; ex/contrl)</td>
<td>3 min stroop, 3 min public speech 30 min post-ex</td>
<td>40 min cycle erg @ 70% HRR</td>
<td>Post-ex ↓ DBPR and SBPR during stroop/ speech (3.2 and 6.2/1.5 and 4.3 mmHg) ES = 0.58 and 0.61b</td>
</tr>
<tr>
<td>Rejeski et al. (1991)</td>
<td>12 m T cyclists (23–38 yrs; 22.7 kg m⁻²)</td>
<td>Within subjects (3 conds: hard and light ex, contrl)</td>
<td>2 × 3 min stroop 30 min post-ex</td>
<td>30 min @ 50% VO₂max, 60 min @ 80% VO₂max (cycle erg)</td>
<td>Post-ex (hard) ↓ DBPR and SBPR (5.87 and 6.7 mmHg). Post-ex (light) ↓ DBPR (2.2 mmHg). ES = 0.78 and 0.91a</td>
</tr>
<tr>
<td>Roy and Steptoe (1991)</td>
<td>30 m healthy (21yrs; 22.8 kg m⁻²)</td>
<td>Parallel r.a. to 3 grps (hard and light ex, contrl)</td>
<td>4 × 5 min MA 20 min post-ex</td>
<td>20 min cycle erg @ 25 or 100 W</td>
<td>Post-ex (hard) ↓ DBPR and SBPR (5 and 10 mmHg) ES = 0.38 and 0.38c</td>
</tr>
<tr>
<td>Flory and Holmes (1991)</td>
<td>18 f healthy collegiate</td>
<td>Within subjects (counterbalanced conds; ex, contrl)</td>
<td>40 min study 15 min post-ex</td>
<td>20 min dance class @ 60-80% age-pred max HR</td>
<td>NS effects ES = 0c</td>
</tr>
</tbody>
</table>
Table 1 (Continued)  

Study |
---|
**Perronet et al. (1989)** |
Within subjects (counterbalanced) |
3 min stroop 1 h post-ex |
20 min cycle erg |
11±5.5 min |
**Roth (1989)** |
Parallel r.a. to ex or cont. |
MA 20 min post-ex |

**Notes:** Male (m); female (f); trained (T); untrained (UT); hypertensive (HT); normotensive (NT); exercise (ex); control (contrl); random assignment (r.a.); age in years (yrs); body mass index (BMI) in kg m\(^{-2}\); body fat (%BF); hour (h); condition (cond); ergometer (erg); beats per minutes (b min); heart rate reserve (HRR); age predicted maximum heart rate (age-pred max body fat %); cold pressor (CP); mental arithmetic (MA); heart rate reserve (HRR); systolic blood pressure reactivity (SBPR); diastolic blood pressure reactivity (DBPR); non-significant (NS); effect size (ES).  

---

3.2. Participant characteristics

The studies contained a total of 496 participants (46% female) with an age range of 17–60 years. Two studies (West et al., 1998; Boone et al., 1993) involved participants who approached hypertensive status. Boone et al. (1993) demonstrated the largest ES compared with all other studies although their study also involved the oldest participants with a high percentage of body fat so it is unclear whether the magnitude of this effect was due to BP status, age or adiposity. In contrast, West et al. (1998) demonstrated relatively small effects although their participants comprised a mixture of slightly younger hypertensive and normotensives. Only three studies demonstrated a significant post-exercise hypotension effect (Probst et al., 1997; Boone et al., 1993; Steptoe et al., 1993) that was also among the studies to show an attenuation in post-exercise stress related BP responses. However, this observation gives limited support to the possibility that post-exercise stress related BP responses. Interestingly, in this study (Steptoe et al., 1993) the two groups of trained and untrained participants displayed significantly different levels of percentage body fat although this also did not moderate the post-exercise stress related BP response. Similarly, extreme inactive and active groups were also not different in their post-exercise stress related BP response (Roth, 1989). Eight studies involved males only and three studies involved females only with the remaining studies consisting of males and females. Two studies specifically examined the moderating effects of gender on post-exercise stress related BP responses (West et al., 1998; Roth, 1989) and these both demonstrated no gender effect. Information regarding the ethnicity of participants was only provided in four studies (Brownley et al., 2003; West et al., 1998; Probst et al., 1997; Rejeski et al., 1992). However, although Rejeski et al. (1992) showed that ethnicity (black or white) did not impact on their findings, others did not appear to consider this factor in their analyses. The majority of studies reported height and body mass that studies was 0.40, which was significant (P < 0.01) and represented an absolute reduction in post-exercise stress related DBP response on average of 3.0 ± 2.7 mmHg. The combined ES for systolic BP (SBP) was 0.38, which was also significant (P < 0.01), representing an absolute reduction in post-exercise stress related SBP response on average of 3.7 ± 3.9 mmHg. For the five studies that demonstrated non-significant findings an ES of zero was assigned in the absence of sufficient data for the calculation.

The analysis demonstrates that the ‘file-drawer’ problem is not an issue because the critical number of studies is less than the fail-safe N (see Table 2).
facilitates the calculation of body mass index (BMI) for comparison across studies. However, because of the limited range of BMI (21.5–24.5 kg m$^{-2}$) it was difficult to observe any trends in relation to post-exercise stress related BP responses. Lastly, one study pre-screened participants to identify and select those that were moderate to high stress reactors that reported one of the highest ESs for post-exercise stress related BP responses (Brownley et al., 2003).

### 3.3. Design characteristics

Nine studies involved within-subject designs, five involved parallel (between-subject designs) and one mixed design (to examine both between- and within-subject effects) (see Table 1). Of these, five within-subject studies involved fewer than 15 participants, and one between-subject study had only 10 participants in each group. However, qualitative examination of the studies suggested that experimental design had no influence on the ES. The within-subject designs took account of the potential order effects, which could be important in desensitising participants to the stressor, by randomising participants to either exercise or no-exercise condition for their first condition. The time period between exercise and stressor was between 10 min and 24 h, although only one study specifically considered any diminution of effects over time (Ebbesen et al., 1992). Effect size seemed consistent when the tasks were administered up to 30 min post-exercise. Thereafter, Ebbesen et al. (1992) reported a small effect at 1 h post-exercise (but not at 3 and 24 h) and Perronnet et al. (1989) reported no significant effects at 1 h. Finally, three studies (Probst et al., 1997; Steptoe et al., 1993; Flory and Holmes, 1991) collected BP using the finapres device on a beat-to-beat basis, one study (Ebbesen et al., 1992) used ambulatory monitoring, three studies (Boone et al., 1993; Rejeski et al., 1992; Perronnet et al., 1989) used manual auscultation, and the remaining studies used automated devices (microprocessor controlled oscillometric). Two of the studies using automated devices reported no significant effect but six studies reported positive effects (Brownley et al., 2003; Bartholomew, 2000; West et al., 1998; Hobson and Rejeski, 1993; Roy and Steptoe, 1991; Rejeski et al., 1991), thus providing no clear evidence that the BP measuring method will impact on the stress related BP response measures.

### 3.4. Exercise characteristics

A variety of exercise doses were used across and within the studies. Four out of the 10 trials that employed mild to moderate exercise (defined as an exercise intensity less than 60% $V_{O2max}$ or 75% heart rate reserve) demonstrated significant post-exercise attenuation of stress related BP responses (West et al., 1998; Hobson and Rejeski, 1993; Rejeski et al., 1991, 1992). In comparison, seven out of the eight studies that employed vigorous exercise (defined as an exercise intensity greater than or equal to 60% $V_{O2max}$ or 75% heart rate reserve) demonstrated significant effects (Brownley et al., 2003; Bartholomew, 2000; Probst et al., 1997; Boone et al., 1993; Steptoe et al., 1993; Rejeski et al., 1991; Roy and Steptoe, 1991). Exercise sessions lasted from 10 min to 2 h, at intensities of 50–100% of $V_{O2max}$, on a cycle ergometer, treadmill or in an aerobic dance class. The majority of studies that were designed to specifically examine dose–response relationships (i.e., duration and/or intensity of exercise) observed greater effects for the larger exercise dose (Hobson and Rejeski, 1993; Steptoe et al., 1993; Rejeski et al., 1991; Roy and Steptoe, 1991). However, no studies specifically compared interactions between intensity and duration (i.e., contrasting the combined effects of both low and high intensity with short and longer duration). In terms of type of exercise, the majority of studies employed the cycle ergometer, with two studies using the treadmill and one study an aerobic dance class thus making it impossible to determine the impact of exercise mode on the stress related BP response (see Table 1).

### 3.5. Stressor characteristics

The Stroop task was employed in nine studies, mental arithmetic in four studies, cold pressor in three studies, a public speech in five and one a period of study (see Table 1). The stressors lasted from 2 to 20 min (disregarding the one study with 40 min of study time), with 10 studies involving stressors lasting six or fewer minutes. Eight studies involved multiple stressors that employed a combination of Stroop, mental arithmetic, cold pressor and public speech. Interestingly, in three of the studies where multiple stressors were used a reduction in stress related BP response was observed after the second but not the first task (Brownley et al., 2003; Bartholomew, 2000; West et al., 1998) that implies there could have been a habituation process. Nevertheless, other studies demonstrated reduced BP responses in both tasks (Rejeski et al., 1992) and only in the first task (Steptoe et al., 1993). A qualitative comparison of effects across type of stressor revealed no differential pattern, except that no effects were observed following a period of study time.
4. Discussion

The results of the present review suggest an acute bout of aerobic exercise (of moderate to high intensity) attenuates stress-related BP responses. Interestingly, the findings from the present review are comparable to a previous meta-analysis of stress reactivity research (Crews and Landers, 1987) that focused on the effects of fitness (a chronic exercise adaptation). Crews and Landers (1987) reported that greater physical fitness reduced responses to psychological stressors, with a moderate overall effect size of 0.42 for SBP and 0.40 for DBP, using 13 and 17 studies, respectively. Findings from the present review suggest that fitness level has no impact on the post-exercise attenuation of stress-related BP responses. We may, therefore, speculate that it is the effects of acute exercise that are responsible for the rather mixed findings from studies that have considered how chronic exercise impacts upon stress-related BP responses. Therefore, the main benefits of chronic exercise may be due to the fact regular exercisers are more often in the post-exercise window when they encounter daily stressors.

4.1. Potential mechanisms

Few of the reviewed studies specifically attempted to examine mechanisms responsible for the effects of acute post-exercise attenuation of stress-related BP responses. That BP response is a function of both central and peripheral responses, we also considered if HR reactivity, in response to the stressor, was also attenuated by acute exercise in the reviewed studies. HR reactivity to stress was not affected by acute exercise [with the exception of Probst et al. (1997)], thereby suggesting that post-exercise attenuation of BP response is a function of regional vascular resistance (Halliwill, 2001). Indeed, West et al. (1998) observed a significant reduction in vascular resistance during mental challenge following acute exercise. Brownley et al. (2003) showed that a reduced norepinephrine response to the stress task was the best single predictor of the attenuation in post-exercise stress-related BP response. Furthermore, significant increases in post-exercise β1- and β2-receptor responsiveness were observed indicating that the BP response was primarily blunted by enhancing β2-mediated vasodilatation (Brownley et al., 2003). Surprisingly, few studies demonstrated a significant post-exercise hypotension effect that suggests post-exercise hypotension does not impact upon the stress-related BP response itself. Other plausible mechanisms that require further research include the β-endorphin hypothesis (McCubbin et al., 1992, 1995), post-exercise changes in regional cerebral blood flow (Williamson et al., 2004) and psycho-social mechanisms, such as self-efficacy and affective state (Biddle, 2000). For example, Bartholomew (2000) uniquely considered the role of positive, neutral and negative performance feedback on post-exercise stress-related BP responses. The results suggested that the stress response could be favourably manipulated by providing positive feedback, which could be interpreted as social support or self-efficacy enhancement (in different theoretical frameworks).

4.2. Implications

Although seemingly small, the BP stress buffering effect of acute exercise may have significant implications for cardiovascular health. For example, Kamarck et al. (1997) have demonstrated that each standard deviation change in stress-related BP responsiveness is associated with an additional 0.02–0.03 mm of carotid intima-media thickness, which is highly significant given that each incremental 0.1 mm of carotid intima-media thickness is associated with an 11% increased risk of acute myocardial infarction (Salonen and Salonen, 1993). Despite the overall significant effects of acute exercise, it is important to note that five of the 15 studies showed no significant difference in stress-related BP response between exercise and control conditions. One may anticipate that studies involving participants who were healthy and low reactors, a weaker dose of exercise, a weaker stressor and a longer period between exercise and presentation of the stressor will be least likely to show significant effects. However, the studies failing to show significant effects of the treatment had no clear distinguishing characteristics. From a public health perspective, it would be desirable to be able to advocate a minimal or threshold dose of acute exercise that could have a beneficial effect on cardiovascular reactivity, and specifically BP responses. Although stronger effects were observed following vigorous exercise (greater than 60% \( V_{O_{2\max}} \) or 75% HR max, lasting for at least 20 min), a significant effect was still observed at lower intensities, such as 30 min at 50% \( V_{O_{2\max}} \) (Rejeski et al., 1991) and 20 min at 60–70% of age-predicted maximum HR (West et al., 1998). This observation is comparable to findings from the post-exercise hypotensive literature that has demonstrated significant effects following a bout of moderate intensity exercise (30 min, 50% \( V_{O_{2\max}} \)) although greater and long-lasting absolute BP reductions are evident following vigorous exercise (Quinn, 2000). The present review suggests that acute aerobic exercise could provide a buffer to real-life psycho-social stressors (e.g., exams, a public performance, such as a musical concert or public speech, or daily work demands) especially since there is evidence that BP responses to laboratory stressors (as in the present review) correlate to ambulatory BP responses to everyday stressful circumstances (Matthews et al., 1992; Kamarck et al., 2003). Methodologically, the challenge with more ecologically valid stressors is to replicate the intensity of the stressor following exercise and non-exercise conditions in within-subject designs, or create a stressor that has...
the same meaning in parallel designs. Nevertheless, future studies should focus on naturally occurring stressors, which have potentially greater meaning to elicit larger responses and the interaction of person (e.g., anxiety prone, Type A, family history of hypertension, etc.) and situation in contributing to the overall psycho-physiological response. Such studies inevitably may involve stressors that create a range of emotions such as frustration, fear, anger and hostility and loss of control, which may produce quite different and stronger physiological reactions than standard laboratory tasks.

5. Conclusions and recommendations for future studies

Acute exercise appears to have a significant impact on BP responses to psychosocial stress that may have important implications for cardiovascular health. Essentially, reductions in peripheral vascular resistance have been implicated as a key mechanism in the acute post-exercise attenuation of stress related BP responses. That certain populations (such as black and obese individuals) are known to demonstrate exaggerated peripheral vascular responses to psychosocial stress tasks (Falkner, 1996; Davis et al., 1999) suggests that future studies should be designed to examine the effects of acute exercise on stress related BP responses in these populations. From a public health perspective, further studies are also needed that involve moderate intensity exercise, such as brisk walking that match national guidelines for exercise (recently published by Department of Health, UK) in order to determine the minimum exercise dose required to elicit an attenuation effect.

In this review, we have speculated that the effects of acute exercise may confound the impact of chronic exercise upon stress related BP responses, although this conclusion has been drawn from two studies that examined fitness level from a cross-sectional perspective. Therefore, a definitive RCT that examines the influence of a chronic exercise training period on stress related BP responses following acute exercise is required. Further future research should also attempt to employ naturalistic designs that potentially have more relevance to real life. Methodologically, the most appropriate stressors appear to be active cognitive and speech tasks, but when multiple tasks are employed randomisation should be considered to avoid order effects. Finally, a significant limitation of this meta-analysis is the paucity of available studies with interpretable ESs that severely limits the possibility to perform subgroup analyses. Therefore, it is recommended that future studies examining the acute exercise–stress response question should report raw data for each point of time in the study (i.e., post-exercise/pre-stressor, during the stressor and/or immediately post-stressor), change scores for each treatment group and ESs that would facilitate greater interpretation of the findings in this area of research.

References


