Cardiovascular stress reactivity tasks successfully predict the hypotensive response of isometric handgrip training in hypertensives

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Abstract

This study aimed to determine whether: (a) isometric handgrip (IHG) training lowers resting blood pressure (BP), (b) cardiovascular reactivity to a serial subtraction (SST), IHG (IHGT), and cold pressor (CPT) task predicts this hypotensive response, and (c) cardiovascular reactivity is attenuated posttraining. Resting BP and cardiovascular reactivity to a SST, IHGT, and CPT were measured in 24 hypertensives (51–74 years) before and after 10 weeks of IHG training (n = 12) or control (n = 12). IHG training lowered resting BP (Δ8/5 mmHg), whereby the decrease in systolic BP was correlated to pretraining systolic BP reactivity to the SST (r = −.85) and IHGT (r = −.79; all ps < .01), but not the CPT (r = .34; p > .01). Furthermore, following IHG training, systolic BP reactivity to the SST (Δ7 mmHg) and IHGT (Δ8 mmHg) was reduced (all ps < .01). The results offer promising implications for hypertensives and may provide a tool to identify IHG training responders.

Descriptors: Isometric handgrip training, Blood pressure, Cardiovascular reactivity, Hypertension

Hypertension, a disease characterized by chronic elevations in resting systolic (≥140 mmHg) or diastolic (≥90 mmHg) blood pressure (BP), is highly prevalent worldwide and represents a major risk factor for all-cause and cardiovascular disease (CVD)-related morbidity and mortality (Chobanian et al., 2003). Current treatment strategies for hypertension include lifestyle modifications, such as diet and exercise training, as well as the use of antihypertensive medications (Chobanian et al., 2003; Pescatello et al., 2004). Poor effectiveness or adherence to traditional lifestyle modifications and antihypertensive therapies has resulted in inadequate rates of BP control, with recent data suggesting that approximately 50% of individuals treated for hypertension still maintain elevated resting BP (Fitz-Simon, Bennett, & Feely, 2005; Hajjar & Kotchen, 2003; Lloyd-Jones, Evans, & Levy, 2005).

A growing body of research suggests that a novel form of exercise training, isometric handgrip (IHG) training, is a time-efficient and effective method of lowering resting BP in young and old individuals without (McGowan, Levy, McCartney, & MacDonald, 2007; Millar, Bray, MacDonald, & McCartney, 2008; Ray & Carrasco, 2000; Wiley, Dunn, Cox, Hueppchen, & Scott, 1992) and with hypertension, including those receiving pharmacotherapy to treat their high BP (McGowan, Visocchi et al., 2007; Millar, Bray, McGowan, MacDonald, & McCartney, 2007; Millar, Levy, McGowan, McCartney, & MacDonald, 2012; Peters et al., 2006; Taylor, McCartney, Kamath, & Wiley, 2003). A recent meta-analysis supports the efficacy of IHG training, documenting significant reductions in systolic BP (~13 mmHg) and diastolic BP (~8 mmHg) in both normotensive and hypertensive populations (Kelley & Kelley, 2010).

Consistent with observations from aerobic exercise training (Bouchard & Rankinen, 2001), IHG training demonstrates a high interindividual variability in training responsiveness (i.e., reductions in resting BP) (Millar et al., 2007). Typically, individuals with higher pretraining BP values experience greater reductions following training (Millar et al., 2007). Furthermore, lower IHG training responder rates are seen in individuals medicated for high BP (McGowan et al., 2006; McGowan, Visocchi et al., 2007; Stiller-Moldovan, Kenno, & McGowan, 2012), an observation thought to be the result of overlapping mechanisms responsible for BP lowering effects.

It has been postulated that one way to probe interindividual differences in cardiovascular regulation is to assess cardiovascular reactivity responses to psychophysiological stressors (Millar, Bray, MacDonald, & McCartney, 2009). Cardiovascular reactivity represents the acute cardiovascular responses, such as BP and heart rate (HR), elicited by the body in response to physical or mental stressors. To date, numerous large-scale studies have reported that high cardiovascular reactivity is associated with the future development of hypertension and CVD (Carroll et al., 2012; Chida & Steptoe, 2010; Flaa, Eide, Kjeldsen, & Rostrup, 2008; Markovitz, 2010; Moldovan, Kenno, & McGowan, 2012), an observation thought to be the result of overlapping mechanisms responsible for BP lowering effects.

The IHG dynamometers used in this study were donated by Zona Health (Boise, ID). This work was supported by the University of Windsor (Grant # 808316; CLM), a Heart and Stroke Foundation of Canada Postdoctoral Fellowship (PJM), and an Ontario Graduate Scholarship (MBB). Address correspondence to: Mark B. Badrov, MHK, Department of Kinesiology, University of Windsor, 401 Sunset Avenue, Windsor, Ontario, N9B 3P4, Canada. E-mail: badrovvm@uwindsor.ca

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Racynski, Wallace, Chettur, & Chesney, 1998; Matthews et al., 2004), although this observation is not universal (Eich & Jacobsen, 1967; Fauvel et al., 2003). Furthermore, attenuation of cardiovascular reactivity to psychophysiological stressors has been implicated as a mechanism by which exercise training lowers CVD-related morbidity and mortality (Blumenthal et al., 1990;Dimsdale, Alpert, & Schneiderman, 1986).

The utility of cardiovascular reactivity testing to probe the variability in exercise training responses is largely unexplored. In the only published study of IHG training and cardiovascular reactivity, cardiovascular reactivity to a serial subtraction task (SST), but not a cold pressor task (CPT), successfully predicted prior effectiveness of IHG training in reducing resting BP in older, normotensive individuals (Millar et al., 2009). These findings, if replicated in individuals with hypertension (medicated or nonmedicated), may help identify those who will respond to IHG training with a clinically relevant reduction in resting systolic and diastolic BP (i.e., ≥2 mmHg) (Pescatello et al., 2004). In addition, given the potential link between heightened cardiovascular reactivity and future risk of high BP (Carroll, Phillips, Der, Hunt, & Benzeval, 2011; Flaa et al., 2008; Markovitz et al., 1998; Matthews et al., 2004; Matthews, Woodall, & Allen, 1993), managing cardiovascular stress reactivity may be important in the prevention and control of hypertension and CVD. Aerobic exercise studies have reported reductions in cardiovascular reactivity following training interventions (Georgiades et al., 2000; Spalding, Lyon, Steel, & Hatfield, 2004); however, at this time, the effects of IHG training on cardiovascular stress reactivity are unknown.

The purpose of the current study was to replicate previous findings that IHG training leads to clinically relevant reductions in resting BP (≥2 mmHg) in individuals with hypertension, as well as to prospectively investigate whether: (a) cardiovascular stress reactivity responses to psychophysiological stressors are predictive of a post-IHG training hypotensive response, and (b) IHG training attenuates cardiovascular reactivity to psychophysiological stressors. We utilized the SST and IHG task (IHGT), primarily centrally mediated BP stress responses, and the CPT, a primarily peripherally mediated BP stress response, to explore these potential relationships (Kline et al., 2002). Based on previous findings of improved central autonomic function following IHG training (Millar et al., 2012; Taylor et al., 2003), it was hypothesized that: (a) cardiovascular stress reactivity responses to the centrally mediated SST and IHGT, but not the peripherally mediated CPT, would be associated with post-IHG training reductions in resting BP, and (b) cardiovascular reactivity to the SST and IHGT, but not the CPT, would be attenuated following IHG training.

**Table 1. Baseline Characteristics of the Participants**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>IHG training (n = 12)</th>
<th>Control (n = 12)</th>
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</thead>
<tbody>
<tr>
<td>Sex (n)</td>
<td></td>
<td></td>
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<tr>
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<tr>
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<tr>
<td>Resting MAP (mmHg)</td>
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<tr>
<td>Resting PP (mmHg)</td>
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<tr>
<td>Resting HR (beats·min⁻¹)</td>
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<tr>
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<tr>
<td>Diuretic</td>
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<td>ACE inhibitor + calcium channel blocker</td>
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<tr>
<td>ACE inhibitor + diuretic</td>
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</tbody>
</table>

*Note. Values are mean (SD). IHG = isometric handgrip; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial blood pressure; PP = pulse pressure; HR = heart rate; ACE = angiotensin converting enzyme.*

**Method**

**Participants**

Twenty-four hypertensive men (n = 13) and women (n = 11) aged 51–74 years were recruited from Windsor, Ontario, Canada. Participants had a resting systolic BP of ≥140 mmHg, or a resting diastolic BP of ≥90 mmHg, or were receiving pharmacotherapy (for a period ≥4 months) to treat their high BP (83% of participants). Participants were excluded if they were normotensive, had diabetes, a paced rhythm at rest, frequent premature beats, heart failure, or had a physical limitation preventing them from performing IHG exercise. Participant baseline characteristics are displayed in Table 1. The University of Windsor Research Ethics Board approved the investigation, and all participants provided written informed consent prior to participation.

**Study Design**

Upon establishment of eligibility, participants were familiarized to all testing procedures and randomized to an IHG training group (n = 12) or a nonexercising control group (n = 12). Following group assignment, all participants underwent three baseline testing sessions to assess resting BP and HR, as well as cardiovascular reactivity (systolic BP, diastolic BP, and HR response) to a SST, IHGT, and CPT. Participants completed one stress task at each testing session, with order of task randomized for each participant. All three testing sessions were repeated following 10 weeks of IHG training, or no training for the control group, and were conducted within 1 week (but at least 48 h following) of IHG training completion. A schematic representation of the study design and timeline can be found in Figure 1.

**IHG Training Protocol**

Participants randomized to the IHG training group trained 3 days per week for 10 weeks, with the aid of a programmed handgrip dynamometer (ZonaPLUS; Zona Health, Boise, ID). Training consisted of four 2-min bilateral contractions at 30% of maximal voluntary contraction (MVC); determined at the onset of each exercise session, in each hand, via electronic linear load cells contained within each handgrip dynamometer), separated by 1-min rest periods. One training session per week took place under the supervision of an exercise trainer in the Physical Activity and Cardiovascular Research Laboratory at the University of Windsor, while the remaining two sessions were completed in the participants’ home. Participants were provided detailed written instructions on
Isometric exercise and cardiovascular reactivity

Figure 1. Schematic representation of study design and timeline. SST = serial subtraction task; IHGT = isometric handgrip task; CPT = cold pressor task; IHG = isometric handgrip; STAI-Y = State-Trait Anxiety Inventory (Form Y); BP = blood pressure; HR = heart rate; MVC = maximal voluntary contraction.

how to complete the IHG exercise to ensure proper at-home training and completed training log books, in which they recorded the date of exercise completion, MVC scores for each training session, and the final compliance score (i.e., percentage of time the participant maintained 30% MVC). In all cases, compliance scores were ≥ 90%.

In comparison, nonexercising control participants underwent a 10-week nonintervention period. To establish similar face time with study investigators, participants in the nonexercising control group visited the laboratory once per week to have their resting BP measured (Dinamap Carescape v100, Critikon) these data were not used for analysis.

All participants in both groups were given a log book to record any changes in exercise, diet, and medication, and this was discussed with participants at each laboratory visit to ensure these potentially confounding factors remained unchanged throughout the 10-week intervention period.

**Experimental Protocol**

All testing was conducted in a quiet, temperature-controlled laboratory (20–24°C) following a light meal, a 24-h abstinence from vigorous exercise and alcohol, and a 12-h abstinence from caffeine. Participants were asked to void their bladder prior to the start of all testing sessions to minimize the effects of bladder distension on resting BP (Fagius & Karhuvaara, 1989). All testing was conducted within 2 h of initial testing time of day, and time of medication ingestion was standardized (McGowan et al., 2006; McGowan, Visocchi et al., 2005). Four BP and HR measures were obtained by the same trained investigator following the 10-min rest period, each separated by 2 min, and the latter three measures were averaged and used in the final analysis.

**Cardiovascular reactivity.** During the completion of each stress task, systolic BP and diastolic BP were collected every minute (i.e., at Minute 1 and Minute 2) using brachial artery oscillometry (Dinamap Carescape v100, Critikon). HR was collected continuously via single-lead electrocardiography. Data collection was completed using PowerLab acquisition equipment (PowerLab 8/30; AD instruments Inc., Colorado Springs, CO) and LabChart software (LabChart v7.3.2, ADinstruments Inc.). To account for the potential confounding influence of state and trait anxiety on cardiovascular reactivity responses, participants completed a State-Trait Anxiety Inventory (Form Y) prior to all testing days (Spielberger, Gorsuch, & Lushene, 1983).

**Serial subtraction task.** Participants were presented a sequence of 25 four-digit numbers on a computer monitor. Each number was displayed for 5 s. Participants were instructed to mentally subtract a two-digit number from each number and say their answer aloud, prior to the appearance of the next number. At pretesting, participants were instructed to subtract the number 13 from each number presented. At posttesting, participants were presented with a different sequence of 25 numbers, and were instructed to subtract the number 17 from each number presented. The number of correct and incorrect answers was recorded at both pre- and posttesting.

**Isometric handgrip task.** Participants completed a single 2-min IHG contraction at 30% MVC, using their nondominant hand, on a programmed handgrip dynamometer (IBX H-101, MD Systems, Inc., Westerville, OH). Participant compliance scores (i.e., percentage of time the participant maintained 30% MVC) were ≥ 92%. The IHGT protocol was identical at both pre- and posttesting.
Cold pressor task. Participants immersed their right hand (up to the wrist) in a temperature-controlled cold water bath (4 ± 1°C) for 2 min. All participants completed the 2-min protocol. The CPT was identical at both pre- and posttesting.

Statistical Analysis

The current study was powered to detect a change in resting BP following IHG training. Based on mean reductions and SDs of resting BP in hypertensive individuals (medicated and nonmedicated) following IHG training (McGowan, Visocchi et al., 2007; Taylor et al., 2003), with an assigned α of 0.05 and β of 0.2, a maximum of 12 participants per group (IHG training group, n = 12; nonexercising control group, n = 12) was deemed sufficient.

To evaluate changes in resting systolic BP, diastolic BP, mean arterial BP (diastolic BP + 1/3(systolic BP—diastolic BP)), pulse pressure (systolic BP—diastolic BP), and HR following IHG training, as well as to determine whether cardiovascular reactivity to the SST, IHGT, and CPT was reduced, in comparison to the nonexercising control group, two-way analyses of variance (ANOVA)s (Group × Time) with repeated measures were used and Tukey’s post hoc procedures were completed to evaluate specific differences between means, where applicable.

Cardiovascular reactivity (systolic BP, diastolic BP, and HR responses) to the SST, IHGT, and CPT was calculated as the difference between the mean stress task-induced values and the mean baseline resting values, as recommended (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). Dependent and independent t tests were used to examine whether cardiovascular reactivity differed from baseline levels and between stressors. To determine the relationship between cardiovascular reactivity and IHG training effects in the IHG training group, Pearson correlation coefficients between the cardiovascular reactivity values and residualized IHG systolic BP change were assessed. As preintervention resting BP and the magnitude of BP reduction following IHG training have shown correlated effects (Millar et al., 2007), residualized change scores for systolic BP were used, and were obtained by regressing pre–post intervention changes in systolic BP on preintervention systolic BP. Changes in resting systolic BP following IHG training were used due to the magnitude of the reduction.

A Bonferroni’s adjustment was performed to maintain the experiment-wise error rate of α = 0.05. Therefore, statistical significance was set at p ≤ .01. All data were analyzed using STATISTICA (Version 10.0; StatSoft, Inc., Tulsa, OK) software.

Results

All baseline participant characteristics were similar between the IHG training and nonexercising control groups (all ps > .01). Adherence to the IHG training prescription was 100%. All participants in the IHG training group completed 30 IHG exercise sessions over the 10-week intervention period. There were no reported changes in exercise, diet, and medication throughout the investigation in both the IHG training and nonexercising control groups.

Effects of Isometric Handgrip Training on Resting Blood Pressure and Heart Rate

IHG training resulted in significant reductions in resting systolic BP, diastolic BP, mean arterial BP, and pulse pressure (Figure 2).

![Figure 2](image-url)

Figure 2. Effects of 10 weeks of isometric handgrip (IHG) training on (a) systolic blood pressure, (b) diastolic blood pressure, (c) mean arterial blood pressure, and (d) pulse pressure. Values are mean (SE). *Significantly different from pre- (all ps ≤ .01).
Specifically, systolic BP decreased in the IHG training group, 129 (SD = 16) mmHg to 121 (SD = 16) mmHg, p < .01, compared to the control group, 130 (SD = 17) mmHg to 131 (SD = 18) mmHg. Diastolic BP also decreased in the IHG training group, 72 (SD = 9) mmHg to 67 (SD = 8) mmHg, p = .01, compared to the control group, 73 (SD = 12) mmHg to 74 (SD = 14) mmHg. Furthermore, IHG training decreased mean arterial BP, 91 (SD = 11) mmHg to 85 (SD = 10) mmHg, p < .01, compared to the control group, 92 (SD = 13) mmHg to 92 (SD = 13) mmHg, while pulse pressure also decreased in the IHG training group, 58 (SD = 11) mmHg to 54 (SD = 11) mmHg, p < .01, compared to the control group, 57 (SD = 10) mmHg to 58 (SD = 8) mmHg. In contrast, resting HR remained unchanged (p > .01) in both the IHG training group, 68 (SD = 11) beats·min⁻¹ to 68 (SD = 11) beats·min⁻¹, and control group, 67 (SD = 8) beats·min⁻¹ to 68 (SD = 7) beats·min⁻¹. In the IHG training group, 83% of participants experienced a clinically relevant reduction in systolic and diastolic BP ≥ 2 mmHg. Measures of state and trait anxiety remained unchanged in both the IHG training and control groups (all ps > .01).

**Cardiovascular Reactivity as a Predictor of Isometric Handgrip Training Effectiveness**

Cardiovascular stress reactivity responses at baseline to the SST, IHGT, and CPT and the relationship to IHG training effects are displayed in Table 2. The SST, IHGT, and CPT all elicited significant increases in systolic BP, diastolic BP, and HR in both the IHG training and control groups (all ps < .01; see Table 2). In the IHG training group, baseline systolic BP reactivity to the SST (Figure 3a) and IHGT (Figure 3b) was significantly associated with IHG training-induced residualized reductions in systolic BP (all ps < .01), while baseline systolic BP reactivity to the CPT (Figure 3c) showed no associations (p > .01). Diastolic BP and HR reactivity to the SST, IHGT, and CPT showed no associations with IHG training effects (all ps > .01). In contrast, no associations between baseline cardiovascular stress reactivity to the SST, IHGT, and CPT and residualized changes in systolic BP were found in the control group (all ps > .01). State and trait anxiety were not significantly related to any of the cardiovascular reactivity responses to the SST, IHGT, or CPT in both the IHG training group and control group (all ps > .01).

**Effects of Isometric Handgrip Training on Cardiovascular Reactivity**

Cardiovascular reactivity responses at baseline to the SST, IHGT, and CPT did not differ between the IHG training and control groups (all ps > .01). Following IHG training, systolic BP reactivity to the SST and IHGT was significantly reduced in the IHG training group (all ps ≤ .01) compared to the control group. Conversely, diastolic BP and HR reactivity to the SST and IHGT, as well as cardiovascular reactivity to the CPT, remained unchanged in both the IHG training group and control group (all ps > .01). This data is presented in Table 3.

**Discussion**

This randomized controlled trial is the first prospective study to demonstrate that, in individuals with hypertension, systolic BP reactivity to a SST and IHGT are related significantly to reductions in systolic BP following 10 weeks of IHG training. Furthermore, this study demonstrated that systolic BP reactivity to both a SST and IHGT are attenuated following IHG training. Taken together, these findings highlight the promising clinical implications that IHG training may have for individuals with high BP, and may also provide a tool to identify hypertensive individuals who will respond to IHG training with reductions in resting BP.

**Effects of Isometric Handgrip Training on Resting Blood Pressure**

The significant reductions in resting systolic BP (8 mmHg), diastolic BP (5 mmHg), and mean arterial BP (6 mmHg) found in the present study highlight the robustness of the IHG training effect and are confirmatory to previous work in medicated and nonmedicated hypertensives (McGowan, Visocchi et al., 2007; Millar et al., 2007, 2012; Peters et al., 2006; Taylor et al., 2003), as well as normotensives (McGowan, Levy et al., 2007; Millar et al., 2008; Ray & Carrasco, 2000; Wiley et al., 1992). An important extension of the existing work is our novel finding that 10 weeks of IHG training decreases significantly resting pulse pressure (4 mmHg). This is key, as pulse pressure has been shown to be a major determinant of CVD risk and all-cause mortality (Blacher et al., 2000; Franklin et al., 2001). The results of this study provide further evidence for the effectiveness of IHG training in reducing resting BP in individuals with hypertension, even in patients who are currently managed with pharmacotherapy. The reductions in resting BP observed (8/5 mmHg) in the present study are of comparable magnitude to those achieved with a single antihypertensive medication, and are far greater than the 3/2 mmHg and 3/4 mmHg reductions demonstrated in large scale meta-analyses of aerobic and resistance exercise training (Cornelissen & Fagard, 2005a, 2005b). Given the simplicity, portability, and reduced time

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**Table 2. Baseline Cardiovascular Stress Reactivity and the Relationship to Isometric Handgrip Training Adaptations**

<table>
<thead>
<tr>
<th>IHG</th>
<th>ΔSBP (mmHg)</th>
<th>r</th>
<th>p</th>
<th>ΔDBP (mmHg)</th>
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<th>p</th>
<th>ΔHR (bpm)</th>
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<tbody>
<tr>
<td>SST</td>
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<td>-.85</td>
<td>&lt; .01</td>
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<td>9 (7)*</td>
<td>-.63</td>
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<td>IHGT</td>
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<td>-.79</td>
<td>&lt; .01</td>
<td>11 (7)*</td>
<td>-.30</td>
<td>&gt; .01</td>
<td>10 (8)*</td>
<td>-.53</td>
<td>&gt; .01</td>
</tr>
<tr>
<td>CPT</td>
<td>24 (17)*</td>
<td>.34</td>
<td>&gt; .01</td>
<td>13 (7)*</td>
<td>.39</td>
<td>&gt; .01</td>
<td>4 (4)*</td>
<td>-31</td>
<td>&gt; .01</td>
</tr>
</tbody>
</table>
| Control | 83% of participants experienced a clinically relevant reduction in systolic and diastolic BP ≥ 2 mmHg. Measures of state and trait anxiety remained unchanged in both the IHG training and control groups (all ps > .01).

**Note.** Values are mean (SD). SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; IHG = isometric handgrip; SST = serial subtraction task; IHGT = isometric handgrip task; CPT = cold pressor task.

*Significant difference between baseline resting, p < .01.
†Significant difference between stress tasks, p < .01.
IHG training may lead to greater adherence versus more traditional forms of antihypertensive therapy. Therefore, IHG training may offer a viable stand-alone or adjunct therapy to more traditional forms of exercise training or pharmacotherapy in the treatment and management of high BP.

Cardiovascular Reactivity as a Predictor of Isometric Handgrip Training Effectiveness

IHG training has been shown to exhibit interindividual variability in the BP adaptations following training (Millar et al., 2007), which may be related to cardiovascular stress reactivity responses (Millar et al., 2009). This is the first study to note in a prospective design that systolic BP reactivity to a SST and IHGT, but not a CPT, is significantly correlated with IHG training-induced reductions in resting systolic BP. These findings are confirmatory to Millar et al. (2009) who, in a retrospective design, found significant correlations between the cardiovascular reactivity to a SST and IHG training adaptations in older, normotensive individuals. We provide an important novel finding in that the cardiovascular reactivity to an IHGT is also significantly associated with the BP lowering effects of IHG training. Taken together, our results suggest that the systolic BP response to a SST and IHGT are predictive of a post-IHG training hypotensive response in individuals with high BP. Highlighting this relationship provides key clinical implications, as it may provide a simple, effective, and portable tool to identify individuals with hypertension who will benefit from IHG training with a reduction in resting BP. Those individuals deemed to be IHG training nonresponders may be responsive to alternative forms of antihypertensive therapies such as traditional exercise training, diet, or pharmacological interventions.

Effects of Isometric Handgrip Training on Cardiovascular Reactivity

Attenuation of cardiovascular reactivity to psychophysiological stressors has been demonstrated following 6 to 24 weeks of aerobic exercise training in individuals with hypertension (Georgiades et al., 2000; Spalding et al., 2004). To our knowledge, we provide the first evidence that IHG training also reduces cardiovascular reactivity to

<table>
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<tr>
<th></th>
<th>IHG training</th>
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<td>ΔDBP (mmHg)</td>
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<td>Cold pressor task</td>
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<td>ΔHR (bpm)</td>
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</table>

Note. Values are mean (SD). IHG = isometric handgrip; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate.
*Significant difference compared to pre-, p ≤ .01.
psychophysiological stressors in individuals with high BP. Following 10 weeks of IHG training, systolic BP reactivity to both the SST (7 mmHg) and IHGT (8 mmHg) was reduced in the IHG training group, while remaining unchanged in the nonexercising control group. In contrast, cardiovascular reactivity to the CPT was not attenuated following IHG training. It is important to note that only systolic BP reactivity to the SST and IHGT was reduced following IHG training, and not diastolic BP and HR reactivity, likely due to the smaller magnitude of reduction following training in comparison to the larger change in systolic BP. The clinical significance of reduced systolic BP reactivity to a SST and IHGT remains unclear; however, as evidence suggests that increased cardiovascular reactivity to psychophysiological stressors is associated with greater risk of hypertension (Carroll et al., 2012; Flaa et al., 2008; Matthews et al., 1993, 2004), and the additive effects of episodically high stress responses have been implicated in the development of high BP (Carroll et al., 2012, 2004), and the additive effects of episodically high stress responses have been implicated in the development of high BP (Carroll et al., 2012, 2004), and the additive effects of episodically high stress responses have been implicated in the development of high BP (Carroll et al., 2012, 2004), and the additive effects of episodically high stress responses have been implicated in the development of high BP (Carroll et al., 2012, 2004). While our sample size may be considered small by some standards, the current study was sufficiently powered to detect changes in our primary end points, and thus we believe our results to be valid and noteworthy. However, we acknowledge the consequent limitation in our inability to assess the influence of age, sex, and specific medication use on our findings. Furthermore, most participants in the study were medicated for hypertension, medication type was diverse between participants, and some antihypertensive medications have been shown to confound stress reactivity (Benschop et al., 1994). To best account for the effects of antihypertensive medications on cardiovascular reactivity, all medications were strictly monitored throughout the study period, all testing procedures were conducted at a standardized time from medication ingestion, and all medications had been maintained for a period greater than 4 months prior to the study. However, this could also be considered a strength of the investigation, as this is more reflective of individuals with hypertension in the general population.

In conclusion, we noted significant reductions in resting BP following 10 weeks of IHG training in older, hypertensive individuals, which were strongly correlated to pretraining systolic BP reactivity responses to a SST and IHGT. IHG training also produced significant attenuations in systolic BP reactivity to the SST and IHGT. Our findings provide additional support for the use of IHG training as a complementary therapy in the treatment and management of high BP. Furthermore, we offer support for the use of a SST or IHGT as a simple tool to identify IHG training respondents who stand to benefit from this type of exercise. This is especially important, as it may increase the number of successfully controlled or treated individuals.

References


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