8-1999

The effects of walking volume on blood pressure in hypertensive postmenopausal women

Kerrie L. Moreau

To the Graduate Council:

I am submitting herewith a dissertation written by Kerrie L. Moreau entitled "The effects of walking volume on blood pressure in hypertensive postmenopausal women." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Education.

Dixie L. Thompson, Major Professor

We have read this dissertation and recommend its acceptance:

Edward Howley, David Bassett, Kathleen Lawler

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

To the Graduate Council:

I am submitting herewith a dissertation written by Kerrie Lynn Moreau entitled "The Effects of Walking Volume on Blood Pressure in Hypertensive Postmenopausal Women." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Education.

Dixie L. Thompson, Major Professor

We have read this dissertation and recommend its acceptance:

Edward T. Howley

David R. Bassett, Jr.

Kathleen A. Fowler

Accepted for the Council:

Associate Vice Chancellor and Dean of the Graduate School

THE EFFECTS OF WALKING VOLUME ON BLOOD PRESSURE IN HYPTERTENSIVE POSTMENOPAUSAL WOMEN

A Dissertation
Presented for the
Doctor of Philosophy
Degree
The University of Tennessee, Knoxville

Kerrie Lynn Moreau
August 1999

ACKNOWLEDGMENTS

First, I would like to give a special thanks to my advisor and chair of my dissertation committee, Dr. Dixie Thompson. She is one of the best mentors that anyone could ever have. I feel fortunate enough for having the opportunity to work with such an energetic and enthusiastic researcher. I would have never have made it through this process without her guidance and constant support, and for her disregard of the two most common words coming out of my mouth: "I Quit!". I'll miss the 5 am testing as I'm sure she will. Next I would like to thank Dr. Edward Howley, a prominent partner of "Team Catecholamine". Dr. Howley gave me constant support and passed on his knowledge and insight not only in exercise physiology but life in general. I would also like to thank him for allowing me to lead the Friday afternoon "601 B Seminar" and for getting me involved in the very quick and easy catecholamine assay. I would also like to thank Dr. David Bassett for turning me on to the world of pedometers and motion sensors, and for providing me with his knowledge regarding physical activity and hypertension research. I would like to thank Dr. Kathleen Lawler for serving on my committee and introducing me to the psychological research.

I would like to give a special thanks to the following students who helped me out throughout the course of this study with some of the data collection: Julie Hetrich, Jason Langley, Eric Lockhart, Colleen McMahon, Ann Swartz, Scott Strath, Shay Riggs, Robin Boughey and Jim McMahon. I would like to give a special thanks to Racheal DeGarmo who served as my "right-hand" lady. Racheal took a pivotal role in helping with the screening process, and scheduling the women for testing. She also helped out with the dietary analyses as well as the testing procedures. This project would have never been completed without all of your help, thanks.

I would also like to give a special thanks to Pam Andrews, our laboratory technician, who was a great help throughout my career here at UT. Special thanks to Dr. Tom Chen who allowed me to use his laboratory for performing some of my assays.

I would like to express my gratitude to all the women who participated in the study. You all are real "troopers".

Finally I would like to thank my mom and stepfather and the rest of my family back home in Wisconsin. Your love and support has been a constant in my life and I would have never have made it this far with out it.

This research was supported with an American College of Sports Medicine Foundation Research Grant by the American College of Sports Medicine Foundation. Their support is greatly appreciated.

ABSTRACT

The American College of Sports Medicine and the Centers for Disease and Control and Prevention (ACSM-CDC) recommend that 30 minutes of accumulated moderate intensity activity, such as brisk walking, should be performed daily for the attainment of health benefits. However, the effectiveness of these recommendations remain untested, particularly for their efficacy in lowering blood pressure in hypertensive postmenopausal women. Therefore, postmenopausal women with borderline to mild hypertension were randomly assigned to either a 16 kilometer/week (low) walking group (N = 12), 32 kilometer/week walking (high) group (N = 12), or sedentary control group (N = 13). The low and high groups walked an average of 13 and 22 kilometers/week respectively, above their baseline walking activity for 12 weeks, while walking activity remained unchanged for the controls. There were no significant reductions in blood pressure with walking in either low or high group. No significant changes were observed in variables associated with blood pressure including body composition, insulin, glucose, insulin/glucose ratio, or caloric intake. Also, there were no changes in depression, hostility, aggression, or self-esteeem scores with walking. There were also no significant differences in submaximal heart rate or blood pressure, however, the respiratory exchange ratio was significantly reduced in the low and high groups ($p < 0.05$), demonstrating a training adaptation. Women who responded favorably (decrease in systolic or diastolic blood pressure $\geq$ 10 mm Hg) to the walking were characterized by having a greater adherence rate to the walking program, higher baseline levels of blood pressure, and a larger reduction in body mass in comparison to non-responders. It was concluded that a 12 week program meeting or exceeding the ACSM-CDC physical activity recommendations was ineffective in lowering blood pressure in postmenopausal women with borderline to mild hypertension. However, the strong associations between exercise adherence and blood pressure reduction as well as between weight loss and blood pressure decline suggest that these factors are critical to blood pressure reduction in postmenopausal hypertensive women.

TABLE OF CONTENTS

LISTS OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Hypertension is associated with increased risk for the development of cardiovascular disease (CVD) (161, 195). An estimated 43 million US adults are hypertensive, with the highest prevalence rates occurring in men and postmenopausal women (27). After menopause, women are more likely than premenopausal women to suffer from hypertension possibly due to increases in body weight, central obesity, androgen and insulin levels, insulin resistance, and physical inactivity. Several of these factors have been reported to be related and often cluster (62, 115, 172). Obesity can lead to insulin resistance, which can cause hyperinsulinemia to occur (44, 116, 172). The increased insulin levels can cause an increase in renal reabsorption of sodium (43, 78), and can stimulate the sympathetic nervous system (177, 179). Both of these mechanisms can cause blood pressure to increase (43, 177, 179). Physical inactivity is a behavioral factor associated with obesity, insulin resistance, and hypertension (32, 49, 50, 91, 164, 173). In addition to physical precursors, psychological factors including hostility, aggression, and depression have been linked to the development of hypertension (127, 138, 202, 220).

Cardiovascular diseases, blood pressure, and associated factors can be favorably influenced by regular physical activity. In fact, walking for 30-45 minutes, 3 times/week has been reported to decrease the risk of myocardial infarction by 50% in postmenopausal women (132). Epidemiological data suggest that increased levels of physical activity are associated with a lower prevalence of hypertension (164, 173, 225, 226). Other risk factors related to hypertension such as obesity and insulin resistance are also impacted by physical activity. For example, men and women who are physically active have a more favorable body composition in comparison to those who are less active (30, 101, 190, 223, 226). Additionally, physical activity has been shown to be inversely related to the development of insulin resistance (95, 139, 140). Also, physical activity has also been reported to decrease depressive symptoms and improve overall psychological well-being (141, 150). Yet, despite the protective effects of leading an active lifestyle, 59% of US adult women engage in little (less than 3 days/week) leisure-time physical activity (LTPA) and 27% participate in no LTPA (35). There is general agreement that encouraging sedentary individuals to become more active is a good first step, however, experts continue to debate the "optimal" exercise prescription for the attainment of health benefits. The "optimal dose" is the type, intensity, duration, frequency, and total amount of exercise that will maximize physical and psychological benefit while minimizing risk and cost (93).

The current recommendations from the American College of Sports Medicine (ACSM) and the Centers for Disease and Control and Prevention (CDC) suggest that all American adults set a goal of accumulating at least 30 minutes of moderate intensity physical activity on most, if not all, days of the week (166). These recommendations are derived from population based studies reporting a dose-response gradient between total amount of physical activity and health outcomes (134, 159, 169). From these studies, experts concluded that substantial health benefits could be obtained with an energy expenditure of 150 kilocalories/day or 1000 kilocalories/week (166, 216). However, the use of self-reported physical activity data to derive exercise prescriptions lacks precision. While the epidemiological data do show a dose-response relationship across physical activity categories, the threshold of activity needed to derive specific health benefits such as reductions in blood pressure in hypertensive post-menopausal women remains elusive. Haskell (92, 93) reviewed the issue of dose-response and described hypothetical curves representing the relationship between physical activity and selected CVD risk factors. Haskell (92, 93) described the relationship between blood pressure and physical activity as curvilinear, with a 65% response in blood pressure at a caloric expenditure of 1000 kcal/week. Jennings et al. (106) suggest that the relationship between exercise level and blood pressure is sigmoidal. However, the limited number of well-designed experimental studies makes it impossible to clearly identify the shape of the exercise and blood pressure dose-response curve.

The minimum ACSM-CDC physical activity recommendation can be met by performing ordinary tasks such as gardening for 30 minutes a day or brisk walking 1.5 to 2 miles (166, 216). With walking emerging as the mode of choice for most adults who exercise regularly and in those with the lowest prevalence of LTPA (188), intervention studies have examined the blood pressure lowering effect of walking for exercise. Some studies have shown significant improvements in blood pressure with walking programs that are less than the minimum physical activity recommendations (82, 152, 186, 224). Seals et al. (186) reported a significant reduction in both systolic and diastolic blood pressure in postmenopausal women who walked an average of 44 minutes per day, 3 days per week for 12 weeks. Similarily, Motoyama et al. (152) reported a 15 and 9 mm Hg reduction in resting systolic and diastolic blood pressures, respectively, in elderly hypertensive patients who walked for 30 minutes, 3 to 6 days per week. In contrast, other studies have shown no significant improvements in blood pressure with walking programs that have induced a caloric expenditure equivalent to or greater than the minimum recommendations (52, 168, 171, 184). The inconsistent findings can possibly be attributed to differences in study duration, intensity of walking, or subject characteristics (differences in gender, age, baseline levels). Therefore, it is difficult to reach a general consensus on the optimal dose of walking needed to achieve normalized blood pressure

Previous walking studies (52, 82, 105, 171) have examined the dose-response relationship for blood pressure and other CVD risk factors. Hagberg et al. (82) investigated this issue by varying walking intensities (low versus moderate intensity) and reported significant blood pressure changes in both groups. However, this study examined the effect of intensity dose and did not explore the issue of exercise volume, as the total amount of exercise was held constant across the groups. Jennings et al. (105) explored the dose-response issue by using four levels of physical activity: extremely low, normal activity, normal activity plus biking 3 times/week for 40 minutes, normal activity plus biking 7 times/week for 40 minutes. Systolic and diastolic blood pressure was significantly reduced by 10 and 7 mm Hg, respectively, with biking 3 times/week, and by 12 and 7 mm Hg , respectively, with biking 7 times/week. However, the researchers used a cross-over design in which each subject was randomized to each of the four activity groups every four weeks. The possibility exists that four weeks may not be long enough to demonstrate a blood pressure lowering effect or a detraining effect where blood pressure rises back to baseline levels. In contrast to Jennings et al.'s (105) findings, a study that investigated different levels of physical activity through varying the weekly walking volume demonstrated no significant improvements in blood pressure from walking either 17 or 26 kilometers/week for 24 weeks (171). The authors explained this finding by suggesting that the women in their study may have reduced their participation in other activities in order to compensate for the walking program or they inaccurately reported their activity and food intake. The authors were not, however, able to support this supposition.

The primary purpose of this study was to extend previous work and evaluate the effectiveness of the ACSM-CDC physical activity recommendation in reducing blood pressure in borderline to mildly hypertensive postmenopausal women. A second purpose of the study was to examine the dose-response relationship between walking volume and blood pressure reduction in postmenopausal hypertensive women. The prescribed walking mileage for the study's design was chosen so that one group met the ACSM-CDC minimum recommendation of 30 minutes/day (equivalent to walking 2.4 km/day or 16 km/wk), and the other group performed twice that amount. A control group which did not change physical activity was also tested. Because blood pressure response has been reported to be associated with body composition, insulin resistance, insulin levels, hostility, aggression, and depression, a third purpose was to examine whether these variables were affected by dose of walking. A fourth purpose was to identify characteristics that differentiate responders (walkers who dropped resting systolic or diastolic blood pressure by ≥ 10 mm Hg) from non-responders (walkers with < 10 mm Hg decline in systolic or diastolic blood pressure).

CHAPTER II

REVIEW OF LITERATURE

Hypertension is defined as a systolic blood pressure (SBP) $\geq$ 140 mm Hg or diastolic blood pressure (DBP) $\geq$ 90 mm Hg (107). Approximately 24% of the U. S. adult population has high blood pressure, and only 47% of Americans have optimal SBP and DBP levels (< 120 mm Hg and < 80 mm Hg, respectively) (30, 101, 190, 223, 226). The prevalence of hypertension increases with age and is more common in African Americans than Caucasians (27). Young men have a greater risk for developing high blood pressure than young women, but after the age of 55, women are at a greater risk for high blood pressure than similarly aged men (27). The risk for developing hypertension is two to threefold higher in those with blood pressure levels in the high-normal range (SBP of 130-139 mm Hg or DBP of 85-89 mm Hg) than those in the optimal range (131). Although various factors are associated with a greater risk of hypertension, the cause of 95% of the cases of elevation in blood pressure cannot be determined (47).

High blood pressure is a major modifiable risk factor for CVD and contributes to an increased risk for myocardial infarction, congestive heart failure, stroke, peripheral vascular disease, and renal failure (114, 195). The risk for CVD increases in a graded fashion with both increasing SBP and DBP (195). Systolic blood pressure in the high-normal category is associated with two times the risk of death from CVD in comparison to blood pressure in the normal category (195). The risk for CVD morbidity and mortality is potentiated when hypertension clusters with other risk factors such as insulin resistance, glucose intolerance, dyslipidemia, and obesity (5, 114). Several of these risk factors, such as obesity and insulin resistance, may be responsible for the elevation in blood pressure or may work in concert with other mechanisms to cause the elevation in blood pressure. The next section will discuss several postulated mechanisms leading to the development of hypertension.

Mechanisms of Hypertension

Approximately 95% of hypertensive cases have no identifiable cause. This type of hypertension is termed essential or primary hypertension. Secondary hypertension which afflicts 5% of the population of hypertensives, is frequently due to some defined structural or hormonal cause and is often curable (47). The onset of essential hypertension usually occurs after young adulthood and gradually progresses with age while secondary hypertension is rather abrupt (47). The mechanisms responsible for the development of essential hypertension are not totally clear. However, proposed mechanisms include obesity, insulin resistance, hyperinsulinemia, aging, menopause, and physical inactivity. In the following pages these mechanisms will be discussed.

Body Composition

Numerous studies have reported an association between measures of body composition and blood pressure (9, 66, 115, 119, 125, 222). Weight gain and increases in adiposity have been shown to be major contributors to the increase in blood pressure (9, 76, 115). In the Framingham Study, 70% and 61% of the hypertensive cases observed in men and women, respectively, were associated with excess body fat (115). Systolic blood pressure was found to increase by 4.5 mm Hg for every 10 lb increase in body mass in this population (115). A linear relationship between BMI and blood pressure has been observed in populations with BMI between 21 kg/m^2 to 29 kg/m^2 (119). While consistent associations have been reported between body composition and blood pressure, the pathophysiological mechanisms linking obesity and hypertension are not clearly understood (124).

Obesity-related hypertension is characterized by an increase in cardiac output and a normal or slightly elevated systemic vascular resistance (124, 175). The increase in cardiac output is due to hypervolemia resulting from an expansion of extracellular fluid volume (84, 85, 124). The increase in peripheral vascular resistance results from a lack of adaptation of the peripheral resistance to the increase in cardiac output (124). Under normal circumstances, intrarenal and neurohumoral mechanisms regulate fluid balance to prevent the rise in cardiac output and blood pressure. When blood pressure increases, sodium and water are excreted through pressure natriuresis and diuresis. This decreases extracellular fluid volume and venous return, which in turn decreases cardiac output and blood pressure (79). However, in obesity-related hypertension, there is a shift in pressure natriuresis to higher blood pressure levels (84, 85, 124). Hall et al. (124) found the marked retention of sodium and water associated with obesity-induced hypertension in dogs was due to an increase in tubular reabsorption. However, the mechanisms responsible for the increase in tubular reabsorption and sodium retention in obesity are unclear (124).

Several mechanisms have been postulated to explain the shift in pressure natriuresis and altered renal function observed in obese hypertensives. One mechanism that could contribute to the increase in tubular reabsorption and decrease in sodium excretion is activation of the renin-angiotensin system (85). The renin-angiotensin system regulates sodium and fluid balance and subsequently blood pressure by adjusting angiotensin II levels. In situations of low sodium levels or extracellular fluid volume, angiotensin II increases tubular reabsorption to achieve a normal level of fluid volume and blood pressure (148). Even small increases in angiotensin II (5 $ng \cdot kg^{-1} \cdot min^{-1}$) can cause increases in blood pressure especially with an increase in sodium intake and fluid volume (88). In obese hypertensives, the renin-angiotensin system may be enhanced despite volume expansion. Plasma renin activity was increased more than two-fold in obese dogs despite an increase in sodium retention and extracellular fluid volume (85). The authors suggested that the inability to suppress angiotensin II levels appropriately, despite an increase in extracellular fluid volume may be due to an underlying stimulus of renin secretion in obesity (84, 85). One underlying stimulus of renin release may be the activation of the sympathetic nervous system (84). However, the amount of involvement of the renin-angiotensin system in increased pressure natriuresis in obesity-related hypertension remains to be fully elucidated.

An increase in sympathetic nervous system activity is another mechanism linked to the increase in tubular reabsorption, sodium retention, and hypertension in obese individuals (84, 85, 215). Sympathetic nervous system activity as measured via muscle microneurography, has been reported to increase with increasing adiposity (109, 110). Increased caloric intake and weight gain have been shown to increase sympathetic nervous system activity and blood pressure (85). The increased sympathetic nervous system activity observed in obesity-related hypertension could possibly be due to the insulin resistant state and elevated insulin levels that often accompany obesity (60, 61, 112). Insulin can stimulate the sympathetic nervous system (179) and increase sodium reabsorption (43, 78). The sympathetic nervous system may also play a role in increasing peripheral vascular resistance particularly in obese hypertensives who are insulin resistant (124). The relationship between insulin resistance, hyperinsulinemia, and hypertension will be examined in the next section.

Hyperinsulinemia and Insulin Resistance

The previous section examined the link between body composition and blood pressure, with obesity-related hypertension being characterized by a shift in pressure natriuresis and increased activity of renin-angiotensin and the sympathetic nervous system. Another mechanism that has been reported to contribute to obesity-related hypertension is insulin resistance and the compensatory hyperinsulinemia (21, 59, 60, 62, 112, 129, 137, 149, 221). Obesity increases the risk for becoming insulin resistant and hyperinsulinemic (67). Elevated fasting insulin levels have been shown to be independently associated with hypertension (137, 149, 221). Lissner et al. (137) found that women with higher insulin levels ($\geq$ 17 $\mu U \cdot L^{-1}$) were at 3 times the risk of developing hypertension over a 12 year period in comparison to women with lower insulin levels ($\leq$ 10 $\mu U \cdot L^{-1}$), even after adjusting for BMI, waist to hip ratio (WHR), and change in body mass. Ferrannini et al. (62) reported an increase in both systolic and diastolic blood pressure by 2 mm Hg for every increase in insulin resistance of 10 $\mu mol \cdot min^{-1} \cdot kg^{-1}$. Although the previous studies (59, 62, 137, 149, 221) show a relationship between insulin resistance, hyperinsulinemia, and blood pressure, several studies have shown weak or non-existant relationships (94, 155, 212). While it is difficult to ascertain a causal relationship from correlational evidence, several authors have examined the mechanistic link between insulin resistance, hyperinsuinemia, and blood pressure through experimental studies.

Arterial blood pressure has been shown to increase after administration of insulin (21, 22, 64, 177). Several mechanisms have been proposed to explain how hyperinsulinemia elevates blood pressure. One postulated mechanism is that elevated insulin levels cause an increase in sodium retention which can increase extracellular fluid volume, increase cardiac output, and cause blood pressure to rise (43, 64, 175). Finch et al. (64) observed a significant reduction in sodium excretion rates after acute administration of pathophysiologically high levels of insulin in both hypertensive and normotensive rats. While an anti-natriuretic effect was observed, there was no change in mean arterial blood pressure in either the hypertensive or normotensive rats. The authors speculated that blood pressure would possibly increase under chronic administration of insulin. However, several studies reporting an anti-natriuretic effect during chronic hyperinsulinemia have shown no change in blood pressure (23, 86, 87). In fact, Hall et al. (87) noticed a decrease in blood pressure during the first few days of insulin infusion in dogs on a high sodium diet. It is possible that the anti-natriuretic effect observed in the dogs was due to a compensatory mechanism to raise blood pressure back up to normal levels since insulin has been shown to have a vasodilatory response (4, 23, 94, 126). While hyperinsulinemia has been demonstrated to have an anti-natriuretic anti-natriuretic effect, this effect does not appear to be responsible for the increase in arterial blood pressure observed in some studies.

A second mechanism postulated to cause blood pressure to become elevated during hyperinsulinemia is an increase in sympathetic nervous system activity (21, 112, 129, 177). Insulin infusion coupled with euglycemic clamp results in increases in measures of sympathetic nervous system activity (177, 179). Rowe et al. (179) reported a dose-dependent increase in norepinephrine levels along with an increase in arterial blood pressure during a hyperinsulinemic/euglycemic clamp technique. Similarly, Rocchini et al.(177) also showed insulin infusion to result in a dose-dependent increase in both plasma norepinephrine and arterial pressure. Within the same study, the authors examined whether hyperinsulinemia could augment the renin-angiotensin-aldosterone system. They found that the insulin infusions also increased the angiotensin II-stimulated aldosterone production and the pressor response to angiotensin II. The authors speculated that the increased pressor response of angiotensin II was probably due to the increased activation of the sympathetic nervous system. In contrast to these findings, Anderson et al. (4) did not find a concomitant increase in blood pressure after infusing insulin despite increases in both muscle sympathetic nervous system activity and norepinephrine levels. Anderson et al. (4) found that forearm vascular resistance and blood pressure decreased during the insulin infusions, demonstrating a vasodilator response. Thus, it is possible that the increase in sympathetic nervous system activity could be a compensatory response for the drop in blood pressure.

A third mechanism through which hyperinsulinemia may elevate blood pressure is its trophic effect on vascular smooth muscle growth and hypertrophy of the vessel wall (176, 200). Chronic hyperinsulinemia and insulin-induced overstimulation of the sympathetic nervous system can lead to structural and functional changes within the vascular smooth muscle walls (112). Hyperinsulinemia has been shown to cause vascular smooth muscle proliferation (200). The proliferation of the smooth muscle walls can lead to vascular hypertrophy and to the subsequent development of hypertension.

Aging and Menopause

Arterial blood pressure and the risk of developing hypertension increases with age and menopause (27, 56, 194, 228). After menopause, women have higher prevalence rates of hypertension than men and premenopausal women (27, 56, 194, 228). The risk for developing hypertension is 2.2 times higher in postmenopausal women in comparison to premenopausal women (194). This increase in blood pressure has a major impact on health because hypertensive women have higher risks of coronary heart disease and stroke with relative risks of 3.5 and 2.6 respectively, in comparison to normotensive women (63). The elevation in blood pressure observed with aging is more likely due to an increase in systemic vascular resistance since cardiac output remains unchanged or decreases with age (102). Factors that could possibly contribute to the increase in systemic vascular resistance and subsequent elevation in blood pressure include endothelial dysfunction, increased sympathetic nervous system activity, weight gain, and hyperinsulinemia.

A loss of endothelial function has been reported to occur with aging and menopause (10, 29, 204, 205). Several studies have shown that older individuals have a blunted response to agonist-induced vasodilation indicating a decrease in endothelium-dependent vasodilation (29, 204, 205). In men, the age-related decline in endothelium-dependent vasodilation occurs during the third decade and is constant and progressive until old age (29, 204). However, in women, the decline in endothelium-dependent vasodilation does not occur until the fifth decade or after menopause (29, 204). The delayed impairment in endothelial function in women before menopause can be attributed to the protective effect of estrogens on the endothelium. Administration of physiological levels of estrogen potentiates endothelium-dependent vasodilation of the brachial artery (136) and in both the large coronary and microvascular arteries (75). The mechanism through which estrogen protects the endothelium may be a direct effect of estrogen on endothelial cells and their release of vasodilators such as nitric oxide.

Another mechanism occurring with aging that can cause a rise in systemic vascular resistance and blood pressure is an increase in sympathetic nervous system activity (99, 109, 110, 157, 203). Several studies have demonstrated that aging is associated with a marked increase in muscle sympathetic nervous system activity as measured through microneurography (109, 110, 157, 203); however, the mechanisms responsible for this increase are not fully established. While it has been speculated that cardiopulmonary and arterial baroreflexes that inhibit muscle sympathetic nervous system activity are desensitized with age, several studies have shown that neither cardiopulmonary nor arterial baroreflex tonic sympatho-inhibition are diminished with age (39, 40, 208). Chronic elevations of increased sympathetic nervous system activity could potentially lead to structural changes within the vascular wall. Arterial thickening and loss of arterial distensibility has been shown to occur with aging (10, 209) but it is unclear if these changes can be attributed to increases in sympathetic nervous system activity. As mentioned previously, adiposity (84, 85, 215), insulin resistance, and hyperinsulinemia (21, 59, 60, 62, 112, 129, 137, 149, 221) have been postulated as mechanisms responsible for the increase in sympathetic nervous system activity and subsequent blood pressure elevation. Approximately 60% of the variance in age-related increase in muscle sympathetic nervous system activity has been shown to be attributed to increases in total body and abdominal adiposity (109, 110). Weight gain, central redistribution of body fat, and a loss of insulin sensitivity often occur with aging and menopause (33, 56, 180). Higher prevalence rates of obesity and diabetes have been observed in postmenopausal women than premenopausal women (180). The body composition changes that occur after menopause have been attributed to the aging process, however, it has been postulated that the increase in central obesity may be due to estrogen deficiency (33). Thus, the increase in sympathetic nervous system activity and subsequent blood pressure elevation may be in part due to the changes in body composition and insulin sensitivity that can occur with aging and menopause.

Psychological Factors

Chronic psychological factors including hostility, aggression, and depression have been linked with adverse health outcomes. Depression and dimensions of hostility have been shown to be associated with an increased risk for CVD (14, 15, 57, 143, 144) and hypertension (48, 73, 156, 220). The increased risk for CVD and hypertension may be attributed to excessive and chronic cardiovascular (146, 198, 202, 219, 220) and neuroendocrine responses to these behavioral stresses (156, 202, 211). Hostility, aggression, and depression have been associated with greater levels of cardiovascular reactivity (130, 146, 198, 202, 219, 220). Hyperreactivity and altered neuroendocrine function can cause damage to the vessel wall, and thus, can contribute to the development of atherogenesis and hypertension (96). The next section will examine the above mentioned psychosocial factors and their role in the development of hypertension.

Hostility is a psychological factor characterized by various cognitive, emotional, and behavioral traits including cynicism, anger, mistrust, and aggression (13). As mentioned previously, traits of hostility have been linked to the development of hypertension. Several studies have shown that individuals high in traits of hostility demonstrate higher resting systolic and diastolic blood pressures than those low in hostility (17, 37, 48, 77, 193). An inverse relationship between anger expression and blood pressure has been observed with both systolic and diastolic blood pressure increasing with anger suppression (77). Dimsdale et al. (48) found resting systolic blood pressure to increase by 2.5 mm Hg for each increment of suppressed anger in comparison to an increase of 1.4 mm Hg for every 10% of being overweight. However, the association between blood pressure and traits of hostility is not universal (127, 181), and the relationship between hostility and blood pressure may be different between men and women (37, 48, 193).

Some investigators have found no relationship between hostility scores and blood pressure in women (37, 48, 127). Davidson et al. (37) reported hostility scores to be related to a higher resting systolic blood pressure in men, but a lower systolic blood pressure in women. Davidson et al. (37) suggested that men may express hostility more aggressively than women. In contrast, Spicer et al. (193) found higher systolic and diastolic blood pressures in women who were more cynically hostile. Lahad et al. (127) reported no relationship between hostility and the prevalence of hypertension but reported a borderline relationship between aggression and hypertension in postmenopausal women. The differences in findings could possibly be due to differences in the measurement of hostility. The study by Davidson et al. (37) assessed hostility using interview-derived hostility subscales (Potential for Hostility and its subscales), while Spicer et al. (193) measured hostility using the Cook-Medley Hostility Scale, and Lahad et al. (127) used subscales (Hostile-Affect and Aggressive-Responding factors) of the Cook-Medley Hostility Scale. Davidson et al. (37) states that the structured-interview-hostility scale may reflect expressive hostility better than other scales and may be better for predicting cardiovascular disease. They also suggest that the structured-interview scales may measure a different construct in women than in men. Lahad et al. (127) used subscales of the Cook-Medley Hostility Scale because they have been shown to be stonger predictors of mortality and left ventricular dysfunction, and like Davidson et al. (37), Lahad et al. (127) found no relationship between hostility and hypertension in women.

Depression is another psychological factor that has been linked to the development of hypertension. Epidemiological evidence has shown a relationship between depression and hypertension (108, 138, 227). Depressive reactions have been shown to increase blood pressure (220), and in chronic cases may be more harmful than traits of hostility (96). However, as is the case with hostility, research is conflicting regarding the relationship between depression and hypertension, as some studies have shown no relationship between depression and blood pressure response (72, 111). Friedman et al. (72) found no significant relationship between hypertension and depression after eliminating individuals with clinical diagnoses of hypertension or depression. However, by eliminating those with hypertension and depression, a relationship between the two may be less likely to be shown. Also, hypertension was diagnosed as a diastolic blood pressure $\geq$90 mmHg, thus, it is possible that the results would be different if systolic hypertension (SBP $\geq$ 140) was also used. In contrast, data from the National Health and Nutrition Examination Survey (NHANES I) showed high depression to predict the incidence of hypertension (108). Caucasians aged 45-64 and African Americans aged 25-64 with high depression had a relative risk of 1.80 and 2.99, respectively, for hypertension. In this study (108), hypertension was defined as a blood pressure of 160/95 or greater, thus, like the previous study (72), the risk for hypertension could be greater if the present criteria for determining hypertensive status was used.

The increased risk of developing hypertension with various psychological factors (hostility, aggression, and depression) could be due to their effects on cardiovascular and neuroendocrine responses. Several studies have shown traits of hostility and depression to be related to cardiovascular hyperreactivity (130, 146, 198, 202, 219, 220) and higher neuroendocrine responses (156, 202, 211) during laboratory-induced stress. High-renin hypertensives have been shown to exhibit more symptoms of depression, anxiety, and hostility in comparison to low-renin patients (211). Waked et al. (220) found diastolic responsivity to be associated with self-reported depression but not hostility, in borderline hypertensives in comparison to normotensives. In contrast, Miller et al. (146) found normotensive males who expressed high neurotic hostility to demonstrate greater systolic blood pressures, heart rate, and cardiac output responses under harrassment conditions.

Similarly, Suarez et al. (202) expanded these findings to include greater responses in diastolic blood pressure, forearm blood flow, forearm vascular resistance, norepinephrine, cortisol, and testosterone during harassed conditions in individuals exhibiting higher hostility scores than those with low hostility scores. Cardiovascular hyperreactivity and increased neuroendocrine responses may lead to structural changes within the vascular wall, which under chronic conditions could potentially lead to hypertension and CVD.

Physical Inactivity

A sedentary lifestyle has been linked to an increased risk for CVD mortality and morbidity. Epidemiological studies have shown that regular physical activity reduces the risk for CVD and CVD mortality (128, 134, 151, 159, 160, 182). Those who are sedentary have two times the risk of coronary heart disease than those who are active (169). With 25% of Americans being inactive and another 54% engaging in inadequate amounts of activity, sedentary living is a major threat to the health of Americans. Women, African Americans, Hispanics, and the elderly are the least active groups. Physical activity appears to reduce CVD risk by altering risk factors (body composition, blood pressure, insulin sensitivity, and blood lipids). Those who are active have a more favorable CVD risk profile with lower blood pressure, increased insulin sensitivity, favorable blood lipids, and lower body weight and body fat.

In general, physical inactivity has been associated with a higher incidence of hypertension (164, 173). Paffenbarger et al. (164) reported a 35% higher risk of developing hypertension in Harvard male alumni who did not engage in vigorous exercise. Similarly, Reaven et al. (173) reported a higher prevalence of systolic, diastolic, and overall hypertension in older women who were inactive. The beneficial effect of physical activity may be due in part to its effects on other health-related factors associated with hypertension (obesity, insulin resistance, hyperinsulinemia). As observed with hypertension, physical inactivity is associated with higher measures of obesity, weight gain (30), insulin resistance, and hyperinsulinemia (90, 174, 192).

Physical Activity in the Treatment of Hypertension

Hypertension is difficult to treat because of its complex etiology. Different biological and environmental aspects as discussed previously can have a significant role in the development and subsequent treatment of hypertension. The "Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure" (JNC VI) encourages lifestyle modification in the prevention and treatment of high blood pressure, particularly in those with borderline hypertension (130-139/85-89 mm Hg) and stage 1 hypertension (140-159/90-99 mm Hg), and in conjunction with pharmacological therapy in those with higher blood pressure levels (107). Lifestyle modification would include weight reduction, moderation in alcohol intake, reduction in sodium and fat intake, and participation in regular physical activity. It has been suggested that hypertensive women may respond better to lifestyle modifications (weight loss and increased physical activity) than men because obesity is more prevalent among hypertensive women than hypertensive men (117).

The American College of Sports Medicine's 1993 Position Stand: "Physical Activity, Physical Fitness, and Hypertension" recommends endurance exercise training as a non-pharmacological approach to reducing elevated blood pressure (2). The endurance exercise training should include large muscle activities, 3-5 days/week, 20-60 minutes in duration, at an intensity of 50-85% VO_{2max}. The ACSM also recognizes that lower intensity (40-70 % VO_{2max}) exercises can lower blood pressure just as much, or even greater, than higher intensity exercises. Along these same lines, the ACSM and the CDC developed a set of physical activity recommendations geared toward getting more people active. These "Exercise Lite" guidelines recommend that most U.S. adults strive for at least 30 minutes of moderate intensity physical activity on most, and preferably all, days of the week (166). The activity can be intermittent or continuous and can include occupational and nonoccupational tasks. These recommendations are based on epidemiological studies that have shown an inverse relationship between physical activity and CVD mortality and morbidity (95, 139, 140). However, is this truly an optimal amount? In the next sections, epidemiological studies reporting the association between physical activity and blood pressure will be reviewed, and issues of optimal amount and dose-response will be addressed.

Epidemiological Studies

Epidemiological studies examining the relationship between physical activity and blood pressure have reported inconsistent results. Several studies have shown a significant relationships between blood pressure and physical activity (54, 145, 164, 173, 199, 225, 226), while others have reported no association (3, 20, 36, 83, 123). Studies that have reported a relationship between physical activity and blood pressure have shown a reduced incidence of hypertension in those who are active in comparison to those less active (164, 173). Reaven et al. (173) reported a significant trend for prevalence of diastolic hypertension to decrease with each increasing physical activity category in women 50-89 years of age. Even light to moderate activities such as walking, gardening, and swimming were associated with a reduced prevalence for overall, systolic, and diastolic hypertension. However, the amount of activity needed to reduce the risk of hypertension is unknown as frequency and duration of activity were not recorded in this study. Paffenbarger et al. (164) reported an inverse relationship between vigorous exercise and incidence of hypertension in 14,998 Harvard male alumni, with the risk of hypertension decreasing with increasing hours of vigorous sports play per week. Low intensity activities such as walking and stair climbing were not associated with hypertension risk.

The relationship between the amount of physical activity and blood pressure has been investigated across a variety of activities and intensities. Williams et al. (225) reported a significant reduction in both systolic and diastolic blood pressure with increased weekly running distance in recreational female runners after adjustment for age, hormone use, menstrual status, and diet. Systolic blood pressure was reduced by 0.06 mm Hg and diastolic blood pressure by 0.028 mm Hg with each kilometer increase in weekly running distance. Similar results were reported in male runners, with running more than 80 km/week being associated with a 50% reduction in hypertension and 79% reduction in the use of medications for blood pressure control. In contrast to these studies, Kokkinos et al. (123) did not find a relationship between blood pressure and weekly running mileage in 2906 men. The differences in findings could be attributed to the differences in running distance. The highest category in Kokinnos et al.'s study had a mean distance of 32 miles/week, whereas in Williams et al.'s study, the highest category was greater than 50 miles/week. Upon careful observation of the data from Williams et al.'s studies, blood pressure is not significantly reduced until a running distance greater than 30 miles/week in males, and greater than 20 miles/week in females (225, 226). Thus, it is possible that a higher running mileage is needed to exert an effect on blood pressure. Hakim et al. (83) reported no differences in hypertension rates across increasing categories of daily walking mileage in men participating in the Honululu Heart Program. A significant association has been reported between high intensity activity and systolic blood pressure in women, and with diastolic blood pressure in men (145). However, light intensity activity and duration of activity has also been associated with blood pressure levels in women, but not in men (145). Bovens et al. (20) reported greater correlations between fitness and systolic and diastolic blood pressure in women than in men and suggested that the blood pressure response to exercise training may be different between genders.

Intervention Studies

As reviewed earlier, physical inactivity is associated with an increased risk of the development of hypertension. A dose-response inverse relationship has been demonstrated between systolic and diastolic blood pressure levels and increasing amount of physical activity (173). Published reviews of the literature conclude an overall blood pressure lowering effect of physical activity in both normotensive and hypertensive individuals, with the average reduction in both systolic and diastolic blood pressure being approximately 6-9 mm Hg, and a greater blood pressure lowering effect occurring in hypertensives than normotensives (8, 185). Arroll and Beaglehole (8) report that physical activity performed daily appeared to exert a greater effect than activity performed 3 days per week, and activities of moderate intensity showed a reduction in blood pressure equal to that observed for vigorous activities. However, since the publication of that review article, there continues to be debate concerning the optimal amount (intensity, frequency, duration) of activity needed to elicit a blood pressure lowering effect. The ACSM-CDC recommends a minimum of 30 minutes of moderate intensity physical activity for attaining health benefits. However, is this amount of physical activity effective in lowering blood pressure? And is the relationship between physical activity volume and blood pressure linear?

The ACSM-CDC "Exercise Lite" recommendations can be met by brisk walking 1.5 to 2 miles a day in a continuous or intermittent fashion. Several studies have shown significant improvements in blood pressure with walking for exercise (38, 82, 97, 152, 186, 210, 224). Seals et al. (186) reported a significant reduction in both systolic and diastolic blood pressure in postmenopausal women who participated in a 12-week moderate-intensity walking program. The women who walked an average of 44 minutes per day, 3 days per week reduced systolic blood pressure by 10 mm Hg and diastolic blood pressure by 7 mm Hg. Women who were categorized as having stage I hypertension, reduced their blood pressure into the borderline hypertensive range, and those with borderline hypertension reduced their blood pressure into the normal range. The weekly mileage and caloric expenditure (~ 9 miles/week and 800 kcal/week) of the walking program was less than the current ACSM-CDC physical activity recommendations. Other studies using walking programs close to the ACSM-CDC guidelines (30 minutes, 5-7 days per week) have showed similar reductions in blood pressure (97, 152, 210). The decreases in systolic and diastolic blood pressure ranged between 7 to 15 mm Hg, and 4 and 9 mm Hg, respectively.

In contrast to the previous findings (97, 152, 186, 210), others have not found low levels of physical activity to be effective in lowering blood pressure (52, 133, 153, 168, 171, 183). Murphy et al. (153) examined the effects of meeting the ACSM-CDC recommendations either intermittently or continuously on blood pressure in women with desirable blood pressure (SBP of 125 and 124 mm Hg, respectively).

Women participating in the study either walked for 30 minutes once per day, or 10 minutes, three times per day. Both groups increased their fitness level similarly, but did not significantly reduce their blood pressure. Pollock et al. (168) reported similar findings in males who walked for 40 minutes, 4 days per week. One possible explanation for the discrepancies between studies could be that in those studies finding no blood pressure reduction the participants all had desirable blood pressures (52, 133, 153, 168, 171, 183), while participants in the studies that reported a reduction in blood pressure (97, 152, 186, 210) had borderline or stage I hypertension. A greater reduction in blood pressure will occur in those with higher baseline values since there is a greater opportunity for improvement with higher blood pressure values (8). Thus, it appears that activities meeting the "Exercise Lite" recommendations such as walking can be an effective method for reducing blood pressure in individuals with elevated blood pressure, but will have little if any impact on the blood pressure of normotensive individuals.

The optimal exercise intensity for lowering blood pressure is another question often debated. Duncan et al. (52) examined the effects of varying walking intensity (56%, 67% and 86% HR_{max}) on blood pressure in premenopausal sedentary women. All 3 groups walked an average of 15 miles/week. After 24 weeks, there were no changes in blood pressure across walking groups. These were, however, young normotensive women. Hagberg et al. (82) also investigated the effects of varying walking intensity (low vs. moderate intensity) on blood pressure but found differing results. Men and women with essential hypertension walked an average of

51 minutes/day, 3 days per week at either an intensity of 53% $\dot{V}O_{2\,max}$ or at an intensity of 73% $\dot{V}O_{2\,max}$. At the end of the 37-week walking program, diastolic and mean arterial blood pressure were significantly reduced in both training groups. Systolic blood pressure was significantly reduced in the low intensity walking group, but was not significantly altered in the high intensity group. Other investigators using intensites equivalent to the blood lactate threshold (40-60% $\dot{V}O_{2\,max}$) have shown a similar reduction in both systolic and diastolic blood pressure in hypertensive individuals (122, 152). Kiyonaga et al. (122) reported a reduction in systolic and diastolic blood pressure by 20 and 10 mm Hg, respectively, in 50% of the participants after 10 weeks, and in 78% after 20 weeks of cycle training. Motoyama et al. (152) reported a 15 and 11 mm Hg reduction in systolic and diastolic blood pressures, respectively, in elderly hypertensive patients who walked at an intensity equivalent to their lactate threshold (40-60 % $\dot{V}O_{2\,max}$) for 30 minutes, 6 days a week. The reduction in observed in blood pressure observed in the previous studies (122, 152) is comparable to what Cade et al. (28) reported in hypertensive individuals participating in an activity of higher intensity (running 2 miles a day for 3 months). Patients with untreated hypertension decreased their systolic and diastolic blood pressure by an average of 17 and 14 mm Hg, respectively, while patients taking antihypertensive medications experienced drops of 28 and 21 mm Hg in these variables. In general, the findings from intervention studies demonstrate that low, moderate, and high intensity exercise can be used successfully in lowering blood pressure. The impact of the

exercise will be determined by both the pre-intervention blood pressures of the participants as well as by the exercise program itself (exercise mode, frequency, duration, and intensity).

When prescribing physical activity for the treatment of high blood pressure, the dose-response relationship should be known in order to prescribe the optimal amount (intensity, frequency, duration) of activity needed to elicit a blood pressure lowering response. Physical activity meeting the minimum ACSM-CDC recommendations has been shown to be effective in lowering blood pressure in hypertensive individuals, however, can a greater effect be observed with doing more? Is there a ceiling in which no greater benefit is going to occur?

Leon et al. (133) examined whether 2000 kcal/week of moderate intensity physical activity (walking and stair climbing) could improve CVD risk factors, including blood pressure, in normotensive men. After 12 weeks, systolic blood pressure did not significantly change, and diastolic blood pressure rose in an uncharacteristic fashion. In a study that specifically examined the dose response of walking on blood pressure response, no significant improvements in blood pressure were observed with walking either 11 or 17 miles per week (171). In both of these studies (133, 171), the authors attributed the lack of response due to a change in dietary intake or a possible compensatory decline in other types of lifestyle physical activity that the participants were accustomed to doing. Another reason for the failure to demonstrate a drop in blood pressure in these studies could be that the participants were normotensive at the onset of training. However, Jennings et al. (105), also investigating the dose-response relationship between volume of exercise and blood pressure, reported a significant reduction in blood pressure in normotensive men. Their subjects cycled at a moderate intensity (60-70% maximum work capacity) for 40 minutes either 3 or 7 days per week. They found the threshold for lowering blood pressure to be lower than the effects of exercise on fitness or other CVD risk factors. Exercising 3 days per week lowered blood pressure (systolic/diastolic) by 10/7 mm Hg, while exercising 7 days per week decreased blood pressure by 12/7 mm Hg, and thus, the authors concluded that the relationship between blood pressure and exercise volume to be sigmoidal. In a follow-up review, Jennings et al. (106) estimated that the amount of moderate intensity exercise needed for blood pressure changes to occur to be approximately 90 minutes/week, with only minor reductions occurring with additional exercise. However, these findings were observed in young normotensive individuals, thus, it is not known whether the same results would be found in older, hypertensive individuals.

Mechanisms of Depressor Effects of Physical Activity

As observed in the previous section, physical activity has been shown to be an effective non-pharmacological approach to lowering blood pressure in hypertensive individuals. The mechanisms behind the depressor effects of physical activity remain unclear. The decrease in blood pressure can be attributed to a reduction in cardiac output and/or systemic vascular resistance. Total peripheral resistance has been shown to decrease by 15% and 22% with 30 minutes of moderate intensity exercise 3 and 7 days per week, respectively (105). A reduction in sympathetic nervous system activity has been proposed as a mechanism responsible for the reduction in cardiac output and systemic vascular resistance observed with exercise training (7, 8, 122, 217). The change in resting plasma norepinephrine after 10 weeks of exercise training at the lactate threshold (40-60% $\dot{V}O_{2\,max}$) has been shown to be positively correlated ($r = 0.69$, $p < 0.05$) with the change in mean blood pressure (217). Jennings et al. (105) reported a significant reduction in norepinephrine spillover rate (average rate at which norepinephrine released from sympathetic activity enters the plasma) with exercise training seven days per week, however, the response was not uniform across the subjects even though blood pressure and total peripheral resistance responses were. Other mechanisms proposed to cause a decrease in systemic vascular resistance and blood pressure are changes in arterial function and structure (97, 210). Tanaka et al (210) reported a decrease in blood pressure along with an increase in peak limb vasodilatory capacity in hypertensives who walked for 42 minutes/day, 3 days/week at a low intensity. The authors speculated that that the decrease in systemic vascular resistance and blood pressure could be due to increases in arterial wall diameter and restructuring of the arterial walls resulting from the increase in limb blood flow.

As discussed previously, body weight and measures of obesity have been shown to be associated with increases in blood pressure (9, 66, 115, 119, 125, 222). A moderate weight loss will have beneficial effects on several CVD risk factors (118). However, weight loss may not be an important mechanism for blood pressure reduction because some studies have shown blood pressure reduction with physical activity even though body weight and body composition remained unchanged (28, 97, 105, 121, 206, 210, 217). In a review of the literature by Arroll and Beaglehole, (8) they concluded that short term reductions in blood pressure may be independent of weight loss, whereas, longer duration studies appear to have the greatest decrease in blood pressure and also the greatest reduction in body weight. It has been postulated that the reduction in blood pressure with weight loss is secondary to the improvement in insulin sensitivity (201), since insulin levels and insulin sensitivity have been associated with blood pressure response (137, 149, 221).

Physical Activity and Body Composition

Epidemiological evidence shows a dose-response relationship between physical activity and selected measures of body composition (30, 101, 190, 223, 226). Most studies have shown that as the level of physical activity increases, BMI, WHR, and skinfold measures decrease (30, 101, 190, 223, 226), and those who remain active decrease the risk of added weight gain later on in life (30, 70, 113). High intensity physical activity appears to exert a greater effect on body composition (49, 70, 113, 213). However, light to moderate activities, such as walking, can result in weight maintenance and decrease the risk of weight gain (70, 113). Since there is low cost and little risk of injury associated with walking, it may be the activity of choice in prescribing weight reduction and weight maintenance programs.

With the prevalence of obesity still rising (65), there is considerable debate as to effective strategies of inducing weight loss and preventing weight gain. Both caloric restriction and exercise can cause a reduction in body weight (11, 45, 147), however, when severe caloric restriction alone is used, a good proportion of the weight that is lost may come from lean muscle mass (71, 218). Exercise or exercise combined with caloric restriction will preserve lean muscle mass and increase the of weight loss from the fat mass (71, 218), but the type and amount of exercise that will induce a significant amount of weight loss and reduce body fat percentage is debated.

Ballor and Keesey (11) conducted a meta-analysis to examine the effects of type, frequency, and duration of exercise on body weight and body composition. They found energy expended during exercise, as well as the initial body fat levels, account for most of the variance associated with changes in body weight, fat mass, and percent body fat associated with exercise training in men and women. In women, the number of weeks of training and the duration of exercise also predicted body composition changes. In a more recent meta-analysis, the average weight loss through diet, exercise, and diet and exercise was 10.7 kg, 2.9 kg, and 11.0 kg, respectively (147). When controlling for the duration of the weight loss program, exercise alone was associated with a weight loss of 0.2 kg/week, while diet plus exercise was associated with a weight loss of 1.0 kg/week.

There is some debate concerning which factor is more important in inducing weight loss – exercise intensity or exercise volume. Most studies that have used low to moderate intensity activities such as walking have not found a significant weight loss to occur (89, 98, 133, 171, 196). In a study conducted by Hinkleman et al. (98), body weight and body fat percentage did not change in women who participated in a supervised walking program for 15 weeks. The women walked approximately 16 miles/week and expended approximately 1400 kcal/week. The total caloric expenditure for the entire 15-week study was estimated to be 9,975 kcal. Assuming that a net energy balance of 7700 kcal is needed to lose a kilogram of fat, and no other adaptations are occurring, the women should have lost 1.4 kg of body weight. The women also reported consuming fewer calories, thus, an even greater weight loss should have occurred. These findings are consistent with Hardman et al. (89) and Stensel (196) who conducted walking programs of longer duration. Both of these programs were 52 weeks in duration and the weekly energy expenditure from the walking programs should have been approximately 1000-1300 kcal/week, enough to induce a 7-10 kg weight loss. While cardiorespiratory fitness increased in both studies, no weight loss occurred.

Several explanations could possibly explain why body composition changes did not occur in the previous walking programs. One reason is that the participants could have inaccurately reported their food intake as assessed through a dietary recall. Overweight individuals are known to underestimate their food intake when asked to keep record of their diet (6). Another reason could be an over-reporting of the amount of physical activity that they performed. Jakicic et al. (103) found that overweight women had a tendency to over-report their physical activity assessed through a questionnaire in comparisons to physical activity measured with an accelerometer. Also, the women who over-reported their exercise were found to have a poorer weight loss in comparison to those who under-reported their exercise (103). A third possibility could be that the participants decreased the amount of daily physical activities that they were accustomed to performing prior to the adoption of an exercise program.

Only studies that have used more aggressive (in amount, not intensity) walking interventions have shown a significant amount of weight loss to occur (80, 81, 135). Leon et al. (135) reported a 5.7 kg reduction in body weight and a 5% decrease in body fat in obese men participating in a 16 week walking program of 90 minutes/day, 5 days a week. Similarly, Gwinup (81) reported a reduction in body weight by 7.7 kg in women walking 60 minutes/day, seven days a week for 24 weeks. They found in this study (81) and in a previous study (80) that weight loss did not occur until walking duration exceeded 30 minutes daily. However, the current recommendations are for the accumulation of 30 minutes of moderate intensity activity most days of the week. Thus, it is possible that these guidelines may not be sufficient for significant weight loss and body composition reduction, and it may be that only exercise of high intensity or long duration will induce a significant amount of weight loss.

Several authors have examined the issue of exercise intensity on weight loss and body composition changes (12, 26). Bryner et al. (26) investigated the effects of moderate (60-70% HR_{max}) versus high (80-90% HR_{max}) exercise intensity on body composition, weight loss, and dietary composition. The program consisted of walking/jogging at either a moderate or high intensity for 40-45 minutes, 4 days/week. After 12 weeks, body weight did not change in either group, however, percent body fat was significantly reduced in those who participated in the high intensity but not the low intensity exercise. The reduction in body fat percentage that was observed in the high intensity group can be attributed to the greater energy expenditure that occurred since exercise frequency and duration were the same. However, the fact that body weight was not reduced in either group, especially the high intensity group, is puzzling since both groups reported no change in their caloric intake.

Ballor et al. (12) compared the effects of energy restriction in combination with exercising at different intensities (Low vs High) at the same caloric expenditure on body composition changes. Both groups consumed a diet of 1200 kcal/day, and either cycled for 25 minutes, 3x/wk at an intensity of 80-90 % $\dot{V}O_{2\,peak}$, or for 50 minutes, 3x/wk at an intensity of 40-50 % $\dot{V}O_{2\,peak}$. At the completion of the 8-week program, both groups significantly reduced body mass, fat mass, percent body fat, and sum of skinfolds, however, there were no significant differences between the groups in these changes. Body weight was reduced by 5-6 kg and percent body fat was 3-4% lower. While no differences were observed between the two groups in body composition changes, the authors suggested that low-intensity exercise may be a more appropriate for an exercise program geared at weight loss in order to increase exercise adherence in overweight and sedentary individuals.

Physical Activity and Insulin Sensitivity

Epidemiological evidence shows that those individuals who are regularly active have lower insulin levels and lower incidences of insulin resistance, impaired glucose tolerance (IGT), and non-insulin dependent diabetes mellitus (NIDDM) (95, 139, 140). Exercise training benefits those who have NIDDM and IGT by increasing insulin sensitivity, insulin responsiveness, and improving glucose tolerance (191, 214, 229). Non-insulin dependent diabetic patients have been shown to have an increase in insulin sensitivity after several bouts of exercise of high intensity / short duration, or exercise of low intensity / long duration (24). Long-term exercise training can enhance insulin action sufficient to normalize glucose tolerance in some NIDDM and IGT patients (100, 191). However, the optimal amount of physical activity needed to induce such effects are unknown.

Regensteiner et al. (174) estimated from epidemiological data that brisk walking for 1 hour a day would be adequate to induce a 10% decrease in insulin area under the curve from an oral glucose tolerance test. Exercise interventions with training programs of similar magnitude show comparable results in insulin reduction. Trovati et al. (214) conducted a 6-week training study and reported a decrease in glycoslylated hemoglobin, an increase in insulin action, and an improvement in glucose tolerance with 1 hour of cycling exercise, seven days a week, at 50-60% $\dot{V}O_{2\,max}$ in NIDDM patients. Yamanouchi et al. (229) reported an increase in insulin sensitivity in obese NIDDM patients who walked at least 10,000 steps per day for 6-8 weeks. A significant reduction in body weight (8 kg) was also observed and was significantly correlated with glucose metabolic clearance rate (MCR). However, when multiple regression analysis was performed to examine the effects of the change in body weight and the number of steps walked on MCR, only the effect of walking was significant. The significance of these studies is that they demonstrate that low intensity activity such as walking can bring about a significant improvement in insulin sensitivity and glucose tolerance.

Vigorous exercise training programs have a significant effect on insulin action and have even been shown to normalize glucose tolerance in both IGT and NIDDM patients (191). Smutok et al. (191) compared the effects of aerobic exercise training versus strength training in men with either NIDDM, IGT, or hyperinsulinemia. The aerobic exercise program consisted of walking or jogging for 30 minutes on a treadmill at an intensity of 80-85% maximal heart rate reserve, three days per week, while the strength training group performed 11 Nautilus exercises three days per week. Both groups significantly reduced the total plasma glucose and insulin areas under the oral glucose tolerance test (OGTT) curve. However, the OGTT was performed within 24 hours of the last training session, thus, it is possible that the improvement in glucose tolerance and insulin sensitivity was due to the short-term effects of the last training. When the last bout of exercise is controlled for, exercise training has no independent effect on insulin action and no improvement in insulin sensitivity (187). However, all men with IGT in the Smutok et al. study (191) normalized their glucose tolerance after completing the exercise programs.

Some of the improvement in insulin sensitivity and glucose tolerance may be attributed to a reduction in body mass and adiposity. Obese young men decreased insulin levels by 43% and the ratio of insulin/glucose concentrations by 36% after participating in a vigorous walking program that expended 1100 kcal/session, five days per week. The men also significantly reduced their body mass by 5.7 kg and body fat by 5% (135). However, no regression analyses were performed between measures of insulin sensitivity and body composition changes, thus, it is not known whether the improvements in insulin sensitivity that were observed were related to the exercise training or to body composition changes. Holloszy et al. (100) examined the long-term effect of exercise training on insulin action and insulin resistance in patients enrolled in a cardiac rehabilitation program. The patients attended an average 3.7 days per week and walked or ran 3-5 miles each session. After 12 months of participating in the exercise program, a significant reduction in insulin levels was observed. Several NIDDM and IGT patients normalized their glucose tolerance. This finding was independent of body weight changes, as three patients who normalized glucose tolerance but did not lose weight had a similar response to those who did lose weight. The authors concluded that approximately 25-35 km (15-20 miles) per week of running or some other comparable form of exercise is necessary to normalize glucose tolerance. However, because of some of the dangers that are associated with NIDDM (i.e., peripheral neuropathy, foot ulcers, gangrene, hypoglycemia), caution should be taken when recommending running for exercise in patients with NIDDM.

Physical Activity and Psychological Factors

Physical activity has been shown to benefit various psychological factors (18, 25, 51, 120, 141, 165). Epidemiological studies have shown that individuals who are physically active have decreased symptoms of the depression, hostility, aggression, and have higher levels of self-esteem and self-efficacy than sedentary individuals (68, 158, 162, 178, 197). Data from NHANES I showed little or no LTPA to be a significant predictor of increased depressive symptom scores, with women being twice as likely of having depressive symptoms if they participated in little or no LTPA in comparison to those who were active (58). Paffenbarger et al. (162) found that male Harvard alumni who reported participating in more than 3 hours of sports play per week had a 27% lower risk for developing depression than those playing less than 1 hour/week. Within this same cohort, a significant dose-response inverse relationship was observed between physical activity reported in kilocalories/week and depression. Men with physical activity levels of 1,000-2,499 kcal/week and > 2500 kcal/week had a 17% and 28% lower risk of developing depression in comparison to men expending < 1,000 kcal/week. In contrast to these findings, Musante et al. (154) reported a positive relationship between vigorous activity and measures of hostility in both men and women. The authors speculated that individuals may use vigorous activity to deal with their feelings of hostility and aggression.

While epidemiology studies show physical activity to be beneficial in improving psychological functioning, the amount (type, frequency, intensity, duration) of activity needed for improvement is unknown. Both moderate and high intensity exercise programs have been shown to be useful in improving self-esteem and symptoms of depression, anxiety, and hostility (18, 25, 51, 120, 141, 165). Blumenthal et al. (18) reported an improvement in several psychological variables in men and women who participated in a supervised walking and/or jogging program for 45 minutes a day, 3 days/week. By the end of the program, most participants were jogging at an intensity of 70-85% HR_{max}. Those individuals who participated in the 10 week program had lower Profile of Moods State (POMS) scores for depression, fatigue, anger, hostility, and anxiety, while the inactive controls increased their scores for all of these measures. King et al. (120) demonstrated that vigorous exercise is not necessary to achieve psychological benefits. They compared the effects of performing high intensity (73-88% HR_{peak}) versus low intensity (60-73% HR_{peak}) exercise either at home or in a group setting, on depression, anxiety, and perceived stress. At the end of the 12 month program, there was no difference between intensity groups on improvement in the above variables, however greater amounts of exercise participation was associated with lower of amounts of depressive symptoms and anxiety. Martinsen et al. (141) showed that there was no difference between aerobic (brisk walking or jogging) or anaerobic exercise (strength training, flexibility, relaxation) performed 3 days/week in reducing depression scores in patients with clinical depression. These studies provide evidence for the utility of prescribing physical activity based on the current ACSM-CDC recommendations.

While physical activity has been shown to be beneficial to psychological health, some have reported no effect of physical activity on some measures of psychological health (41, 165, 167). De Geus et al. (41) found no effects of intensive exercise training (running, aerobics, soccer/basketball games) 1.5 to 2.5 hours a week on psychological measures of depression, hostility, anxiety, or self-esteem. Similarly, after 16 weeks of either aerobic exercise (70% $\dot{V}O_{2\,max}$) or strength training and flexibility, hypertensive patients in a study conducted by Pierce et al. (167) showed no significant improvement in hostility, anxiety, or depressive symptoms. However, those patients that participated in the exercise programs did perceive themselves as functioning better in cognitive and psychosocial functioning. The findings of no beneficial effect of physical activity on psychological health may be attributed to the fact that the participants of the previous studies (41, 167) had normal psychological functioning (i.e., scored low on depression and hostility scales) prior to beginning the exercise intervention. But as mentioned earlier, the participants in these studies reported fewer psychological problems and improved self-esteem after the training intervention (41, 167).

Conclusion

In general, the data from physical activity interventions show a beneficial effect on blood pressure in individuals with hypertension. However, because of differences in research designs and exercise programs, its difficult to determine the optimal amount of activity needed to lower blood pressure. Significant reductions in blood pressure have been demonstrated with performing activities of low to moderate intensity (82, 122, 152, 186, 210, 217). The average reduction observed in systolic and diastolic blood pressure in well designed studies is approximately 6-7 mm Hg for both variables (8). Individuals with hypertension usually have a greater blood pressure lowering effect and may be able to normalize their blood pressure.

Benefits in insulin sensitivity and glucose tolerance can be observed with both high intensity and low to moderate intensity activity. The amount of improvement is not necessarily dependent on body weight changes, although body weight/fat changes have been associated with improvements in insulin sensitivity. For body weight/fat changes to occur, a negative energy balance must occur. Changes in body mass, fat mass, and body fat percentage have been observed in studies utilizing various exercise intensities. However, it may be more appropriate to prescribe exercise of low to moderate intensity for weight loss in order to increase exercise adherence. In addition to physiological changes, physical activity in general (low, moderate, and high intensity) can improve psychological health by decreasing symptoms of depression, hostility, aggression, and increasing levels of self-esteem.

The ACSM-CDC recommends that US adults accumulate at least 30 minutes or more of moderate intensity physical activity most days of the week. Experts contend that frequent short bouts of moderate intensity activity are as effective as one continuous longer bout in attaining health benefits (166). This notion is based on studies showing similar benefit from performing multiple bouts of activity in comparison to one continuous bout (42, 55, 104). Within the past few years, the effectiveness of these recommendations in eliciting health benefits, including lowering blood pressure, has been investigated (153, 171). Murphy et al. (153) specifically examined the effects of intermittent physical activity (three, 10 minute bouts vs one 30 minute bout) and found this type of physical activity to be ineffective in lowering blood pressure (153). However, one important finding of this study was that excellent adherence was observed to shorter bouts of exercise (153).

Due to differences in study designs it is difficult to detect what the "optimal" amount of activity is to lower blood pressure and improve body composition and insulin sensitivity. The amount of activity necessary for the improvement in blood pressure may not be adequate to benefit body composition or insulin sensitivity. More research needs to be conducted investigating what the optimal amount of activity is for benefiting each of these risk factors.

CHAPTER III

METHODOLOGY

Subjects

Sedentary postmenopausal women with elevated blood pressure were recruited from the Knoxville, Tennessee area via advertisements to participate in a 24 week walking study. Subject inclusion criteria were: cessation of menses for at least 1 year; mild to moderate hypertension (systolic pressure of 130-159 mm Hg and / or diastolic pressure 85-99 mm Hg); not participating in regular physical activity (< 2 d/wk) within the past six months; nonsmoker; no orthopedic limitations to walking; and absence of known cardiovascular disease. Women taking medication known to impact blood pressure were included in the study if (1) the medication had been taken long enough to stabilize blood pressure, and (2) neither the medication nor the dosage was changed during the study. Each subject underwent an initial screening process in which a consent form approved by the University of Tennessee's Institutional Review Board (see Appendix A) was read and signed, a training and health history questionnaire (see Appendix B) was completed, and blood pressure was measured. Women not meeting the requirements were excluded from consideration as subjects.

After completion of the baseline testing, the subjects were assigned to one of three groups: (1) walking 16 km/week (low), (2) walking 32 km/week (high), or (3) control (con), i.e., no change in activity. The groups were randomly assigned as much as possible, however, 8 women assigned to walking groups were placed into the control group because they did not change their level of activity from baseline. Thirty-seven of the original fifty-two subjects completed all phases of the study (12 low, 12 high, 13 control). Two women were moved from the high group to the low group because of inability to meet the higher walking requirements. Seventeen women who completed the study were taking antihypertensive medications (Table 1): 8 taking ACE inhibitors, 5 taking diuretics, 2 taking beta blockers, 2 taking calcium channel blockers, 2 taking a combination calcium channel blocker/ACE inhibitor drug, and 1 taking an alpha blocker. Twenty-seven women were on stable hormone replacement therapy (Table 1). Antihypertensive medications and hormone supplements were maintained throughout the course of the study in each of these women, and all medications were taken at the same time of day when testing was performed. The subject characteristics are presented in Table 1.

Walking Program

In order to control for the possible compensatory decline in other physical activities, the subjects were given a pedometer to wear throughout the day prior to beginning the walking program. The number of steps accumulated each day was recorded for 1-2 weeks and an average value was computed for the week and served as the baseline value for walking that was being performed within their lifestyle. The walking intervention was designed so that one group was meeting the minimum ACSM-CDC physical activity recommendation (166) of walking 2.4 km/day (16km/week) while the other group was walking twice the minimum

Table 1. Subject characteristics at baseline (mean ± SE).

Variable	Control (N = 13)	Low (N = 12)	High (N = 12)
Age (yr)	56.5 ± 1.2	54.9 ± 2.0	53.8 ± 1.4
Body Mass (kg)	76.9 ± 5.5	84.7 ± 5.7	75.4 ± 5.7
Height (cm)	165.9 ± 1.0	165.1 ± 2.0	163.9 ± 2.1
Walking Activity (km/wk)	33.5 ± 3.4	24.9 ± 2.8	25.5 ± 2.8
HRT	6	7	12
Antihypertensive Medications	5	4	7
ACE Inhibitors			
Diuretics	2	1	4
Beta Blockers	3		
Calcium Channel Blocker	1	1	
Alpha Blocker		1	1
Combination ACE/Calcium Channel Blocker		1	1

Control, control group; Low, low mileage; High, high mileage; HRT, hormone replacement therapy.

recommendation (4.8 km/day or 32 km/week). The control group was advised not to make any changes in their baseline physical activity. Each subject in the walking groups was given an Yamax SW-200 pedometer (Yamax, Inc., Tokyo, Japan) to wear daily and a target number of steps to walk weekly in order to meet her prescribed walking distance. Previous work conducted in our laboratory has shown a similar Yamax electronic pedometer (DW-500) to measure walking distances with an accuracy of ± 10% between speeds of 2 and 3.5 miles/hr (16). Women in the control group wore the pedometer for a one-week period each month to ensure that they were maintaining their current lifestyle habits.

Both walking groups were prescribed a distance of 9.6 km above their baseline walking distance during week one. The distance was then increased by 3.2 km/week until the desired walking distance was achieved by the third week in the 16 km/week group or the eighth week in the 32 km/week group. To determine the number of steps needed to meet the prescribed walking distance, stride length was measured in each subject. Stride length was measured by having the subjects walk 10 steps down a hallway at their normal walking pace and dividing the distance covered by 10. This value was then divided into 5280 feet in order to determine the number of steps needed to walk 1.6 km. For example, if a subject's average stride length was 2.5 ft, and given that 5280 feet equals 1.6 km, then approximately 2,000 steps is equal to 1.6 km. Thus, if a subject was in the 16 km/week group she needed to walk approximately 20,000 more steps/week above her baseline weekly steps.

The women were instructed to walk at a self-selected, comfortable pace, and were allowed to accumulate their steps throughout the day to help them achieve their targeted steps. Walking duration, distance, and steps were recorded on daily log sheets along with any additional physical activities. These were collected on a biweekly basis. Other than walking, subjects were asked not to make any changes in their current lifestyle activities. To check this, the Paffenbarger Physical Activity Questionnaire (163) (see Appendix C) was administered at the beginning and at week 12 of the study. This questionnaire measures leisure time physical activity in kilocalories per week and asks questions regarding time spent doing activities such as walking, stair climbing, and recreation and sports play during the past year. Because of the need to record activities completed during the past month rather than the previous year, a modification was made to one of the questions to read: *List any sports or recreation you have actively participated in during the past month*. To encourage subject compliance and maintain contact, supervised walking sessions were held 5 days/week at the outdoor track on the University of Tennessee Knoxville campus. The women in the walking groups were encouraged to attend at least 3 sessions per week.

Testing Procedures

Testing procedures were performed on all subjects at baseline and 12 weeks of the program. On each occasion, the women came into the laboratory after fasting and abstaining from caffeine for at least 10 hours. All testing procedures were conducted in the morning between 0600 and 1000 h and at approximately the same time of day at each testing period.

Blood Pressure and Heart Rate

Resting blood pressure and heart rate were measured in triplicate on all subjects by the same technician who was blinded to the group assignment. On each occasion the subject rested quietly in a seated position for at least five minutes prior to measurement. Heart rate was calculated from a 15 second radial pulse rate. The blood pressure measurements were taken according to the guidelines established by the American Society of Hypertension (1). Blood pressure was taken in the left arm in all subjects using a stethoscope and a mercury sphygmomanometer with at least three minutes separating each measurement. Systolic (SBP) and diastolic (DBP) blood pressures were recorded at the first and fifth Korotkoff sounds, respectively. Mean arterial blood pressure (MABP) was calculated using the following equation: MABP = DBP + 1/3(SBP-DBP). The two closest heart rate and blood pressure readings were averaged and served as the resting values.

Blood Sampling and Analysis

Blood samples were obtained from an antecubital vein. The blood samples were collected in tubes containing EDTA and were used for determination of plasma glucose and insulin concentrations. Samples were centrifuged for 10 minutes, and subsequently plasma was removed and frozen at – 20 °C. Plasma insulin was determined in duplicate using a commercially available radioimmunoassay kit (ICN Biomedicals, Inc., Costa Mesa, CA). For this assay, 100 μL each of insulin standards, controls and subject samples were added to polypropylene tubes coated with anti-insulin serum. To all of these tubes, 900 μL of Insulin-^{125}I Tracer/Insulin Buffer Solution was added. The tubes were then vortexed and incubated at room temperature for 18 hours. Next the free antigen was decanted from the tubes, leaving only antibody-bound antigen. The tubes were then subsequently counted for one minute in a Beckman Gamma 4000 counter (Beckman Instruments Inc., Fullerton, CA) for ^{125}I to determine the level of antibody bound insulin ^{125}I.

Plasma glucose was analyzed in duplicate using the hexokinase (HK) enzymatic method (Procedure No. 16-UV, Sigma Diagnostics, St. Louis, MO). In this assay, 1.0 mL of Glucose (HK) reagent was added to 10 μL of the subject's sample in a cuvet. The Glucose (HK) reagent contains the following ingredients: nicotinamide adenine dinucleotide (NAD), adenosine triphosphate (ATP), HK, glucose-6-phosphate dehyrogenase (G-6-PDH), magnesium ions, and buffer. The assay is based on the following enzymatic reactions:

$$\text{Glucose} + \text{ATP} \xrightarrow{\text{HK}} \text{Glucose-6-phosphate} + \text{adenosine diphosphate}$$

$$\text{G-6-P} + \text{NAD} \xrightarrow{\text{G-6-PDH}} \text{6-phosphogluconate} + \text{NADH.}$$

After allowing the sample and the reagent to incubate at room temperature for 5 minutes, the absorbance of NADH was read at 340 nm with a Spectronic® 21 Spectrophotometer (Milton Roy Co., Rochester, NY).

Body Composition

Body weight was measured to the nearest 0.01 kg using a calibrated scale and height was measured to the nearest 0.1cm. Body mass index was calculated from the ratio of weight (kg) to height (m^2). Waist was measured at the umbilicus, and hip circumference was measured at the maximal circumference of the buttocks with a plastic tape fitted with a tension-handle. Sagittal diameter was measured with an anthropometer at the level of the umbilicus (measured front to back) with the subjects standing. Anthropometric measurements were performed on all subjects in duplicate by the same technician at each testing period.

Body composition was assessed by air displacement plethysmography using the BOD POD® system (LMI, Inc., Concord, CA). The subjects sat inside a sealed chamber wearing either a lycra swimsuit or undergarments and uncorrected body volume was measured. Body volume was corrected for thoracic gas volume using a predicted equation based on gender, age, and height. Thoracic gas volume was predicted instead of measured to maintain consistency between measurement periods. Additionally, it has been reported that there are no significant differences in percent body fat when using predicted compared with measured thoracic gas volume (142). Body density was determined from the ratio of body weight to corrected body volume. Percent body fat was then calculated from body density using the Siri equation (189).

Submaximal Walking Test

Subjects completed a submaximal graded exercise test on a motorized treadmill in order to assess changes in submaximal heart rate, blood pressure, rating of perceived exertion (RPE), and respiratory exchange ratio (RER). The submaximal exercise test consisted of three, 3-minute stages in which the speed remained the same while the elevation increased by 3% each stage. Oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), RER, and heart rate were measured continuously throughout the test. The subjects breathed through a two-way non-rebreathing valve (Hans-Rudolph, Kansas City, MO) while wearing nose clips and oxygen consumption was measured by a S-3A Oxygen Analyzer (Applied Electrochemistry, Inc., Sunnyvale, Ca), and $\dot{V}CO_2$ by a Beckman CO_2 Gas Analyzer Model LB-2 (Sensormedics Corp., Anaheim, Ca). Oxygen consumption, $\dot{V}CO_2$, and RER were recorded each minute using a computer-based system (Rayfield REP 200C Software, Waitsfield, VT), and heart rate was monitored with a heart rate monitor (Polar Electro Inc., Woodbury, NY). The $\dot{V}O_2$, RER, and heart rate during the final minute of each stage were subsequently used in statistical comparisons. Rating of perceived exertion was recorded at the beginning of the third minute of each stage using the Borg Scale (19). Blood pressure was measured during the last minute of each stage of the protocol and during recovery.

Dietary Intake

All subjects were asked to maintain their current dietary habits for the duration of the study. To document dietary compliance, subjects completed a three-day dietary food record at baseline and at week 12 of the study. The food records were analyzed for dietary composition using Nutritionist III (N-Squared Computing, Silverton, OR), a computerized dietary analysis program. The variables of interest included total kilocalories, fat, carbohydrate, and protein percentages, and sodium, potassium, calcium, and alcohol intake.

Psychological Measures

To evaluate changes in psychological factors (depression, hostility, and aggression) associated with hypertension, the Center for Epidemiologic Studies Depression Scale (CES-D) (170) and two sub-scales of the Cook–Medley hostility scale (13, 34) were administered at each testing period (see Appendix D). The CES-D is a validated and reliable scale designed to study the relationship between depression and other variables in the general population. The CES-D scale is comprdised of twenty questions and measures current symptoms of depression including: depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, loss of appetite, and sleep disturbances. Each answer is scored from 0 to 3 on a scale of frequency of occurrence of symptoms.

The Hostile-Affect (HOS) and Aggressive-Responding (AGGR) factors of the Cook-Medley hostility scale were constructed into a questionnaire. The HOS and

AGGR scales have been validated and contain five and nine questions, respectively. The HOS scale measures the experience of negative emotions associated with social relationships, while the AGGR scale measures the tendency to use anger and aggression as responses to problems. The questions are answered as true or false, and are scored from 0 (least hostile) to 5 (most hostile) on the HOS scale, and 0 (least aggressive) to 9 (most aggressive) on the AGGR scale.

The subject's self perception was also measured at each testing period using the Physical Self Perception Profile (PSPP) and the Perceived Importance Profile (PIP) (69) (see Appendix E). The PSPP consists of five, 6-item sub-scales measuring how the subjects rate themselves in terms of sports competence, physical condition, body attractiveness, strength, and overall physical self-worth. The PIP measures how the subjects rate the above factors in assessing their physical self-worth.

Statistical Analyses

A two-way (group x trial) analysis of variance (ANOVA) with repeated measures was used to determine the effects of the walking program on resting blood pressure and heart rate, body composition, glucose, insulin, and psychological variables. Exercise blood pressures, heart rates, $\dot{V}O_2$, RPE, and RER responses during the submaximal exercise tests were statistically analyzed using a three-way (group x time x trial) ANOVA with repeated measures. An independent t-test was used to examine differences between responders and non-responders in resting blood pressure and heart rate, body mass, body fat, anthropometric measusres, and fasting insulin and glucose levels. Pearson product moment correlations were used to determine whether the changes in blood pressure, glucose and insulin levels observed in the walkers and responders were related to baseline values, changes in body composition and anthropometric measures, and changes in walking volume. In addition, correlational analysis was conducted to observe the relationship between the change in blood pressure and the change in glucose and insulin levels. Statistical significance for all tests was established at $p < 0.05$. All analyses were performed using SPSS® 9.0 for Windows (SPSS Inc., Chicago, IL).

CHAPTER IV

RESULTS

Subject and Walking Program Characteristics

Fifty-two women were randomly assigned to either a control, low or high walking group. Women who had an adherence rate of 70% or greater were used in the analyses. Thirty-seven of the original 52 women (13 controls, 12 low group, 12 high group) adhered to the program and completed all phases of the study. Table 1 contains the subject characteristics for all three groups. There were no significant differences in age, height, body mass, or the amount of walking activity.

The women in the low and high groups were instructed to walk 16 and 32 kilometers/week, respectively, in addition to their baseline daily walking activity, while the control group was asked to maintain their baseline level of activity. Figure 1 shows the change in weekly walking volume from baseline among the three groups. Women in the high group had the greatest change in kilometers/week in comparison to women in the low and control group (21.7 ± 0.9, 13.4 ± 1.3, -2.8 ± 1.6 kilometers/week, respectively). The low group walked significantly more than the control group ($p < 0.05$).

Walking distance was increased weekly until the participants in the low and high groups achieved their prescribed distance by either the third or eighth week of the walking program, respectively. The mean walking distances for each week for the low and high groups are presented in Figure 2. The total prescribed walking distance for

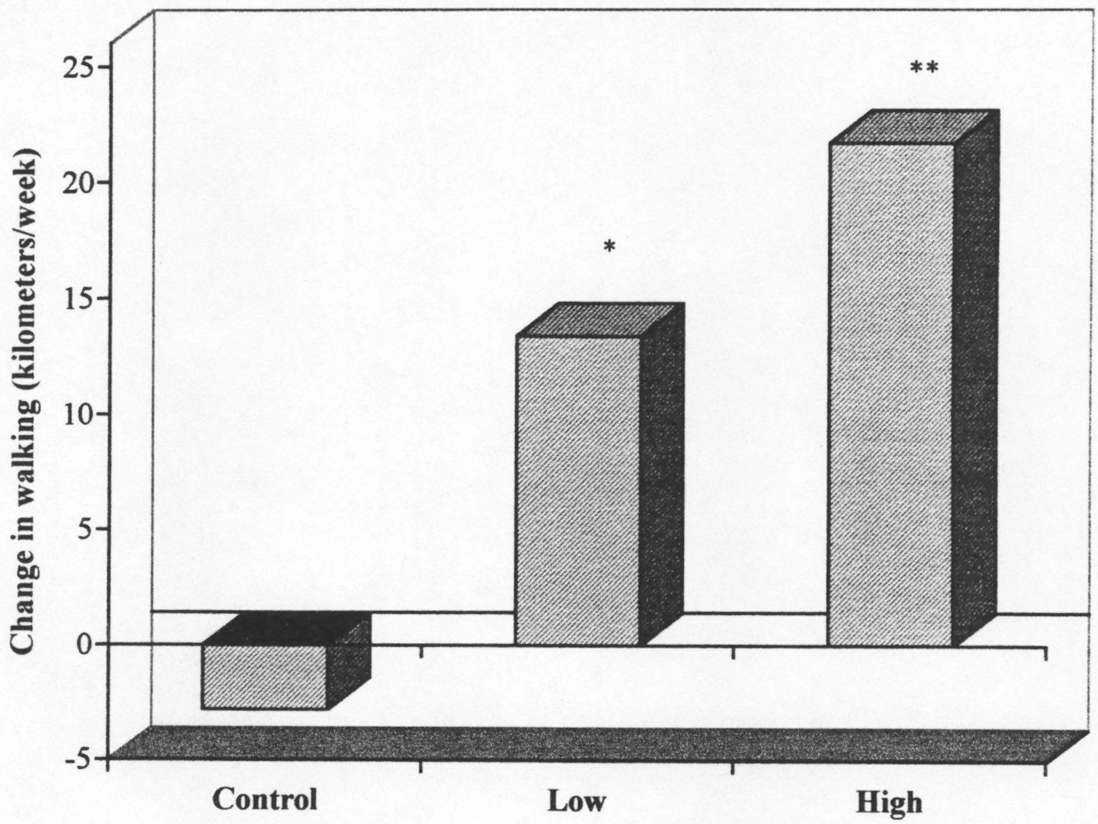


Figure 1. The comparison among the control, low, and high walking groups in the change in walking distance from baseline after a 12 week walking program.
* significant difference low vs control, $p < 0.0001$; ** significant difference high vs low, high vs control, $p < 0.0001$.

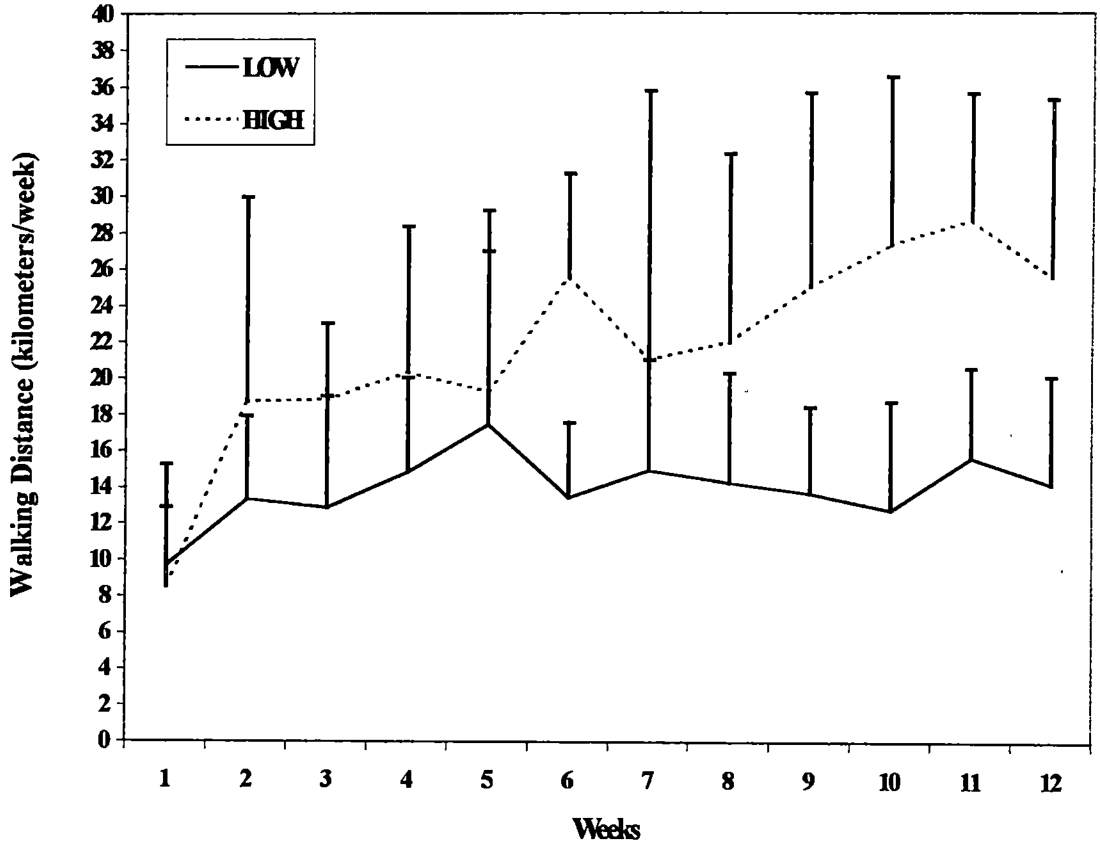


Figure 2. Weekly walking distance above baseline walking activity in the low and high groups during the 12 week walking program (mean + SD).
LOW, low mileage group; HIGH, high mileage group.

the 12 week program was 178 kilometers for the low group and 287 kilometers for the high groups. Women in the low group walked a total of 157.3 ± 7.1 kilometers (88.4% adherence) and women in the high group walked a total of 250.5 ± 32.9 kilometers (87.3% adherence).

Resting Heart Rate and Blood Pressure

The data for resting heart rate and blood pressure are presented in Table 2. No significant group × time interactions or group effects were observed for any of the variables. However, significant time effects for resting heart rate, diastolic blood pressure, and mean arterial blood pressure were observed ($p < 0.05$). Resting heart rate, diastolic blood pressure and mean arterial blood pressure were lower after 12 weeks. Resting systolic and diastolic blood pressures were reduced by 0.6/1.3, 4.8/1.5, and 3.4/4.2 mm Hg, and mean arterial blood pressure by 1.1, 2.6, and 3.9 mm Hg in the control, low and high groups, respectively (Figure 3). However, the reductions in these blood pressures were not differentially statistically significant across groups.

Correlational analyses demonstrated that the changes in systolic ($r = 0.46$, $p = 0.023$) and mean arterial ($r = 0.42$, $p = 0.040$) blood pressures with 12 weeks of walking were significantly related to the change in body mass (Figure 4, Table 3). Changes in systolic and mean arterial pressure were also related to the change in BMI. ($r = 0.43$, $p = 0.035$; $r = 0.42$, $p = 0.04$, respectively), indicating that those women who

Table 2. Resting heart rate and blood pressure (mean ± SE).

Variable	Control N = 13	Low N = 12	High N = 12
Heart Rate (beats·min^{-1})			
Baseline	75.1 ± 2.6	75.3 ± 2.7	76.2 ± 2.7
12 week	71.4 ± 2.2	71.7 ± 2.2	71.0 ± 2.2
SBP (mm Hg)			
Baseline	140.4 ± 2.7	142.9 ± 2.9	137.2 ± 2.9
12 week	139.8 ± 3.3	138.1 ± 3.5	133.8 ± 3.5
MABP (mm Hg)			
Baseline	102.4 ± 1.6	102.8 ± 1.7	101.6 ± 1.7
12 week	101.3 ± 2.2	100.2 ± 2.3	97.7 ± 2.3
DBP (mm Hg)			
Baseline	83.7 ± 1.6	83.1 ± 1.7	84.1 ± 1.7
12 week	82.4 ± 2.2	81.6 ± 2.3	79.9 ± 2.3

Control, control group; Low, low mileage group; High, high mileage group; SBP, systolic blood pressure; MABP, mean arterial blood pressure; DBP, diastolic blood pressure.

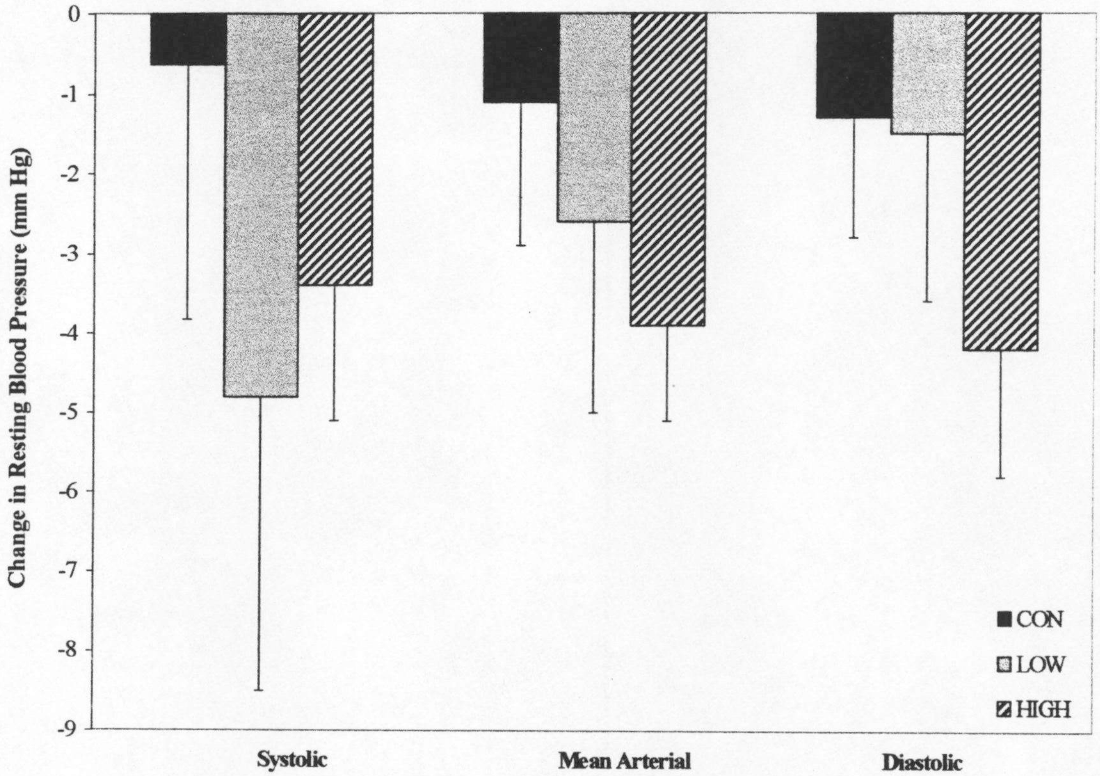
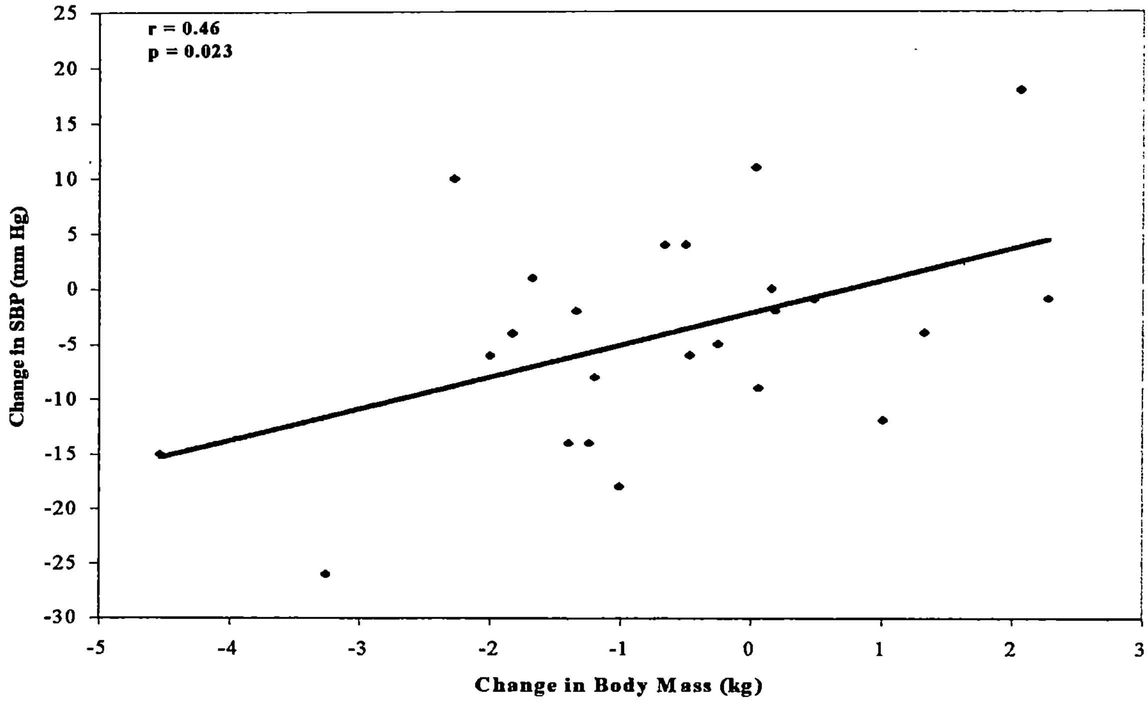


Figure 3. The change in resting systolic, mean arterial, and diastolic blood pressure from baseline to 12 weeks. Values for the control group (CON), low mileage group (LOW), and high mileage group (HIGH) are presented (mean ± SE).



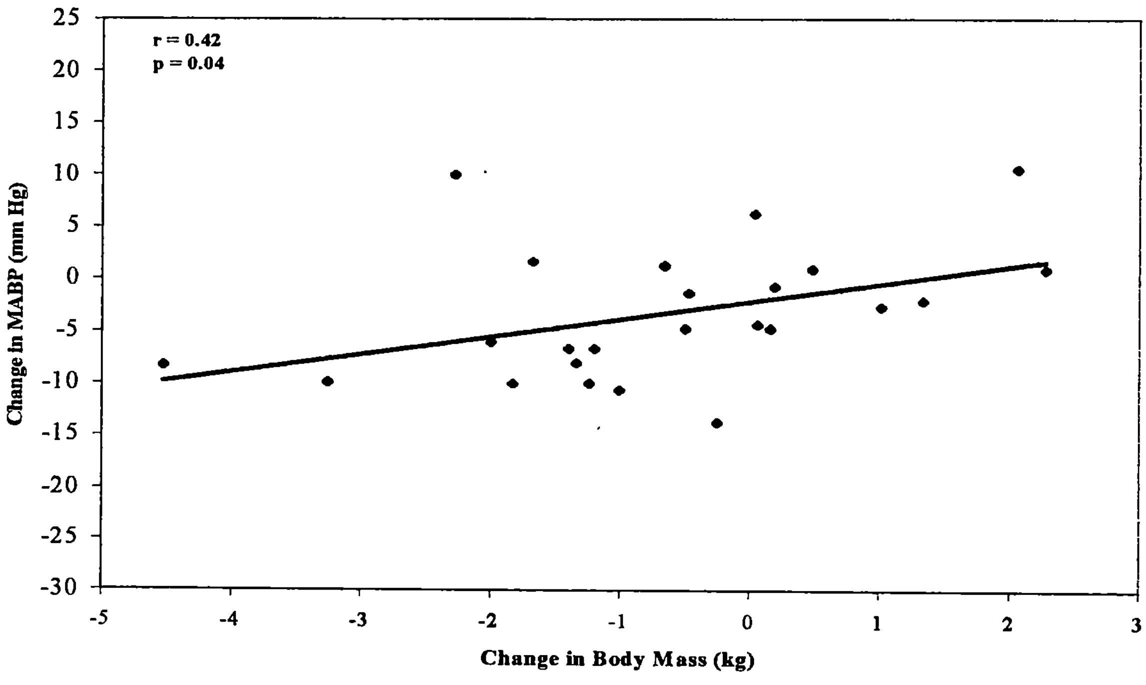


Figure 4. The relationship between the change in body mass and changes in systolic and mean arterial blood pressure.
SBP, systolic blood pressure; MABP, mean arterial blood pressure.

Table 3. Correlational analyses between changes in resting blood pressure and selected variables.

Variable	Δ SBP (mm Hg)	Δ MABP (mm Hg)	Δ DBP (mm Hg)
Baseline SBP (mm Hg)	-0.42*	-0.25	-0.05
Baseline DBP (mm Hg)	-0.08	-0.32	-0.41*
Baseline MABP (mm Hg)	-0.33	-0.35	-0.28
Δ **Body Mass** (kg)	0.46*	0.42*	0.28
Δ **BMI** ($kg \cdot m^{-2}$)	0.43*	0.39	0.26
Δ **Body Fat** (%)	0.08	0.67	0.04
Δ **Waist** (cm)	-0.04	-0.33	-0.45*
Δ **Hip** (cm)	-0.25	-0.21	-0.12
Δ **Sagittal** (cm)	0.15	-0.01	-0.13
Δ **Walking Activity** (km/week)	-0.12	-0.32	-0.38
Total Walking (km)	0.22	0.15	0.05
Compliance (%)	-0.53**	-0.48**	-0.32
Δ **Insulin** ($\mu IU \cdot ml^{-1}$)	0.27	0.19	0.09
Δ **Glucose** ($mmol \cdot L^{-1}$)	-0.30	-0.30	-0.22
Δ **Insulin/Glucose**	0.32	0.27	0.16

*Significant relationship, $p < 0.05$; ** Significant relationship, $p < 0.01$.
Control, control group; Low, low mileage group; High, high mileage group; SBP, systolic blood pressure, DBP; diastolic blood pressure; MABP, mean arterial blood pressure; BMI, body mass index.

lost the most weight had the greatest reduction in blood pressure. The change in systolic and diastolic blood pressures were related to baseline levels with $r = -0.42$, $p = 0.041$ and $r = -0.41$, $p = 0.045$, respectively (Figure 5, Table 3). The changes in systolic and mean arterial blood pressures were also related with adherence to the walking program with $r = -0.53$, $p = 0.008$ and $r = -0.48$, $p = 0.017$, respectively (Figure 6, Table 3). This indicates that those women who adhered to the walking program had the greatest decreases in blood pressures. There were no significant correlations between the changes in blood pressures with the change in walking distance. However, there was a trend for diastolic blood pressure to decrease in those who had the greatest change in walking activity from baseline ($r = -0.38$, $p = 0.065$).

Stepwise multiple regression analyses were used to determine which variables accounted for the variances in the changes in systolic, mean arterial, and diastolic blood pressures. For each of the dependent variables, the change in body mass, the change in weekly walking distance, and baseline levels of either systolic, mean arterial, or diastolic blood pressure were included into the model. For the changes in systolic and mean arterial blood pressure only the change in body mass contributed significantly to the predictive equations. For the change in diastolic blood pressure, only baseline level of diastolic blood pressure was a significant predictor, none of the other variables (change in body weight or change in weekly walking distance) added significantly to the model.

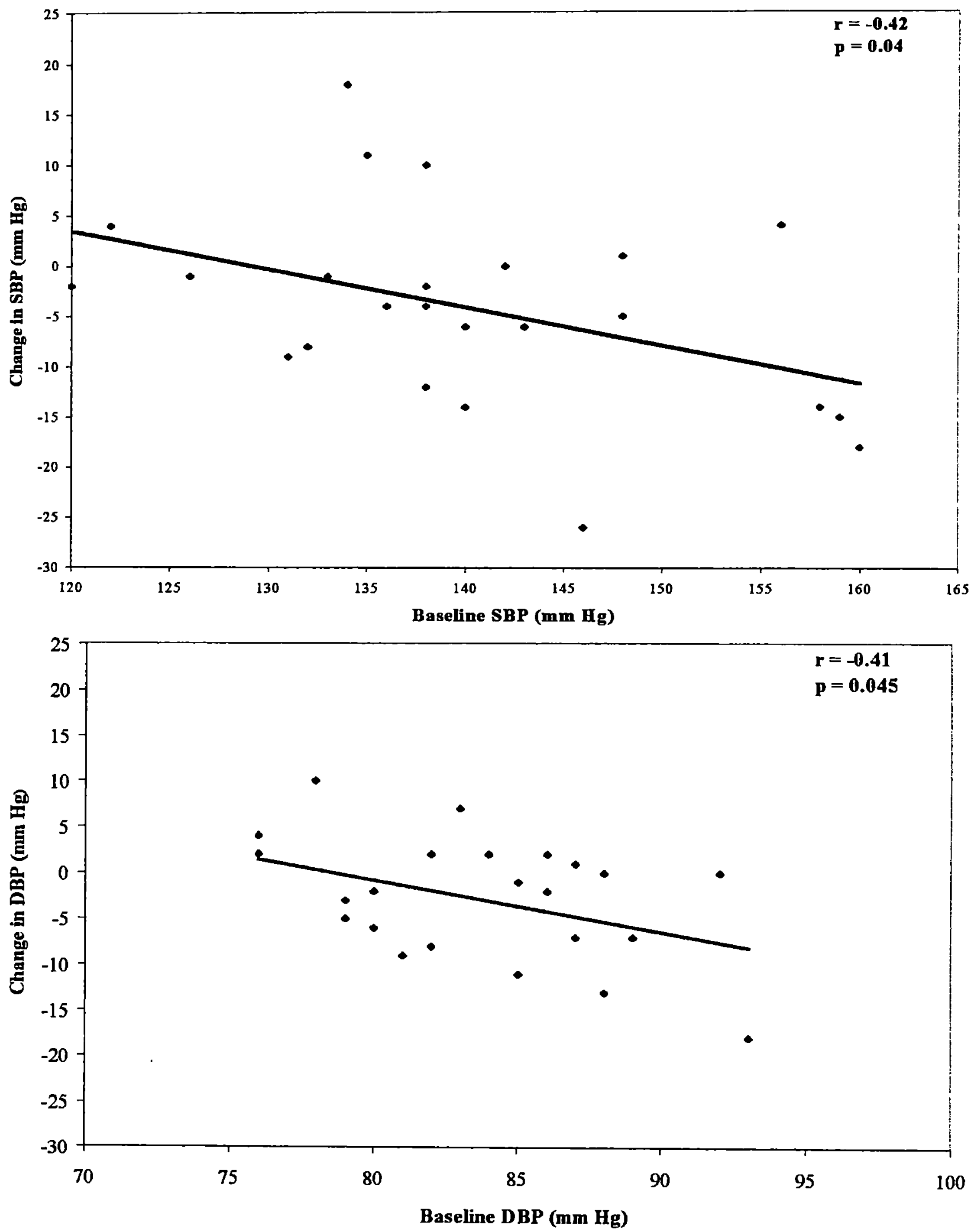
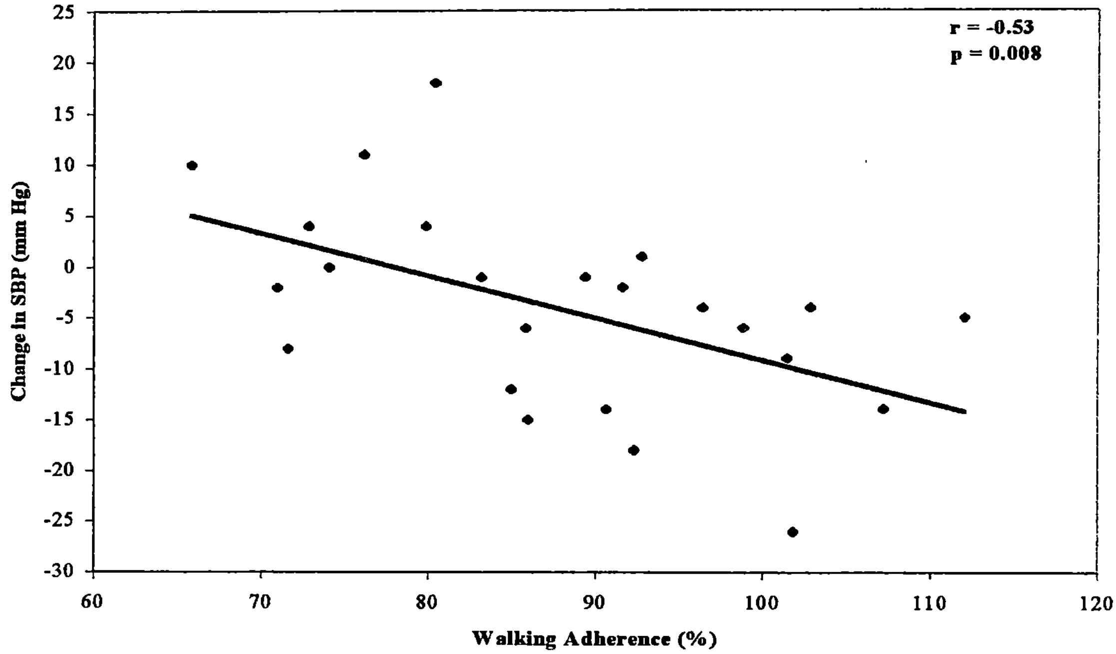


Figure 5. The relationship between baseline levels of systolic and diastolic blood pressure and the change in systolic and diastolic pressure after 12 weeks of walking.SBP, systolic blood pressure; DBP, diastolic blood pressure.



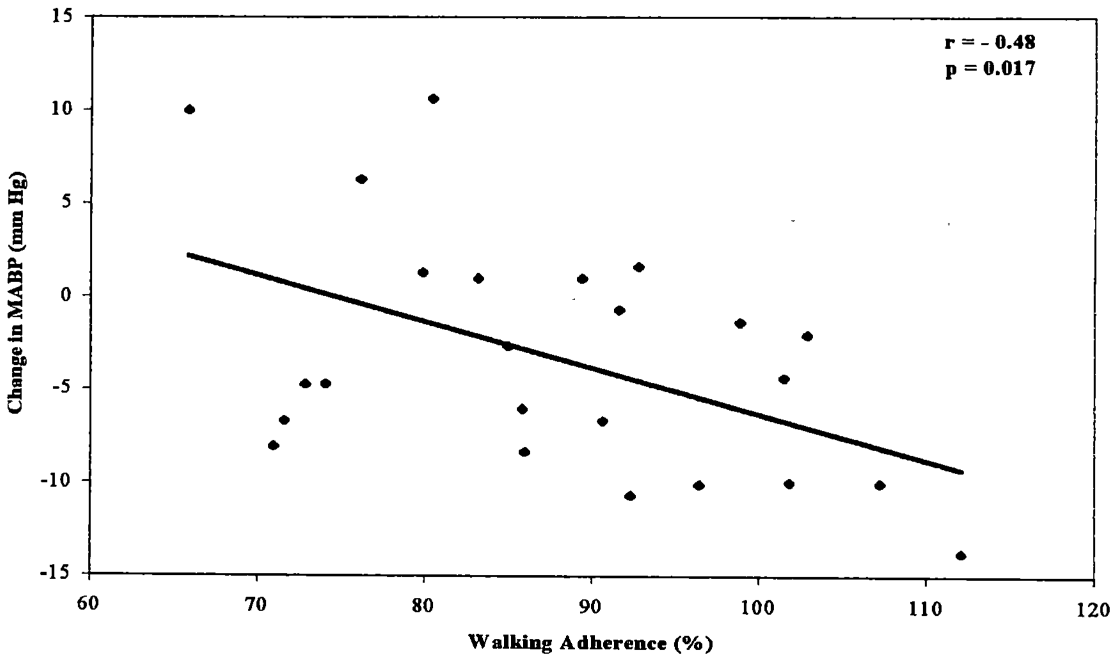


Figure 6. The relationship between adherence to the walking programs and changes in systolic and mean arterial blood pressure at the end of 12 weeks.
SBP, systolic blood pressure; MABP, mean arterial blood pressure.

Measures of Body Composition

Table 4 contains the body composition variables measured at baseline and after 12 weeks. There were no significant group × time interactions or main effects for any of the body composition variables. However, there was a trend for group × time interaction effects for body mass ($p = 0.065$) and BMI ($p = 0.054$). No significant correlations were observed between any of the variables and the change from baseline in walking distance.

Insulin and Glucose Levels

Fasting insulin, glucose, and insulin/glucose ratio (I/G) data are presented in Table 5. No significant differences between groups or changes from baseline to 12 weeks were observed in any of the variables. Correlational analyses showed significant correlations between the change in hip circumference and the change in glucose ($r = 0.69$, $p < 0.0001$) and the change in I/G ($r = -0.53$, $p = 0.01$) (Table 6) after 12 weeks of walking. Those women who had the greatest reduction in hip circumference showed the largest decreases in glucose. The change in glucose was also related to the change in BMI ($r = 0.43$, $p = 0.039$), and had a trend to decrease with a decrease in body mass ($r=0.41$, $p = 0.051$). Also, those women who had the higher glucose levels at baseline showed the greatest reduction in glucose ($r = -0.68$, $p = < 0.0001$). The change in I/G after 12 weeks of walking was also related to the baseline level of glucose ($r = 0.42$, $p = 0.046$), and demonstrated a trend to decrease with walking adherence ($r = -0.411$, $p = 0.051$).

Table 4. Body composition variables (mean ± SE).

Variable	Control N = 13	Low N = 12	High N = 12
Body Mass (kg)			
Baseline	76.9 ± 5.5	84.7 ± 5.7	75.4 ± 5.7
12 week	77.5 ± 5.4	84.0 ± 5.7	74.8 ± 5.7
BMI ($kg \cdot m^{-2}$)			
Baseline	28.0 ± 1.9	30.9 ± 1.9	28.0 ± 1.9
12 week	28.2 ± 1.8	30.6 ± 1.9	27.8 ± 1.9
Body Fat (%)			
Baseline	42.8 ± 2.0	45.2 ± 2.1	42.8 ± 2.1
12 week	43.0 ± 2.0	44.9 ± 2.0	42.3 ± 2.0
FFM (kg)			
Baseline	43.2 ± 1.9	44.7 ± 2.0	43.1 ± 2.0
12 week	43.4 ± 2.0	43.6 ± 2.1	42.7 ± 2.1
FM (kg)			
Baseline	33.7 ± 3.9	40.0 ± 4.1	32.3 ± 4.1
12 Week	34.1 ± 3.8	39.3 ± 4.0	32.1 ± 4.0
Waist (cm)			
Baseline	96.7 ± 4.6	104.8 ± 4.8	95.9 ± 4.8
12 week	99.8 ± 5.0	102.2 ± 5.2	95.6 ± 5.2
Hip (cm)			
Baseline	108.6 ± 3.1	112.9 ± 3.3	106.8 ± 3.3
12 week	108.2 ± 3.5	112.8 ± 3.6	105.1 ± 3.6
Sagittal (cm)			
Baseline	26.3 ± 1.6	27.7 ± 1.7	24.6 ± 1.7
12 week	26.4 ± 1.6	26.8 ± 1.7	24.4 ± 1.7

Control, control group; Low, low mileage group; High, high mileage group; BMI, body mass index; FFW, fat-free mass; FM, fat mass; Waist, waist circumference; Hip, hip circumference; Sagittal, sagittal diameter.

Table 5. Fasting insulin and glucose concentrations (mean ± SE).

Variable	Control N = 12	Low N = 11	High N = 12
Insulin ($\mu IU \cdot ml^{-1}$)			
Baseline	13.92 ± 1.9	15.08 ± 2.0	11.67 ± 1.9
12 week	15.51 ± 2.4	14.25 ± 2.5	10.90 ± 2.4
Glucose ($mmol \cdot L^{-1}$)			
Baseline	5.55 ± 0.29	5.51 ± 0.29	5.72 ± 0.29
12 week	5.46 ± 0.19	5.50 ± 0.19	5.57 ± 0.19
Insulin/Glucose			
Baseline	2.59 ± 0.28	2.59 ± 0.29	2.04 ± 0.28
12 week	2.82 ± 0.38	2.55 ± 0.40	1.95 ± 0.38

Control, control group; Low, low mileage group; High, high mileage group.

Table 6. Correlations between changes in fasting insulin and glucose concentrations and selected variables (values are Pearson Product Moment Correlations).

Variable	**Δ Insulin** ($\mu IU \cdot ml^{-1}$)	**Δ Glucose** ($mmol \cdot L^{-1}$)	**Δ Insulin/Glucose**
Baseline Insulin ($\mu IU \cdot ml^{-1}$)	-0.01	-0.59**	0.26
Baseline Glucose ($mmol \cdot L^{-1}$)	0.22	-0.68**	0.42*
Baseline I/G	-0.22	-0.41	0.04
Δ Body Mass (kg)	-0.18	0.41	-0.28
Δ BMI ($kg \cdot m^{-2}$)	-0.22	0.43*	-0.31
Δ Body Fat (%)	-0.16	0.08	0.17
Δ Waist (cm)	0.14	-0.24	0.12
Δ Hip (cm)	-0.31	0.69**	-0.53**
Δ Sagittal (cm)	-0.18	-0.07	-0.17
Δ Walking Distance (km/week)	-0.12	-0.09	-0.14
Total Walking Activity (km)	0.33	-0.18	0.30
Compliance (%)	-0.34	0.25	-0.41

*Significant $p < 0.05$; ** significant $p < 0.01$.
Control, control group; Low, low mileage group; High, high mileage group; I/G, insulin/glucose ratio; BMI, body mass index; Waist, waist circumference; Hip, hip circumference; Sagittal, sagittal diameter.

Responders vs. Non-responders

Figure 7 shows the individual changes in blood pressure from baseline to 12 weeks for the women in the walking groups. When testing for blood pressure changes, group × time interaction did not exist; however, 9 out of 24 women who participated in the walking program did show large decreases (i.e., a drop ≥ 10 mm Hg) in systolic and/or diastolic blood pressure. These women were therefore categorized as responders to the walking program. The characteristics of the responders and non-responders are presented in Table 7. T-tests comparison revealed no significant differences between the responders and non-responders in walking adherence, change in kilometers walked per week, or total kilometers walked for the 12 weeks. The data for resting blood pressure, heart rate, body composition, and fasting insulin and glucose concentrations for the responders and non-responders are presented in Table 8. When comparing responders and non-responders significant group × time interactions and time effects were observed for resting systolic, mean arterial, and diastolic blood pressure as well as for body mass and BMI ($p < 0.05$). Paired t-tests with Bonferroni adjustments were used to clarify the interactions within each group. The results showed systolic, mean arterial, and diastolic blood pressures, and BMI, but not body mass, to be significantly reduced in the responders. There were no significant changes in any of the variables in the non-responders. Of course, the reduction in blood pressure for the responders was a result of group selection. In responders, the changes in diastolic and mean arterial blood pressures were inversely related to baseline diastolic blood pressure ($r = -0.73$, $p = 0.027$ and $r = -0.71$,

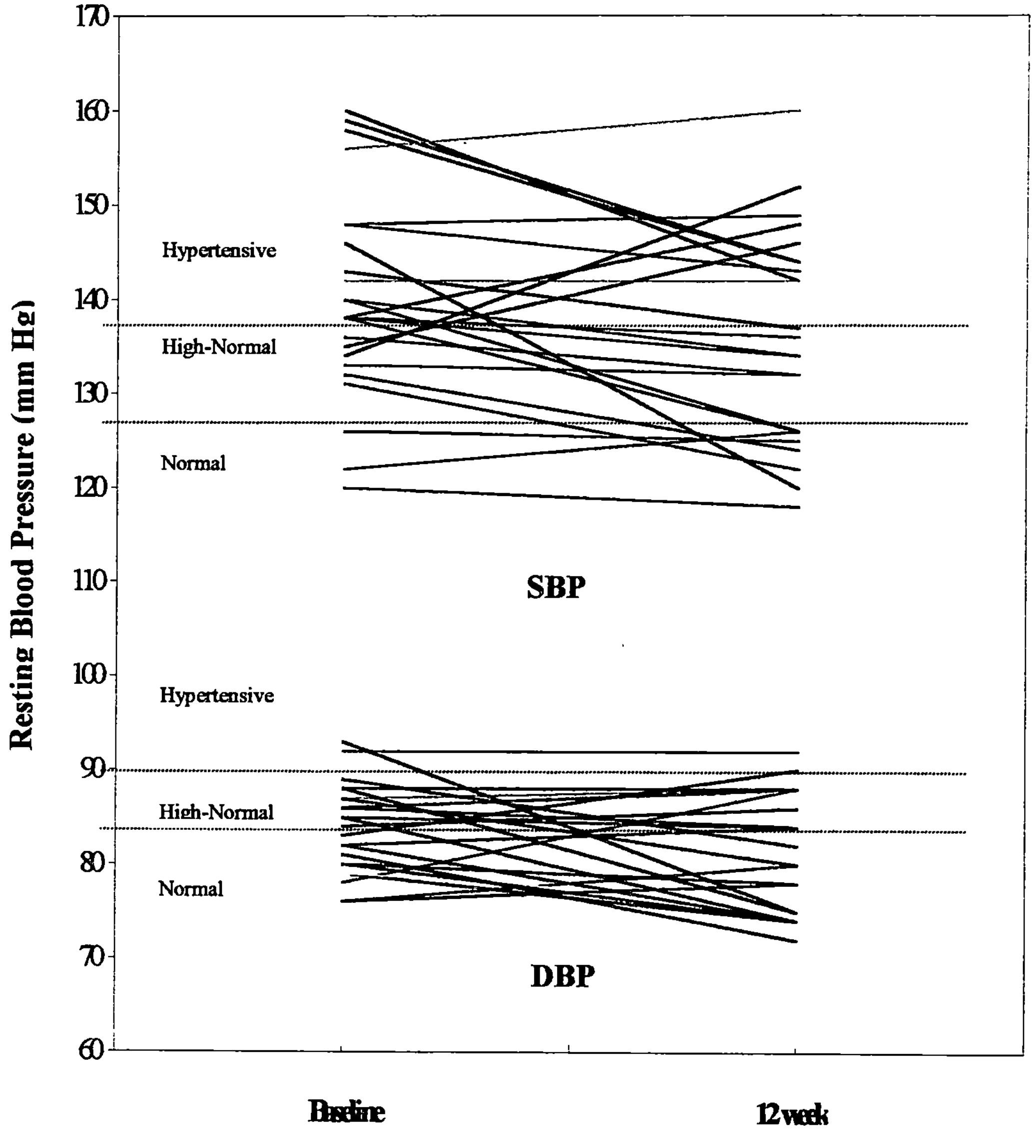


Figure 7. Individual changes in systolic (SBP) and diastolic blood pressures (DBP) from baseline to 12 weeks in women in the walking groups (N = 24).

Table 7. Characteristics of responders and non-responders (mean ± SE).

Variable	Responders (N = 9)	Non-responders (N = 15)
Compliance (%)	93.6 ± 4.2	84.4 ± 3.0
Total Distance Walked (km)	210.0 ± 18.2	200.2 ± 14.8
ΔWalking (km/wk)	18.7 ± 2.0	16.8 ± 1.4
Walking Group		
Low (N)	5	7
High (N)	4	8
HRT (N)	7	11
AntiHTN (N)	3	8

HRT, hormone replacement therapy; AntiHTN, antihypertensive medications.

Table 8. Resting blood pressure, heart rate, body composition, and fasting insulin and glucose in responders and non-responders (mean ± SE).

Variable	Responder (N = 9) Baseline	Responder 12 week	Responder Δ	Non-Responder (N = 15) Baseline	Non-Responder 12 week	Non-Responder Δ
SBP (mm Hg)*	145.2 ± 3.4	133.0 ± 3.7	-12.2 ± 2.5†	136.9 ± 2.7	137.7 ± 2.9	0.7 ± 7.6
DBP (mm Hg)*	84.6 ± 1.6	77.3 ± 1.9	-7.2 ± 6.1†	83.0 ± 1.2	82.8 ± 1.5	-0.2 ± 5.2
MAP (mm Hg)*	104.6 ± 1.7	95.7 ± 2.1	-8.9 ± 3.1†	100.8 ± 1.3	100.9 ± 1.6	0.1 ± 5.4
Heart Rate (bpm)	74.4 ± 3.2	69.8 ± 2.5	-4.7 ± 3.2	76.5 ± 2.5	72.3 ± 1.9	-4.3 ± 1.7
Body Mass (kg)*	80.0 ± 6.9	78.5 ± 6.8	-1.5 ± 1.6†	80.1 ± 5.4	79.9 ± 5.3	-0.1 ± 1.4
BMI ($kg \cdot m^{-2}$)*	28.8 ± 2.2	28.3 ± 2.2	-0.5 ± 0.6†	29.8 ± 1.7	29.8 ± 1.7	-0.08 ± 0.5
Body Fat (%)	40.9 ± 2.6	39.9 ± 2.4	-1.9 ± 4.1	45.8 ± 2.0	45.9 ± 1.9	-0.06 ± 1.1
Fasting Insulin ($\mu IU \cdot ml^{-1}$)	10.71 ± 2.56	10.43 ± 2.86	-0.28 ±	14.68 ± 1.87	13.61 ± 2.09	-1.06 ±
Fasting Glucose ($mmol \cdot L^{-1}$)	5.27 ± 0.34	5.38 ± 0.26	0.10 ±	5.80 ± 0.25	5.60 ± 0.19	-0.21
Insulin/Glucose	2.01 ± 0.32	1.94 ± 0.43	-0.07	2.46 ± 0.23	2.39 ± 0.32	-0.07

*Significant group × time effect, $p < 0.05$; † Significant difference between responders and non-responders, $p < 0.05$.
SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; BMI, body mass index.

p = 0.033, respectively). There were no group × time interaction effects or differences between responders and non-responders in changes in heart rate, fasting insulin, fasting glucose, I/G, body fat, waist and hip circumferences, or sagittal diameter. However, a significant time effect was observed for heart rate ($p < 0.05$), with heart rate being lower after 12 weeks in comparison to baseline in both responders and non-responders. Also, the change in waist circumference in the responders was significantly related to the change in kilometers walked per week ($r = -0.67$, $p = 0.047$), indicating those who walked more showed the greatest reduction in waist circumference.

Submaximal Heart Rate, Oxygen Consumption, RER, and RPE

Heart rate, $\dot{V}O_2$, RER, and RPE responses during submaximal graded exercise are shown in Table 9. The results of a three-way ANOVA (group × time × trial) showed no significant interaction effects for heart rate, $\dot{V}O_2$, RER, or RPE during the graded submaximal exercise test. A significant group × trial interaction was observed for RER ($p < 0.05$). Further analyses showed that the mean RER value from baseline was decreased in the walking groups after 12 weeks. As expected, all three variables increased with each submaximal stage in all groups ($p < 0.05$). A significant trial effect was also observed for heart rate ($p < 0.05$). The mean heart rate for all groups was lower after 12 weeks in comparison to baseline.

Table 9. Heart rate, oxygen consumption, and RPE during submaximal graded exercise (mean ± SE).

Variable	Control		Low		High	
	Baseline	12 week	Baseline	12 week	Baseline	12 week
Heart Rate (bpm)						
Stage I	104.8 ± 4.4	98.3 ± 4.4	109.4 ± 4.4	100.0 ± 4.4	110.3 ± 5.1	105.8 ± 5.1
Stage II	111.5 ± 4.7	105.3 ± 4.2	116.0 ± 4.7	107.3 ± 4.7	118.6 ± 5.4	112.7 ± 4.9
Stage III	121.0 ± 5.2	115.9 ± 4.7	124.8 ± 5.2	115.9 ± 4.7	129.9 ± 6.1	122.2 ± 5.4
$\dot{V}O_{2\,max}$ ($L \cdot min^{-1}$)						
Stage I	0.85 ± 0.07	0.78 ± 0.06	0.95 ± 0.07	0.89 ± 0.07	0.89 ± 0.08	0.95 ± 0.07
Stage II	0.98 ± 0.07	0.95 ± 0.07	1.09 ± 0.08	1.10 ± 0.08	1.10 ± 0.08	1.08 ± 0.08
Stage III	1.14 ± 0.08	1.10 ± 0.08	1.24 ± 0.09	1.20 ± 0.09	1.30 ± 0.09	1.32 ± 0.09
RER*						
Stage I	0.79 ± 0.02	0.78 ± 0.01	0.83 ± 0.02	0.76 ± 0.01†	0.81 ± 0.02	0.78 ± 0.01†
Stage II	0.82 ± 0.02	0.82 ± 0.01	0.86 ± 0.02	0.81 ± 0.01†	0.86 ± 0.02	0.82 ± 0.02†
Stage III	0.87 ± 0.02	0.88 ± 0.02	0.91 ± 0.02	0.86 ± 0.02†	0.92 ± 0.02	0.87 ± 0.02†
RPE						
Stage I	10.2 ± 0.4	10.0 ± 0.5	9.8 ± 0.4	9.8 ± 0.5	10.2 ± 0.4	9.6 ± 0.6
Stage II	11.2 ± 0.4	10.9 ± 0.5	11.8 ± 0.4	10.8 ± 0.5	11.7 ± 0.4	10.6 ± 0.6
Stage III	12.2 ± 0.3	12.3 ± 0.5	12.4 ± 0.5	12.4 ± 0.5	13.1 ± 0.4	11.9 ± 0.6

VO_2, oxygen consumption; RER, respiratory exchange ratio; RPE, rating of perceived exertion.
* Significant trial × group interaction, $p < 0.05$; † Significant difference baseline vs 12 weeks, $p < 0.05$.

Submaximal Blood Pressure

No significant group × time × trial interactions were observed for systolic, diastolic, or mean arterial blood pressure. However, significant time and trial main effects were found for systolic and mean arterial blood pressure ($p < 0.05$). The mean systolic and mean arterial blood pressures increased with each stage, and were lower after 12 weeks in comparison to baseline (Table 10). Significant time ($p < 0.05$) and trial ($p < 0.05$) effects were also observed for diastolic blood pressure, with the mean diastolic blood pressure decreasing with exercise stage, and after 12 weeks.

Dietary Intake and Composition

The food intake and dietary composition at baseline and after 12 weeks for all three groups are presented in Table 11. There were no significant differences between groups at baseline or after 12 weeks in total calories, carbohydrate, fat, protein, potassium, magnesium, or alcohol intake. The low group had a significantly higher sodium intake in comparison to the control and high groups at baseline ($p < 0.05$). Significant time × group interaction and time effects were observed for sodium intake ($p < 0.05$). Paired t-tests revealed that the high group significantly increased their sodium intake, but no significant differences were observed for the low and control groups. In each of the 3 groups, there were subjects who failed to complete 3-day dietary records. Therefore, caution should be used when interpreting these data.

Table 10. Systolic, mean arterial, and diastolic blood pressure during submaximal graded exercise (mean ± SE).

Variable	Control		Low		High	
	Baseline	12 week	Baseline	12 week	Baseline	12 week
SBP (mm Hg)						
Stage I	160.8 ± 4.8	155.5 ± 4.2	171.3 ± 5.0	157.7 ± 4.4	168.6 ± 5.5	162.0 ± 4.8
Stage II	170.8 ± 4.9	165.4 ± 4.5	184.0 ± 5.1	166.0 ± 4.6	181.4 ± 5.6	172.0 ± 5.1
Stage III	182.3 ± 5.1	174.3 ± 4.4	193.2 ± 5.3	180.3 ± 4.6	190.8 ± 5.8	180.4 ± 5.0
MABP (mm Hg)						
Stage I	113.9 ± 2.1	108.6 ± 2.2	116.8 ± 2.2	109.9 ± 2.3	118.8 ± 2.4	110.9 ± 2.5
Stage II	115.7 ± 2.3	111.6 ± 2.3	120.6 ± 2.4	112.3 ± 2.4	121.2 ± 2.6	114.2 ± 2.6
Stage III	119.5 ± 2.3	114.1 ± 2.2	122.4 ± 2.4	116.0 ± 2.3	124.6 ± 2.6	115.0 ± 2.5
DBP (mm Hg)						
Stage I	90.8 ± 1.7	85.5 ± 1.9	90.0 ± 1.8	86.3 ± 2.0	94.2 ± 1.9	85.8 ± 2.1
Stage II	88.6 ± 1.9	85.1 ± 1.8	89.3 ± 1.9	85.8 ± 1.9	91.6 ± 2.1	85.8 ± 2.1
Stage III	88.6 ± 2.0	84.5 ± 2.0	87.5 ± 2.1	84.3 ± 2.0	92.0 ± 2.3	82.8 ± 2.2

SBP, systolic blood pressure; MABP, mean arterial blood pressure; DBP, diastolic blood pressure.

Table 11. Dietary intake (mean ± SE).

Variable	Control N = 9	Low N = 10	High N = 11
Total Kilocalories			
Baseline	1786 ± 178	1873 ± 161	1774 ± 161
12 week	1888 ± 198	1698 ± 179	2041 ± 179
Carbohydrate (%)			
Baseline	58.3 ± 2.7	56.7 ± 2.4	54.2 ± 2.4
12 week	58.0 ± 3.6	58.0 ± 3.3	51.9 ± 3.3
Fat (%)			
Baseline	29.5 ± 2.0	29.3 ± 1.8	30.8 ± 1.8
12 week	29.5 ± 2.9	28.6 ± 2.7	32.5 ± 2.7
Protein (%)			
Baseline	13.9 ± 1.3	15.2 ± 1.2	15.2 ± 1.2
12 week	14.1 ± 1.6	16.2 ± 1.4	16.7 ± 1.4
Sodium (mg)*			
Baseline	2368 ± 220	3111 ± 199	2398 ± 199
12 week	3279 ± 398	2575 ± 360	3176 ± 360†
Potassium (mg)			
Baseline	2191 ± 626	3472 ± 566	2439 ± 566
12 week	2497 ± 274	2655 ± 248	3029 ± 248
Magnesium (mg)			
Baseline	286 ± 63	329 ± 57	293 ± 57
12 week	311 ± 36	286 ± 32	280 ± 32
Calcium (mg)			
Baseline	612 ± 69	780 ± 63	638 ± 63
12 week	653 ± 87	674 ± 78	775 ± 78
Alcohol (g)			
Baseline	0	1.3 ± 1.8	4.4 ± 1.8
12 week	0	3.4 ± 1.8	2.6 ± 1.8

Control, control group; Low, low mileage group, High, high mileage group.
* Significant group × time interaction, $p < 0.05$; † Significant difference between baseline and 12 weeks, $p < 0.05$.

Psychological Factors

Table 12 contains the mean scores for the measures of depression, hostility, and aggression. There were no significant group differences in any of the variables at baseline. The women in all the groups demonstrated low scores of depression, hostility, and aggression. Hostility scores were shown to be significantly related to resting systolic blood pressure ($r = -0.39$, $p < 0.027$), indicating that women who scored higher on the hostility scale demonstrated a lower resting systolic blood pressures. No significant group × time interaction effects were found for scores of depression, hostility or aggression. A significant time effect was observed for hostility, showing the mean hostility score for all the groups to be higher at 12 weeks in comparison to baseline ($p < 0.05$). No significant correlations were observed between the change in depression, hostility, and aggression scores from baseline to 12 weeks and the change in blood pressure or walking activity.

There were no significant interactions or main effects observed in measures of self perception (Table 13). At the end of 12 weeks, there were no changes in how the women in the control, low and high walking groups rated themselves in sports competence, physical condition, body attractiveness, strength, and overall physical self- worth. There was also no difference in how they perceived the importance of each of the above factors in assessing their self-worth. Note that in both Table 12 and 13 the subject number is reduced. This is due to the fact that some subjects did not provide answers or provided answers that could not be used in the analyses.

Table 12. Depression, hostility, and aggression scores (mean ± SE).

Variable	Control	Low	High
Depression	N = 10	N = 10	N = 12
Baseline	14.4 ± 1.4	14.8 ± 1.4	15.6 ± 1.1
12 week	15.2 ± 1.1	14.7 ± 1.1	15.0 ± 1.0
Hostility	N =10	N = 8	N = 9
Baseline	1.3 ± 0.3	1.0 ± 0.4	1.3 ± 0.3
12 week	2.1 ± 0.4	1.3 ± 0.5	1.4 ± 0.5
Aggression	N = 11	N = 8	N = 9
Baseline	2.4 ± 0.5	2.4 ± 0.6	2.3 ± 0.6
12 week	2.5 ± 0.5	2.4 ± 0.6	2.7 ± 0.6

Control, control group; Low, low mileage group; High, high mileage group.

Table 13. PSPP and PIP items and subscales (mean ± SE).

Variable	Control N = 10		Low N = 11		High N = 9	
	Baseline	12 Week	Baseline	12 Week	Baseline	12 Week
PSPP						
SPORT	10.6 ± 1.3	10.7 ± 1.5	9.8 ± 1.2	9.5 ± 1.4	12.1 ± 1.3	11.9 ± 1.6
CONDITION	11.5 ± 0.9	11.4 ± 1.3	10.4 ± 0.8	11.7 ± 1.3	12.6 ± 0.9	13.7 ± 1.4
BODY	12.3 ± 1.1	10.9 ± 1.5	10.6 ± 1.0	11.0 ± 1.4	11.7 ± 1.2	11.9 ± 1.6
STRENGTH	13.8 ± 0.7	13.0 ± 1.1	13.1 ± 0.7	13.1 ± 1.1	14.7 ± 0.8	14.1 ± 1.2
PSW	13.3 ± 1.1	12.3 ± 1.6	11.8 ± 1.1	12.3 ± 1.5	13.6 ± 1.2	14.3 ± 1.7
PIP						
SPORTIMP	3.5 ± 0.4	3.5 ± 0.5	3.3 ± 0.4	3.1 ± 0.5	4.4 ± 0.4	4.3 ± 0.5
CONDITONIMP	5.2 ± 0.5	5.3 ± 0.4	4.9 ± 0.5	4.9 ± 0.4	5.8 ± 0.5	5.9 ± 0.4
BODYIMP	4.4 ± 0.4	5.1 ± 0.4	4.2 ± 0.4	4.5 ± 0.3	5.1 ± 0.4	5.7 ± 0.4
STRENGTHIMP	5.0 ± 0.4	4.7 ± 0.4	4.4 ± 0.4	4.3 ± 0.4	5.7 ± 0.4	5.0 ± 0.4

PSPP, Physical Self Perception Profile; PIP, Perceived Importance Profile; SPORT, sports competence; CONDITION, physical condition; BODY, body attractiveness; STRENGTH, physical strength; PSW, physical self worth. PIP provides a measure of importance attached to four of the PSPP subscales.

CHAPTER V

DISCUSSION

Impact of Walking on Resting Blood Pressure

The current ACSM-CDC minimum physical activity recommendations for the attainment of health benefits suggest that all US adults should strive to accumulate 30 minutes of moderate intensity physical activity on most, and preferably all, days of the week (166). Few studies have examined the effectiveness of these recommendations in lowering blood pressure, particularly in postmenopausal women with borderline to mild hypertension. The present study examined this question by prescribing a 12 week walking program that met the minimum physical activity recommendations (16 kilometers/week) to sedentary, hypertensive, postmenopausal women. A second aim of the study was to determine whether women walking more than the current recommendations would achieve a greater benefit. The findings from the present investigation showed that 12 weeks of walking activity meeting or exceeding the ACSM-CDC minimum physical activity recommendations was ineffective in lowering blood pressure in hypertensive postmenopausal women. Although the women walkers experienced small statistically non- significant reductions in resting systolic and diastolic blood pressures of 5/3 and 3/4 mm Hg for the low and high groups, respectively, these reductions translate to a decrease in coronary heart disease risk of 14% and a decrease in stoke risk of 42% (31). These reductions were smaller than the average reduction of 6-9 mm Hg reported for both systolic and diastolic blood pressures in previous studies (8, 185).

The finding of a non-significant decrease in blood pressure is similar to others who have investigated the effectiveness of the ACSM-CDC physical activity recommendations (153, 171). Murphy et al. (153) reported no significant reductions in systolic blood pressure with either one continuous bout of 30 minutes of walking, or three, 10 minute bouts of walking, 5 days per week. Although the mean decreases in systolic blood pressures were 5.4 and 2.6 mm Hg for the short and the long bout walkers, respectively, this change was no different from what was observed in the controls. It is possible that the walking volume used by Murphy et al. (153) was an inadequate stimulus for lowering blood pressure. Although their purpose was to test the effectiveness of the ACSM-CDC physical activity recommendations (153), the amount of walking that their participants performed (~12 kilometers/week) was less than the minimum recommendation of ~ 16 kilometers per week. Also, the participants in their study were normotensive, and therefore, were less likely to see significant changes in blood pressure.

Ready et al. (171) examined the issue of dose-response by varying the frequency of walking. Postmenopausal women walked 57 minutes a day, either 3 or 5 days/week. The amount of walking the women did was equivalent to 16.5 and 26.3 kilometers/week, respectively, which is comparable to the amount of walking that the women did in the present study. Systolic and blood pressures were reduced, but not significantly, by 7 and 5 mm Hg (3 day and 5 day, respectively), findings comparable with Murphy et al.'s study (153) and the present study. The authors hypothesized that the lack of a blood pressure lowering effect could be attributed to a decline in other daily activities. The present study attempted to avoid this potential problem by recording baseline activity, adding walking to this baseline, and documenting distance walked daily. Using this approach, women reported completing, on average, 88.4% and 87.3% of the prescribed walking distance for the low and high goups, respectively. The subjects also completed physical activity questionnaires which were used to screen for non-walking changes in physical activity. Although there were documented increases in daily walking (~13 kilometers and 22 kilometers, for the low and high groups, respectively), no significant change in blood pressure was observed.

Is it possible that allowing subjects to accumulate walking throughout the day, rather than prescribing a set exercise duration and intensity, led to the lack of significant blood pressure reduction? The ACSM-CDC recommendations contend that physical activity can be *accumulated* throughout the day, and thus, the approach adopted in the present study allowed the women to accumulate their walking activity instead of performing it in one continuous bout. Dunn et al. (53) reported significant reductions in systolic and diastolic blood pressures with this type of "lifestyle intervention". In the Dunn et al. (53) study, the mean decreases in systolic and diastolic blood pressures (4 and 5 mm Hg, respectively) were similar to the reductions observed in the previous studies (153, 171) and the present study. The significance of their findings is due in part to their large sample size (N = 121). In the current study, although blood pressure was not reduced, a significant shift in RER occurred in both walking groups during the submaximal exercise bouts. This demonstrates that the walking was sufficient to shift substrate utilization toward a greater reliance on fats, which is an important fitness adaptation.

Although a fitness adaptation was experienced by the women in the walking groups, it is possible that the exercise intensity was too low to elicit a reduction in blood pressure. The literature is contradictory on this point. A number of studies have shown walking to be ineffective in altering blood pressure (52, 74, 133, 183). Santiago et al. (183) reported no significant reductions in blood pressure in women who walked at a moderate intensity (71% HR_{max}) 55 minutes a day, 4 days a week. Walking 20 kilometers/week (55% HR_{max}) in addition to climbing 50 floors of stairs/week (82% HR_{max}) proved to be an insufficient stimulus to lower blood pressure even though the caloric expenditure was ~ 2000 kcal/week (133). Duncan et al. (52) found no blood pressure lowering effect with low, moderate or high intensity walking programs. On the other hand, several studies have shown walking at moderate intensities to be beneficial in reducing blood pressure (82, 97, 152, 186, 210, 224). Seals et al. (186) reported significant reductions in systolic and diastolic blood pressures of 10 and 7 mm Hg in postmenopausal women who walked 45 minutes a day, 3 days per week. Similarly, Tanaka et al. (210) reported systolic and diastolic blood pressure decreases of 7/5 mm Hg with walking at an intensity of 45% of heart rate reserve for 45 minutes, 3 days per week in men and women with stage 1 and 2 hypertension.

The walking program in the present study was 12 weeks in duration. Thus, it can be questioned whether 12 weeks is long enough to see a significant reduction in blood pressure. Short-term intervention studies may not show the same kind of response as seen with a lifetime of regular physical activity. However, based upon the literature, significant reductions in blood pressure can be observed with short-term physical activity programs. The average study duration for blood pressure lowering effects is 10-12 weeks (28, 97, 121, 122, 186, 206, 217); however, blood pressure has been shown to be lowered with physical activity programs as little as 4 weeks in duration (105). Jennings et al. (105), using a cross-over design, reported significant reductions in systolic and diastolic blood pressure of 10 and 7 mm Hg, respectively, with 40 minutes of cycling, three days per week. No further benefit was observed with cycling 7 days per week as blood pressure was decreased by 12 and 7 mm Hg (systolic and diastolic, respectively). Kiyonaga et al. (122) reported reductions in systolic and diastolic blood pressures of more than 20 and 10 mm Hg, respectiviely, in 50% of their participants after 10 weeks of cycling for 60 minutes a day, 3 days per week. After 20 weeks, 78% of their study's participants saw drops of 20/10 mm Hg in systolic/diastolic blood pressure.

The discrepancies in the aforementioned studies can partially be attributed to the differences in subject populations. Many studies showing no significant reductions in blood pressure with walking used normotensive individuals as participants (52, 133, 153, 171, 183), while those demonstrating a blood pressure lowering effect used hypertensive individuals (82, 97, 152, 186, 210, 224). In the present study, postmenopausal women with borderline to mild hypertension served as subjects. Therefore, it was anticipated that these women would experience a blood pressure reduction. Although, no significant group × time interaction was observed, there was a significant correlation between baseline blood pressure and change in blood pressure with walking. Specifically, the women who had higher baseline resting blood pressure experienced the greatest reduction in blood pressure with walking. This finding is commonly reported (8).

Another potential confounding variable in the present study was the inclusion of women who were taking antihypertensive medications (5 in the control, 4 in the low, and 7 in the high). The rationale for their inclusion comes from the work of Cade et al. (28) who reported similar reductions in blood pressure in those taking antihypertensive medications in comparison to those who were not taking medications. To control for any medication effect, the women in the present study took their medications at the same time of day at each testing measurement. Additionally, medications remained stable throughout the 12 week intervention. Statistical analyses revealed that the blood pressure responses of the medicated subjects were not significantly different to that of the non-medicated subjects.

Maintaining compliance and avoiding dropouts is a major concern with intervention studies. An obvious result of low subject numbers is a drop in statistical power. For example, the statistical power calculated for the two-way ANOVA used to test for a significant group × time interaction for blood pressure was only 0.128. Preliminary calculations for this study demonstrated a need for 22 participants per group. Although this goal was attempted, exclusion criteria and dropouts depleted the final number of subjects to 37.

Factors Potentially Interacting with Blood Pressure

Other factors known to influence blood pressure such as body composition and diet were assessed in order to explain any masking effect of the walking on blood pressure. The dietary recall revealed no change in total calories or macronutrient distribution over the course of the study, however, an increase in sodium intake was observed. It should be noted that the amount of walking performed by the subjects was insufficient to cause a large reduction in body mass. The women in the low group walked a total of 157 kilometers above their baseline walking activity across 12 weeks, expending ~ 8000 kilocalories, while women in the high group walked a total of 250 kilometers above baseline, equivalent to expending 12,800 kilocalories. Since there were no significant differences in reported food intake, one would calculate that the women in the low and high groups should have lost ~ 1 and 2 kg, respectively. These values are slightly higher than the 0.7 kg lost in the low and the 0.6 kg lost in the high group. The differences between the estimated and actual amount of weight lost could possibly be due to inaccurate reporting of dietary intake by the women.

In the present study, significant correlations were shown between the changes in systolic and mean arterial blood pressures and the change in body mass ($r = 0.46$ and $r = 0.42$, respectively). Those women who lost weight had a tendency to have the greatest reductions in systolic and mean arterial blood pressures. Thus, the lack of a group × time interaction for blood pressure could be attributed to the lack of a significant group × time interaction for body mass or body fat. Stepwise multiple repression analyses revealed that for systolic blood and mean arterial blood pressure the only factor useful in explaining blood pressure change was change in body mass. No other factors significantly improved the prediction of systolic or mean arterial blood pressure changes.

Although we observed a relationship between the change in systolic and mean arterial blood pressures and the change in body mass, other studies have shown blood pressure to be lowered even though body mass and body composition remained unchanged (28, 97, 105, 121, 206, 207, 217). Cade et al. (28) noted that the decrease in blood pressure observed in their study was similar between those who lost weight and those who increased their weight. Su et al. (201) observed a significant reduction blood pressure along with a reduction in body weight. However, correlational analyses showed no significant relationship between weight loss and blood pressure reduction, thus, they suggested that the reduction in blood pressure observed with weight loss may be secondary to the improvement in insulin sensitivity. Dengel et al. (46) reported a 39% increase in insulin sensitivity and a decrease in systolic and diastolic blood pressures by 14 and 10 mm Hg, respectively, with aerobic exercise and weight loss. In the present study no significant reductions in fasting insulin, glucose, or I/G occurred with the walking programs. Thus, it is possible that the lack of impact on blood pressure was linked to no significant change in weight and/or measures of insulin sensitivity.

The findings of no improvement in glucose, insulin, and I/G are in contrast to those studies which have shown an improvement in insulin sensitivity with walking (135, 229). Yamanouchi et al. (229) reported an increase in insulin sensitivity in NIDDM patients who walked 19,200 steps/day (~ 13 kilometers/day) as measured with a pedometer. The women in the present study walked ~ 6.7 (high group) and 5.5 (low group) kilometers/day. Therefore, it may be that the walking programs did not provide a sufficient stimulus to cause improvements in insulin or glucose levels. However, it must be noted that the women in the present study had normal baseline levels of glucose and insulin, while the participants in the study by Yamanouchi et al. (229) were NIDDM patients. The women in the present study who had higher levels of glucose and insulin at baseline had the greatest reduction in glucose levels after 12 weeks. Also, the participants in the previous study (229) were also on a caloric restricted diet. It is also possible that changes in insulin sensitivity may have occurred, but were undetectable with our measure of insulin sensitivity (I/G). A better but more costly and time-consuming measure of insulin sensitivity is a oral glucose tolerance test.

Psychological variables previously reported to be associated with the development of hypertension were also assessed. Moderate and high intensity physical activity programs have been shown to be beneficial in improving self-esteem and lowering symptoms of depression, hostility, and aggression (18, 25, 51, 120, 141, 165). In the present study, there were no changes in scores of hostility, aggression or depression after 12 weeks of walking. We also found no significant alterations in sub-scale scores of self-esteem. These findings are similar to other studies which have reported physical activity to have no benefit on measures of depression, hostility, and anxiety (41, 165, 167). The lack of a beneficial effect in the above mentioned studies could possibly be due to the fact that the participants had normal psychological functioning prior to beginning the physical activity intervention (41, 165, 167). The normal scores at baseline in the present study's participants may also have influenced the lack of change.

Responders versus Non-responders

Although walkers, in general, did not experience a lowering of blood pressure, 5 women did normalize their blood pressure after 12 weeks of walking, and several women responded to the walking programs by having reductions in systolic or diastolic blood pressures $\geq$ 10 mm Hg. Thus, an additional purpose of this study was to identify and compare characteristics associated with women who responded favorably to the walking programs to those who did not respond. Responders were defined as those women who decreased either systolic or diastolic blood pressures by 10 mm Hg or more. Five women from the low group and 4 from the high group met these criteria. As expected, the responders to the walking program had significantly greater reductions in systolic, mean arterial, and diastolic blood pressures than non-responders (12, 9, and 7 vs 0.7, 0.1, and 0.2, respectively) (Table 8). In the present study, adherence to the walking programs correlated significantly with changes in systolic and mean arterial blood pressures, meaning that those who best met their walking goals experienced the greatest decreases in systolic and mean arterial blood pressures. As was observed for all of the walkers, those responders with the highest baseline values saw the greatest reduction in diastolic and mean arterial blood pressures. Additionally, the responders lost more body mass and lowered their BMI after the walking program in comparison to non-responders. This suggests that in this group of postmenopausal hypertensive women the loss of body mass was linked with lowering blood pressure.

Conclusion

In conclusion, the present study found that 12 weeks of meeting or exceeding the ACSM-CDC minimum physical activity recommendations through walking was ineffective in lowering blood pressure in postmenopausal women with borderline to mild hypertension. The modest reduction in blood pressure observed in the walking groups was not significantly different from changes in the control group. It was demonstrated, however, that among walkers those who adhered most closely to their prescribed walking program experienced the largest reduction in systolic and mean arterial blood pressure. The overall lack of a blood pressure lowering effect with walking could possibly be attributed to non-significant changes in body composition, insulin levels, I/G, or psychological variables frequently linked with hypertension. We found that adherence to the walking program was associated with the greatest reduction in systolic and mean arterial blood pressures in all walkers.

Even though there were no statistically significant group $\times$ time interactions for blood pressure, some women responded favorably to the program by either normalizing their blood pressure or by decreasing their blood pressure from hypertensive to borderline levels. Those women who responded to the program were characterized by having better adherence to the walking program, higher baseline levels of blood pressure, and greater reductions in body mass.

The effectiveness of ACSM-CDC recommendations needs further study to determine if accumulated amounts of moderate intensity physical activity can be beneficial in lowering blood pressure. The continuation of this study for an additional 12 weeks will indicate if 24 weeks is more effective for lowering blood pressure. Other studies which document exercise intensity, as well as exercise volume are critical.

LIST OF REFERENCES

1. American Society of Hypertension Public Policy Position Paper: Recommendations for routine blood pressure measurement by indirect cuff sphygmomanomety. *American Journal of Hypertension* 5: 207-209, 1992.

2. ACSM. Physical activity, physical fitness, and hypertension. *Medicine and Science in Sports and Exercise* 25 (10): i-x, 1993.

3. Andersen LB, Haraldsdottir J. Coronary heart disease risk factors, physical activity, and fitness in young Danes. *Medicine and Science in Sports and Exercise* 27 (2): 158-163, 1995.

4. Anderson EA, Hoffman RP, Balon TW, Sinkey CA, Mark AL. Hyperinsulinema produces both sympathetic neural activation and vasodilation in normal humans. *Journal of Clinical Investigation* 87: 2246-2252, 1991.

5. Anderson KM, Wilson PWF, Odell PM, Kannel WB. An updated coronary risk profile: a statement for health professionals. *Circulation* 83 (1): 356-362, 1991.

6. Andersson I, Rossner S. Energy intake of obese women. *International Journal of Obesity* 13: 247-253, 1989.

7. Arakawa K. Hypertension and exercise. *Clinical and Experimental Hypertension* 15 (6): 1171-1179, 1993.

8. Arroll B, Beaglehole R. Does physical activity lower blood pressure: a critical review of the clinical trials. *Journal of Clinical Epidemiology* 45 (5): 439-447, 1992.

9. Arroyo P, Fernandez V, Avila-Rosas H. Overweight and hypertension: Data from the 1992-1993 Mexican survey. *Hypertension* 30 (part 2): 646-649, 1997.

10. Avolio AP, Fa- Quan D, Wei-Qiang L, Yao-Fei L, Zhen-Dong H, Lian-Fen X, O'Rourke MD. Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. *Circulation* 71 (2): 202-210, 1985.

11. Ballor DL, Keesey RE. A meta-analysis of the factors affecting exercise-induced changes in body mass, fat mass and fat-free mass in males and females. *International Journal of Obesity* 15: 717-726, 1991.

12. Ballor DL, McCarthy JP, Wilterdink EJ. Exercise intensity does not affect the composition of diet- and exercise-induced body mass loss. *American Journal of Clinical Nutrition* 51: 142-146, 1990.

13. Barefoot JC, Dodge KA, Peterson BL, Dahlstrom G, Williams RB. The Cook-Medley hostility scale: item content and ability to predict survival. *Psychosomatic Medicine* 5: 46-57, 1989.

14. Barefoot JC, Larsen S, von der Lieth L, Schroll M. Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women. *American Journal of Epidemiology* 142 (5): 477-484, 1995.

15. Barefoot JC, Schroll M. Symptoms of depression, acute myocardial infarction, and total mortality in a community sample. *Circulation* 93: 1976-1980, 1996.

16. Bassett DR, Leggett SR, Mathien CA, Main JA, Hunter DC, Duncan GE, Ainsworth BE. Accuracy of five electronic pedometers for measuring distance walked. *Medicine and Science in Sports and Exercise* 9: 1099-1103, 1996.

17. Benotsch EG, Christensen AJ, McKelvey L. Hostility, social support, and ambulatory cardiovascular activity. *Journal of Behavioral Medicine* 20 (2): 163-176, 1997.

18. Blumenthal JA, Williams RS, Needels TL, Wallace AG. Psychological changes accompany aerobic exercise in healthy middle-aged adults. *Psychosomatic Medicine* 44 (6): 529-536, 1982.

19. Borg GAV. Physical Performance and Perceived Exertion. Lund, Sweden: Gleerup, 1962.

20. Bovens AM, Van Baak MA, Vrencken JG, Wijnen JA, Saris WH, Verstappen FT. Physical activity, fitness, and selected risk factors for CHD in active men and women. *Medicine and Science in Sports and Exercise* 25 (5): 572-576, 1993.

21. Brands MW, Hildebrandt DA, Mizelle HL, Hall JE. Hypertension during chronic hyperinsulinemia in rats is not salt-sensitive. *Hypertension* 19: I83-I89, 1992.

22. Brands MW, Hildenbrandt DA, Mizelle HL, Hall JE. Sustained hyperinsulinemia increases arterial pressure in conscious rats. *American Journal of Applied Physiology* 260: R764-R768, 1991.

23. Brands MW, Mizelle HL, Gaillard CA, Hildenbrandt DA, Hall JE. The hemodynamic response to chronic hyperinsulinemia in conscious dogs. *American Journal of Hypertention* 4: 164-168, 1991.

24. Braun B, Zimmermann MB, Kretchmer N. Effects of exercise intensity on insulin sensitvity in women with non-insulin-dependent diabetes mellitus. *Journal of Applied Physiology* 78 (1): 300-306, 1995.

25. Brown DR, Wang Y, Ward A, Ebbeling CB, Fortlage L, Puleo E, Benson H, Rippe JM. Chronic psychological effects of exercise and exercise plus cognitive strategies. *Medicine and Science in Sports and Exercise* 27 (5): 765-775, 1995.

26. Bryner RW, Toffle RC, Ullrich IH, Yeater RA. The effects of exercise intensity on body compostion, weight loss, and dietary composition in women. *Journal of the American College of Nutrition* 16 (1): 68-73, 1997.

27. Burt VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M, Horan MJ, Labarthe D. Prevalence of hypertension in the US adult population: Results from the third national health and nutrition examination survey, 1988-1991. *Hypertension* 25: 305-313, 1995.

28. Cade R, Mars D, Wagemaker H, Zauner C, Packer D, Privette M, Cade M, Peterson J, Hood-Lewis D. Effect of aerobic exercise training on patients with systemic arterial hypertension. *American Journal of Medicine* 77: 785-790, 1984.

29. Celernajer DS, Sorensen KE, Spiegelhalter DJ, Georgakopoulos D, Robinson J, Deanfield JE. Aging is associated with endothelial dysfunction in healthy men years before the age-related decline in women. *Journal of the American College of Cardiology* 24: 471-476, 1994.

30. Ching P, Willet WC, Rimm EB, Colditz GA, Gortmaker SL, Stampfer MJ. Activity level and risk of overweight in male health professionals. *American Journal of Public Health* 86: 25-30, 1996.

31. Collins R, Peto R, MacMahon S, Hebert P, Fiebach NH, Eberlein KA, Godwin J, Qizilbash N, Taylor JO, Hennekens CH. Blood pressure, stroke, and coronary heart disease Part 2, short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context. *The Lancet* 335: 827-838, 1990.

32. Collins VR, Dowse GK, Toelupe PM, Imo TT, Aloaina FL, Spark RA, Zimmet PZ. Increasing prevalence of NIDDM in the Pacific island population of Western Samoa over a 13-year period. *Diabetes Care* 17 (4): 288-296, 1994.

33. Colombel A, Charbonnel B. Weight gain and cardiovascular risk factors in the post-menopauseal woman. *Human Reproduction* 12 (Suppl. 1): 134-145, 1997.

34. Cook WW. Proposed hostility and pharisaic-virtue scale for MMPI2. *Journal of Applied Psychology* 38: 414-418, 1954.

35. Crespo CJ, Keteyian SJ, Heath GW, Sempos CT. Leisure-time physical activity among US adults. *Archives of Internal Medicine* 156: 93-98, 1996.

36. Dannenberg AL, Keller JB, Wilson PWF, Castelli WP. Leisure time physical activity in the Framingham offspring study. *American Journal of Epidemiology* 129 (1): 76-88, 1989.

37. Davidson K, Hall P, MacGregor M. Gender differences in the relation between interview-derived hostility scores and resting blood pressure. *Journal of Behavioral Medicine* 19 (2): 185-201, 1996.

38. Davison R, Grant S, Mutrie N, Nash AS, Kelly MPT, Dargie HJ. Walk for health? *Journal of Sport Sciences.* 10: 556, 1992.

39. Davy KP, Seals DR, Tanaka H. Augmented cardiopulmonary and integrative sympathetic baroreflexes but attenuated peripheral vasoconstriction with age. *Hypertension* 32 (2): 298-304, 1998.

40. Davy KP, Tanaka H, Andros EA, Gerber JG, Seals DR. Influence of age on arterial baroreflex inhibition of sympathetic nerve activity in healthy adult humans. *American Journal of Physiology* 275 (5 Pt 2): H1768-H1772, 1998.

41. de Geus EJ, van Doornen LJ, Orlebeke JF. Regular exercise and aerobic fitness in relation to psychological make- up and physiological stress reactivity. *Psychosomatic Medicine* 55 (4): 347-363, 1993.

42. Debusk RF, Stenestrand U, Sheehan M, Haskell WL. Training effects of long versus short bouts of exercise in healthy subjects. *American Journal of Cardiology* 65: 1010-1013, 1990.

43. DeFronzo RA. The effects of insulin on renal sodium metabolism. *Diabetologia* 21: 165-171, 1981.

44. Defronzo RA, Ferrannini E. Insulin resistance. A mulitfaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* 14 (3): 173-194, 1991.

45. Dengel DR, Hagberg JM, Coon PJ, Drinkwater DT, Goldberg AP. Comparable effects of diet and exercise on body composition and lipoproteins in older men. *Medicine and Science in Sports and Exercise* 26: 1307-1315, 1994.

46. Dengel DR, Hagberg JM, Pratley RE, Rogus EM, Goldberg AP. Improvements in blood pressure, glucose metabolism, lipoprotein lipids after aerobic exercise plus weight loss in obese, hyptertensive middle-aged men. *Metabolism* 47 (9): 1075-1082, 1998.

47. Deshmukh R, Smith A, Lilly LS. Hypertension. In: Lilly LS, ed. *Pathophysiology of Heart Disease. Second Edition.* Baltimore, ML: Williams & Wilkins, pp. 267-288, 1998.

48. Dimsdale JE, Pierce C, Schoenfeld D, Brown A, Zusman R, Graham R. Suppressed anger and blood pressure: The effects of race, sex, social class, obesity, and age. *Psychosomatic Medicine* 48: 430-435, 1986.

49. DiPietro L. Physical activity, body weight, and adiposity: an epidemiologic perspective. In: Holloszy JO, ed. *Exercise and Sport Science Reviews.* Vol. 23. Baltimore: Williams & Wilkins, pp. 275-303, 1995.

50. Dowse GK, Zimmet PZ, Gareeboo H, George K, Alberti MM, Tuomilehto J, Finch CF, Chitson P, Tulsidas H. Abdominal obesity and physical inactivity as risk factors for NIDDM and impaired glucose tolerance in Indian, Creole, and Chinese Mauritians. *Diabetes Care* 14 (4): 271-282, 1991.

51. Doyne EJ, Ossip-Kline DJ, Bowman ED, Osborn KM, McDougal- Wilson IB. Running versus weight lifting in the treatment of depression. *Journal of Consulting Clinical Psychology* 55: 748-754, 1987.

52. Duncan JJ, Gordon NF, Scott CB. Women walking for health and fitness. *Journal of the American Medical Association* 266: 3295-3299, 1991.

53. Dunn AL, Marcus BH, Kampert JB, Garcia ME, Kohl HW, Blair SN. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness. *Journal of the American Medical Association* 281: 327-334, 1999.

54. Eaton CB, Lapane KL, Garber CE, Assaf AR, Lasater TM, Carleton RA. Physical activity, physical fitness, and coronary heart disease risk factors. *Medicine and Science in Sports and Exercise* 27 (3): 340-346, 1995.

55. Ebisu T. Splitting the distance of endurance running: on cardiovascular endurance and blood lipids. *Japanese Journal of Physical Education* 30: 37-43, 1985.

56. Etchegoyen GS, Ortiz D, Goya RG, Sala C, Panzica E, Sevillano A, Dron N. Assessment of cardiovascular risk factors in menopausal Argentinian women. *Gerontology* 41: 166-172, 1995.

57. Everson SA, Kauhanen J, Kaplan GA, Goldberg DE, Julkunen J, Tuomilehto J, Salonen JT. Hostility and increased risk of mortality and acute myocardial infarction: the mediating role of behavioral risk factors. *American Journal of Epidemiology* 146 (2): 142-152, 1997.

58. Farmer ME, Locke BZ, Moscicki EK, Dannenberg AL, Larson DB, Radloff LS. Physical activity and depressive symptoms: the NHANES I Epidemiologic Follow-up Study. *American Journal of Epidemiology* 128 (6): 1340-1351, 1988.

59. Ferrannini E, Buzzigoli G, Bonadonna R, Giorico MA, Oleggini M, Graziadei L, Pedrinelli R, Brandi L, Bevilacqua S. Insulin resistance in essential hypertension. *New England Journal of Medicine* 317: 350-357, 1987.

60. Ferrannini E, Haffner SM, Stern MP. Essential hypertension: an insulin-resistant state. *Journal of Cardiovascular Pharmacology* 45 (suppl. 5): S18-S25, 1990.

61. Ferrannini E, Haffner SM, Stern MP. Insulin sensitivity and hypertension. *Journal of Hypertension* 8 (Suppl. 7): S169-S173, 1990.

62. Ferrannini E, Natali A, Capaldo B, Lehtovorta M, Jacob S, Yki-Jarvinen H. Insulin resistance, hyperinsulinemia, and blood pressure: role of age and obesity. *Hypertension* 30: 1144-1149, 1997.

63. Fiebach N, Harbert P, Stampfer M. A prospective study of high blood pressure and cardiovascular disorders in women. *American Journal of Epidemiology* 130: 646-654, 1989.

64. Finch D, Davis G, Bower J, Kirchner K. Effect of insulin on renal sodium handling in hypertensive rats. *Hypertension* 15: 514-518, 1990.

65. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *International Journal of Obesity* 22: 39-47, 1998.

66. Folsom AR, Prineas RJ, Kaye SA, Soler JT. Body fat distribution and self-reported prevalence of hypertension, heart attack, and other heart disease in older women. *International Journal of Epidemiology* 18: 361-367, 1989.

67. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of US adults. *American Journal of Epidemiology* 146 (3): 214-222, 1997.

68. Foreyt JP, Brunner RL, Goodrick GK, St. Jeor ST, Miller GD. Psychological correlates of reported physical activity in normal-weight and obese adults: the Reno diet-heart study. *International Journal of Obesity* 19 Suppl 4: S69-S72, 1995.

69. Fox KR. The Physcial Self-Perception Profile Manual, Northern Illinois University, 1990.

70. French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter JE. Predictors of weight change over two years among a population of working adults: the healthy worker project. *International Journal of Obesity* 18: 145-154, 1994.

71. Frey-Hewitt B, Vranizan KM, Dreon DM, Wood PD. The effect of weight loss by dieting or exercise on resting metabolic rate in overweight men. *International Journal of Obesity* 27: 327-334, 1990.

72. Friedman MJ, Bennet PL. Depression and hypertension. *Psychosomatic Medicine* 39: 134-142, 1977.

73. Gentry WD, Chesney AP, Gary HE, Hall RP, Harburg E. Habitual anger-coping styles: I. Effect on mean blood pressure and risk for essential hypertension. *Psychosomatic Medicine* 44: 195-201, 1982.

74. Gilders RM, Voner C, Dudley GA. Endurance training and blood pressure in normotensive and hypertensive adults. *Medicine and Science in Sports and Exercise* 21: 629-636, 1989.

75. Gilligan DM, Quyyumi AA, Cannon III RO. Effects of physiological levels of estrogen on coronary vasomotor function in postmenopausal women. *Circulation* 89: 2545-2551, 1994.

76. Gillum RF, Mussolino ME, Madans JH. Body fat distribition and hypertension incidence in women and men: The NHANES I epidemiologic follow-up study. *Intererenational Journal of Obesity* 22: 127-134, 1998.

77. Goldstein HS, Edelberg R, Meier CF, Davis L. Relationship of resting blood pressure and heart rate to experienced anger and expressed anger. *Psychosomatic Research* 50: 321-329, 1988.

78. Gupta AK, Clark RV, Kirchner KA. Effects of insulin on renal sodium excretion. *Hypertension* 19: I78-I82, 1992.

79. Guyton AC. Kidneys and fluids in pressure regulation: small but large pressure changes. *Hypertension* 19 (Suppl I): I2-I8, 1992.

80. Gwinup G. Effect of exercise alone on the weight of obese women. *Archives of Internal Medicine* 135: 676-680, 1975.

81. Gwinup G. Weight loss without dietary restriction: efficacy of different forms of aerobic exercise. *American Journal of Sports Medicine* 15 (3): 275-279, 1987.

82. Hagberg JM, Montain SJ, Martin WH, Ehsani AA. Effect of exercise training in 60- to 69-year-old persons with essential hypertension. *American Journal of Cardiology* 64: 348-353, 1989.

83. Hakim AA, Petrovitch H, Burchfiel CM, Ross W, Rodriguez BL, White LR, Yano K, Curb JD, Abbott RD. Effects of walking on mortality among nonsmoking retired men. *New England Journal of Medicine* 338: 94-99, 1998.

84. Hall JE. Renal and cardiovascular mechanisms of hypertension in obesity. *Hypertension* 23: 381-394, 1994.

85. Hall JE, Brands MW, Dixon WN, Smith MJ, Jr. Obesity-induced hyptertension: renal function and systemic hemodynamics. *Hypertension* 22: 292-299, 1993.

86. Hall JE, Brands MW, Kivlighn SD, Mizelle HL, Hildenbrandt DA, Gaillard CA. Chronic hyperinsulinemia and blood pressure: Interaction with catecholamines? *Hypertension* 4: 164-168, 1990.

87. Hall JE, Coleman TG, Mizelle HL, Smith MJ, Jr. Chronic hyperinsulinemia and blood pressure regulation. *American Journal of Physiology* 258: F722-F731, 1990.

88. Hall JE, Guyton AC, Smith MJ, Coleman TG. Blood pressure and renal function during chronic changes in sodium intake: role of angiotensin. *American Journal of Physiology* 239 (F271-F280), 1980.

89. Hardman AE, Jones PRM, Norgan NG, Hudson A. Brisk walking improves endurance fitness without changing body fatness in previously sedentary women. *European Journal of Applied Physiology* 65: 354-359, 1992.

90. Harris M. Epidemiolgical correlates of NIDDM in Hispanics, whites, and blacks in the U.S. population. *Diabetes Care* 14: 639-648, 1991.

91. Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, Wiedmeyer H-M, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults: The third National Health and Nutrition Examination Survey, 1988-1994. *Diabetes Care* 21 (4): 518-524, 1998.

92. Haskell WL. Dose-response issues from a biological perspective. In: Bouchard C, Shepard RJ, Stephens T, eds. *Physical Activity, Fitness and Health: International Proceedings and Consensus Statement.* Champaign, IL: Human Kinetics, pp. 1030-1039, 1994.

93. Haskell WL. Health consequences of physical activity: understanding and challenges regarding dose-response. *Medicine and Science in Sports and Exercise* 26 (6): 649-660, 1994.

94. Heise T, Magnusson K, Heinemann L, Sawicki PT. Insulin resistance and the effect of insulin on blood pressure in essential hypertension. *Hypertension* 32: 243-248, 1998.

95. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *The New England Journal of Medicine* 325: 147-152, 1991.

96. Henry JP, Stephens PM, Santisteban GA. A model of psychosocial hypertension showing reversibility and progression of cardiovascular complications. *Circulation Research* 36: 156, 1975.

97. Higashi U, Sasaki S, Sasaki N, Nakagagwa K, Ueda T, Yoshimuzu A, Kurisu S, Matsuura H, Kajiyama G, Oshima T. Daily aerobic exercise improves reactive hyperemia in patients with essential hypertension. *Hypertension* 33 (part II): 591-597, 1999.

98. Hinkleman LL, Nieman DC. The effects of a walking program on body composition and serum lipids and lipoproteins in overweight women. *Journal of Sports Medicine and Physical Fitness* 33: 49-58, 1993.

99. Hogikyan RV, Supiano MA. Arterial alpha-adrenergic responsiveness is decreased and SNS activity is increased in older humans. *American Journal of Physiology* 266: E717-E724, 1994.

100. Holloszy JO, Schultz J, Kusnierkiewicz J. Effects of exercise on glucose tolerance and insulin resistance. *Acta Medica Scandinavica* 711: 55-65, 1985.

101. Hsieh SD, Yoshinaga H, Muto T, Sakurai Y. Regular physical activity and coronary risk factors in Japanese men. *Circulation* 97 (7): 661-665, 1998.

102. Hunt BE, Davy KP, Jones PP, DeSouza CA, Van Pelt RE, Tanaka H, Seals DR. Systemic hemodynamic determinants of blood pressure in women: age, physical activity, and hormone replacement. *American Journal of Physiology* 273 (2 Pt 2): H777-H785, 1997.

103. Jakicic JM, Polley BA, Wing RR. Accuracy of self-reported exercise and the relationship with weight loss in overweight women. *Medicine and Science in Sports and Exercise* 30: 634-638, 1998.

104. Jakicic JM, Wing RR, Butler BA, Robertson RJ. Prescribing exercise in multiple short bouts versus one continuous bout: effects on adherence, cardiorespiratory fitness, and weight loss in overweight women. *International Journal of Obesity* 19: 893-901, 1995.

105. Jennings G, Nelson L, Nestel P, Esler M, Korner P, Burton D, Bazelmans J. The effects of changes in physical activity on major cardiovascular risk factors, hemodynamics, sympathetic function, and glucose utilization in man: a controlled study of four levels of activity. *Circulation* 73 (1): 30-40, 1986.

106. Jennings GL, Deakin G, Korner P, Merideth I, Kingwell B, Nelson L. What is the dose-respose relationship between exercise training and blood pressure? *Annals of Medicine* 23: 313-318, 1991.

107. Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The Sixth Report. *Archives of Internal Medicine* 157: 2413-2445, 1997.

108. Jonas BS, Franks P, Ingram DD. Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. *Archives of Family Medicine* 6 (1): 43-49, 1997.

109. Jones P, Davy KP, Seals DR. Relations of total and abdominal adiposity to muscle sympathetic nerve activity in healthy older males. *International Journal of Obesity* 21: 1053-1057, 1997.

110. Jones PP, Davy KP, Alexander S, Seals DR. Age-related increase in muscle sympathetic nerve activity is associated with abdominal adiposity. *American Journal of Physiology* 272: E976-E980, 1997.

111. Jones-Webb R, Jacobs DR, Jr., Flack JM, Liu K. Relationships between depressive symptoms, anxiety, alcohol consumption, and blood pressure: results from the CARDIA Study. Coronary Artery Risk Development in Young Adults Study. *Alcohol and Clinical Experimental Research* 20 (3): 420-427, 1996.

112. Julius S, Gudbrandsson T, Jamerson K, Shahab ST, Andersson O. The hemodynamic link between insulin resistance and hypertension. *Journal of Hypertension* 9: 983-986, 1991.

113. Kahn HS, Tatham LM, Rodriguez C, Calle EE, Thun MJ, Heath CW. Stable behaviors associated with adults' 10-year change in body mass index and likelihood of gain at the waist. *American Journal of Public Health* 87: 747-754, 1997.

114. Kannel WB. Blood pressure as a cardiovascular risk factor. *Journal of the American Medical Association* 275: 1571-1576, 1996.

115. Kannel WB. Secular blood pressure trends in normotensive persons: the Framingham study. *American Heart Journal* 125: 1154-1158, 1993.

116. Kaplan NM. The deadly quartet. Upper-body obesity, glucose tolerance, hypertriglyceridemia, and hypertension. *Archives of Internal Medicine* 149: 1514-1520, 1989.

117. Kaplan NM. The treatment of hypertension in women. *Archives of Internal Medicine* 155: 563-567, 1995.

118. Katzel LI, Bleecker ER, Colman EG, Rogus EM, Sorkin JD, Goldberg AP. Effects of weight loss vs aerobic exercise training on risk factors for coronary disease in healthy, obese, middle-aged and older men. *Journal of the American Medical Association* 274 (24): 1915-1921, 1995.

119. Kaufman JS, Asuzu MC, Mufunda J, Forrester T, Wilks R, Luke A, Long AE, Cooper RS. Relationship between blood pressure and body mass index in lean populations. *Hypertension* 30: 1511-1516, 1997.

120. King AC, Taylor CB, Haskell WL. Effects of differing intensities and formats of 12 months of exercise training on psychological outcomes in older adults. *Health Psychology* 12 (4): 292-300, 1993.

121. Kinoshita A, Urata H, Tanabe Y, Ikeda M, Tanaka H, Shindo M, Arakawa K. What types of hypertensives respond better to mild exercise therapy. *Journal of Hypertension* 6 (suppl 4): S631-S633, 1988.

122. Kiyonaga A, Arakawa K, Tanaka H, Shindo M. Blood pressure and hormonal responses to aerobic exercise. *Hypertension* 7: 125-131, 1985.

123. Kokkinnos PF, Holland JC, Narayan P, Colleran JA, Dotson CO, Papademetriou V. Miles run per week and high-density lipoprotein cholesterol levels in healthy, middle-aged men. *Archives of Internal Medicine* 155: 415-420, 1995.

124. Kolanowski J. Obesity and hypertension: from pathophysiology to treatment. *Interernational Journal of Obesity* 23 (Suppl. 1): 42-46, 1999.

125. Kroke A, Bergmann M, Klipstein-Brobusch K, Boeing H. Obesity, body fat distribution and body build: Their relation to blood pressure and prevalence of hyptertension. *International Journal of Obesity* 22: 1062-1070, 1998.

126. LaFontaine T, Dabney S, Brownson R, Smith C. The effect of physical activity on all cause mortality compared to cardiovascular mortality: a review of research and recommendations. *Missouri Medicine* 91 (4): 188-94, 1994.

127. Lahad A, Heckbert SR, Patrick DL, Psaty BM. Hostility, aggression and the association with hypertension in post- menopausal women. *Journal of Human Hypertension* 10 Suppl 3: S115-S121, 1996.

128. Lakka TA, Venalainen JM, Rauramaa R, Salonen R, Tuomilehto J, Salonen JT. Relation of leisure-time physical activity and cardiorespiratory fitness to the risk of acute myocardial infarction. *The New England Journal of Medicine* 330 (22): 1549-1554, 1994.

129. Landsberg L. Hyperinsulinemia: possible role in obesity-induced hypertension. *Hypertension* 19 (suppl I): I61-I66, 1992.

130. Lawler KA, Harralson TL, Armstead CA, Schmied LA. Gender and cardiovascular responses: what is the role of hostility. *Journal of Psychosomatic Research* 37: 603-613, 1993.

131. Leitschuh M, Cupples A, Kannel W, Gagnon D, Chobanian A. High-normal blood pressure progression to hypertension in the Framingham Heart Study. *Hypertension* 17: 22-27, 1991.

132. Lemaitre RN, Heckbert SR, Psaty BM, Siscovick DS. Leisure-time physical activity and the risk of nonfatal myocardial infarction in postmenopausal women. *Archives of Internal Medicine* 155: 2302-2308, 1995.

133. Leon AS, Casal D, Jacobs D, Jr. Effects of 2,000 kcal per week of walking and stair climbing on phycial fitness and risk factors for coronary heart disease. *Journal of Cardiopulmonary Rehabilitation* 16: 183-192, 1996.

134. Leon AS, Connett J, Jacobs DR, Rauramaa R. Leisure-time physical activity levels and risk of coronary heart disease and death. The Multiple Risk Factor Intervention Trial. *Journal of the American Medical Association* 258 (6): 2388-2395, 1987.

135. Leon AS, Conrad J, Hunninghake DB, Serfass R. Effects of a vigorous walking program on body composition, and carbohydrate and lipid metabolism of obese young men. *American Journal of Clinical Nutrition* 32: 1776-1787, 1979.

136. Lieberman EH, Gerhard MD, Uehata A, Walsh BW, Selwyn AP, Ganz P, Yeung AC, Creager MA. Estrogen improves endothelium-dependent, flow-mediated vasodilation in postmenopausal women. *Annals of Internal Medicine* 121: 936-941, 1994.

137. Lissner L, Bengtsson C, Lapidus L, Kristjansson K, Wedel H. Fasting insulin in relation to subsequent blood pressure changes and hypertension in women. *Hypertension* 20: 797-801, 1992.

138. Lyketsos G, Arapakis G, Psaras M, Photiou I, Blackburn IM. Psychological characteristics of hypertensive and ulcer patients. *Journal of Psychosomatic Research* 26 (2): 255-262, 1982.

139. Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC, Hennekens CH. A prospective study of exercise and incidence of diabetes among US male physicians. *Journal of the American Medical Association* 268 (1): 63-67, 1992.

140. Manson JE, Rimm EB, Stampfer MJ, Colditz GA, Willett WC, Krolewski AS, Rosner B, Hennekens CH, Speizer FE. Physical activity and incidence of non-insulin-depedent diabetes mellitus in women. *The Lancet* 338: 774-778, 1991.

141. Martinsen EW, Hoffart A, Solberg O. Comparing aerobic with nonaerobic forms of exercise in the treatment of clinical depression: a randomized trial. *Comprehensive Psychiatry* 30 (4): 324-331, 1989.

142. McCrory M, Mole PA, Gomez T, Dewey KG, Bernauer EM. Body compostion by air-displacement plethysmography by using predicted and measured thoracic gas volumes. *Journal of Applied Physiology* 84 (4): 1475-1479, 1998.

143. Meesters CMG, Muris P, Backus IPG. Dimensions of hostility and myocardial infarction in adult males. *Journal of Psychosomatic Research* 40 (1): 21-28, 1996.

144. Mendes de Leon CF, Krumholz HM, Seeman TS, Vaccarino V, Williams CS, Kasl SV, Berkman LF. Depression and risk of coronary heart disease in elderly men and women. *Archives of Internal Medicine* 158: 2341-2348, 1998.

145. Mensink GB, Heerstrass DW, Neppelenbroek SE, Schuit AJ, Bellach BM. Intensity, duration, and frequency of physical activity and coronary risk factors. *Medicine and Science in Sports and Exercise* 29 (9): 1192-1198, 1997.

146. Miller SB, Dolgoy L, Friese M, Sita A. Dimensions of hostility and cardiovascular response to interpersonal stress. *Journal of Psychosomatic Research* 41: 81-95, 1996.

147. Miller WC, Koceja DM, Hamilton EJ. A meta-analysis of the past 25 years of weight loss research using diet, exercise, or diet plus exercise intervention. *International Journal of Obesity* 21: 941-947, 1997.

148. Mitchell KD, Braam B, Navar LG. Hypertensinogenic mechanisms mediated by renal actions of renin-angiotensin system. *Hypertension* 19: I18-I27, 1992.

149. Modan M, Halkin H, Alrnog S, Lusky A, Eshkol A, Schefl M. Hyperinsulinemia: a link between hypertension, obesity, and glucose tolerance. *Journal of Clinical Investigation* 75: 809-817, 1985.

150. Morgan WP. Physical Activity, fitness, and depression. In: Bouchard C, Shepard RJ, Stephens T, eds. *Physical Activity, Fitness and Health: International Proceedings and Consensus Statement.* Champaign, IL: Human Kinetics, pp. 851-866, 1994.

151. Morris JN, Heady JA, Raffle PAB, Roberts CG, Parks JW. Coronary heart-disease and physical activity of work. *Lancet* 2: 1053-1057, 1953.

152. Motoyama M, Sunami Y, Kinoshita F, Kiyonaga A, Tanaka H, Shindo M, Irie T, Urata H, Sasaki J, Arakawa K. Blood pressure lowering effect of low intensity aerobic training in elderly hypertensive patients. *Medicine and Science in Sports and Exercise* 30 (6): 818-823, 1998.

153. Murphy MH, Hardman AE. Training effects of short and long bouts of brisk waking in sedentary women. *Medicine and Science in Sports and Exercise* 30 (1): 152-157, 1998.

154. Musante L, Treiber FA, Davis H, Strong WB, Levy M. Hostility: relationship to lifestyle behaviors and physical risk factors. *Behavior Medicine* 18 (1): 21-26, 1992.

155. Mykkanen L, Haffner SM, Ronnemaa T, Watanabe RM, Laakso M. Relationship of plasma insulin concentration and insulin sensitivity to blood pressure. Is it modified by obesity? *Journal of Hypertension* 14: 399-405, 1996.

156. Netter P, Neuhauser-Metternich S. Types of aggressiveness and catecholamine response in essential hypertensives and healthy controls. *Journal of Psychosomatic Research* 35: 409-419, 1991.

157. Ng AV, Callister R, Johnson DG, Seals DR. Age and gender influence muscle sympathetic nerve activity at rest in healthy humans. *Hypertension* 21 (4): 498-503, 1993.

158. Nouri S, Beer J. Relations of moderate physical exercise to scores on hostility, aggression, and trait-anxiety. *Perceptual and Motor Skills* 68: 1191-1194, 1989.

159. Paffenbarger RS, Hyde RT, Wing AL, Hsieh CC. Physical activity, all-cause mortality, and longevity of college alumni. *The New England Journal of Medicine* 314: 605-613, 1986.

160. Paffenbarger RS, Hyde RT, Wing AL, Lee IM, Jung DL, Kampert JB. The association of changes in physical-activity level and other lifestyle characteristics with mortality among men. *The New England Journal of Medicine* 328: 538-545, 1993.

161. Paffenbarger RS, Hyde RT, Wing AL, Steinmetz CH. A natural history of athleticism and cardiovascular health. *Journal of the American Medical Association* 252: 491-495, 1984.

162. Paffenbarger RS, Jr., Lee I-M, Leung R. Physical activity and personal characteristics associated with depression and suicide in American college men. *Acta Psychiatrica Scandinavica Supplementum* 377: 16-22, 1994.

163. Paffenbarger RS, Jr., Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. *American Journal of Epidemiology* 108 (3): 161-175, 1978.

164. Paffenbarger RS, Jr., Wing AL, Hyde RT, Jung DL. Physical activity and incidence of hypertension in college alumni. *American Journal of Epidemiology* 117 (3): 245-257, 1983.

165. Palmer LK. Effects of a walking program on attributional style, depression, and self-esteem in women. *Perceptual and Motor Skills* 81 (3 Pt 1): 891-898, 1995.

166. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW, King AC, Kriska A, Leon AS, Marcus BH, Morris J, Paffenbarger RS, Patrick K, Pollock ML, Rippe JM, Sallis J, Wilmore JH. Physical activity and public health: A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association* 273: 402-407, 1995.

167. Pierce TW, Madden DJ, Siegal WC, Blumenthal JA. Effects of aerobic exercise on cognitive and psychosocial functioning in patients with mild hypertension. *Health Psychology* 12: 286-291, 1993.

168. Pollock ML, Miller HS, Jr., Janeway R, Linnerud AC, Robertson B, Valentino R. Effects of walking on body composition and cardiovascular function of middle-aged men. *Journal of Applied Physiology* 30: 126-130, 1971.

169. Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. *Annual Reviews of Public Health* 8: 253-287, 1987.

170. Radloff LS. The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement* 1 (3): 385-401, 1977.

171. Ready AE, Naimark B, Ducas J, Sawatzky JV, Boreskie SL, Drinkwater DT, Oosterveen S. Influence of walking volume on health benefits in women post-menopause. *Medicine and Science in Sports and Exercise* 28 (9): 1097-1105, 1996.

172. Reaven GM. Banting Lecture 1988: Role of insulin resistance in human disease. *Diabetes* 37: 1595-1607, 1988.

173. Reaven PD, Barrett-Connor E, Edelstein S. Relation between leisure-time physical activity and blood pressure in older women. *Circulation* 83 (2): 559-565, 1991.

174. Regensteiner JG, Shetterly SM, Mayer EJ, Eckel RH, Haskell WL, Baxter J, Hamman RF. Relationship between habitual physical activity and insulin area among individuals with impaired glucose tolerance. The San Luis Valley Diabetes Study. *Diabetes Care* 18 (4): 490-497, 1995.

175. Rocchini AP. Cardiovascular regulation in obesity-induced hypertension. *Hypertension* 19: I56-I60, 1992.

176. Rocchini AP. Insulin resistance and blood pressure regulation in obese and nonobese subjects. *Hypertension* 17: 837-842, 1991.

177. Rocchini AP, Moorehead C, DeRemer S, Goodfriend TL, Ball DL. Hyperinsulinemia and the aldosterone and pressor responses to angiotensin II. *Hypertension* 15: 861-866, 1990.

178. Ross CE, Hayes D. Exercise and psychologic well-being in the community. *American Journal of Epidemiology* 127: 762-771, 1988.

179. Rowe JW, Young JB, Mimaker KL, Stevens AL, Pallotta J, Landsberg L. Effect of insulin and glucose infusion on sympathetic nervous system activity in normal man. *Diabetes* 30: 219-225, 1981.

180. Ryan AS, Nicklas BJ. Age-related changes in fat deposition in mid-thigh muscle in women: relationships with metabolic cardiovascular disease risk factors. *International Journal of Obesity* 23: 126-132, 1999.

181. Sallis JF, Johnson CC, Trevorrow TR, Kaplan RM, Hovell MF. The relationship between cynical hostility and blood pressure reactivity. *Journal of Psychosomatic Research* 31 (1): 111-116, 1987.

182. Salonen JT, Slater JS, Tuomilehto J, Rauramaa R. Leisure time and occupational physical activity: risk of death from ischemic heart disease. *American Journal of Epidemiology* 127 (1): 87-94, 1988.

183. Santiago MC, Alexander JF, Stull GA, Serfass RC, Hayday AM, Leon AS. Physiological responses of sedentary women to a 20-week conditioning program of walking or jogging. *Scandinavian Journal of Sports Science* 9: 33-39, 1987.

184. Santiago MC, Leon AS, Serfass RC. Failure of 40 weeks of brisk walking to alter blood lipids in normolipemic women. *Can. J. Appl. Physiol.* 20 (4): 417-428, 1995.

185. Seals DR, Hagberg JM. The effect of exercise training on human hypertension: a review. *Medicine and Science in Sports and Exercise* 16 (3): 207-215, 1984.

186. Seals DR, Silverman HG, Reiling MJ, Davy KP. Effect of regular aerobic exercise on elevated blood pressure in postmenopausal women. *American Journal of Cardiology* 80: 49-55, 1997.

187. Segal KR, Edano A, Abalos A, Albu J, Blando L, Tomas MB, Pi-Sunyer FX. Effect of exercise training on insulin sensitivity and glucose metabolism in lean, obese, and diabetic men. *Journal of Applied Physiology* 71 (6): 2402-2411, 1991.

188. Siegel PZ, Brackbill RM, Heath GW. The epidemiology of walking for exercise: implications for promoting activity among sedentary groups. *American Journal of Public Health* 85 (5): 706-710, 1995.

189. Siri NE. Body composition from fluid spaces and density. In: Brozek J, Henschel A, eds. *Techniques For Measuring Body Composition*. Washington, D.C.: National Academy of Science, pp. 223-224, 1961.

190. Slattery ML, McDonald A, Bild DE, Caan BJ, Hilner JE, Jacobs DR, Jr., Liu K. Associations of body fat and its distribution with dietary intake, physical activity, alcohol, and smoking in blacks and whites. *American Journal of Clinical Nutrition* 55: 943-949, 1992.

191. Smutok MA, Reece C, Kokkinos PF, Farmer CM, Dawson PK, DeVane J, Patterson J, Goldberg AP, Hurley BF. Effects of exercise training modality on glucose tolerance in men with abnormal glucose regulation. *International Journal of Sports Medicine* 15 (6): 283-289, 1994.

192. Sowers M, Gonzalez VC, Stern MP, Fox C, Mitchell BD. Relationships between physical activity, insulin levels and lipids in non-diabetic low income residents of Mexico City: the Mexico City Diabetes Study. *Archives of Medical Research* 26: 133-140, 1995.

193. Spicer J, Chamberlain K. Cynical hostility, anger, and resting blood pressure. *Journal of Psychosmatic Research* 40: 359-368, 1996.

194. Staessen J, Bulpitt CJ, Fagard R, Lijnen P, Amery A. The influence of menopause on blood pressure. *Journal of Human Hypertension* 3: 427-433, 1989.

195. Stamler J. Blood Pressure and high blood pressure: aspects of risk. *Hypertension* 18 (Suppl I): I95-I107, 1991.

196. Stensel DJ, Brooke-Wavell K, Hardman AE, Jones PRM, Norgan NG. The influence of a 1-year programme of brisk walking on endurance fitness and body composition in previously sedentary men aged 42-59 years. *European Journal of Applied Physiology* 68: 531-537, 1994.

197. Stephens T. Physical activity and mental health in the United States and Canada: evidence from four population surveys. *Preventive Medicine* 17: 35-47, 1988.

198. Steptoe A, Melville D, Ross A. Behavioral response demands, cardiovascular reactivity, and essential hypertension. *Psychosomatic Medicine* 46 (1): 33-48, 1984.

199. Stevenson ET, Davy KP, Jones PP, Desouza CA. Blood pressure risk factors in healthy postmenopausal women: physical activity and hormone replacement. *Journal of Applied Physiology* 82 (2): 652-660, 1997.

200. Stout RW, Bierman E, Ross R. Effect of insulin on the proliferation of cultured primate smooth muscle cells. *Circulation Research* 36: 319-327, 1975.

201. Su H-Y, Sheu WH-H, Chin H-ML, Jeng C-Y, Chen Y-DI, Reaven GM. Effect of weight loss on blood pressure and insulin resistance in normotensive and hypertensive obese individuals. *American Journal of Hypertension* 8: 1067-1071, 1995.

202. Suarez EC, Kuhn CM, Schanberg SM, Williams RB, Zimmerman EA. Neuroendocrine, cardiovascular, and emotional response of hostile men: the role of interpersonal challenge. *Psychosomatic Medicine* 60: 78-88, 1998.

203. Sundlof G, Wallin BG. Human muscle nerve sympathetic activity at rest. Relationship to blood pressure and age. *Journal of Physiology* 274: 621-637, 1978.

204. Taddei S, Virdis A, Ghiadoni L, Mattei P, Sudano I, Bernini G, Pinto S, Salvetti A. Menopause is associated with endothelial dysfunction in women. *Hypertension* 28: 576-582, 1996.

205. Taddei S, Virdis A, Mattei P, Ghiadoni L, Gennari A, Fasolo CB, Sudano I, Salvetti A. Aging and endothelial function in normotensive subjects and patients with essential hypertension. *Hypertension* 91: 1981-1987, 1987.

206. Tanaka H, Bassett Jr. DR, Howley ET, Thompson DL, Ashraf M, Rawson FL. Swimming training lowers the resting blood pressure in individuals with hypertension. *Journal of Hypertension* 15: 651-657, 1997.

207. Tanaka H, Clevenger CM, Jones PP, Seals DR, DeSouza CA. Influence of body fatness on the coronary risk profile of physically active postmenopausal women. *Metabolism* 47 (9): 1112-1120, 1998.

208. Tanaka H, Davy KP, Seals DR. Cardiopulmonary baroreflex inhibition of sympathetic nerve activity is preserved with age in healthy humans. *Journal of Physiology* 515 (Pt 1): 249-254, 1999.

209. Tanaka H, Dinenno FA, Hunt BE, Jones PP, DeSouza CA, Seals DR. Hemodynamic sequelae of age-related increases in arterial stiffness in healthy women. *American Journal of Cardiology* 82 (9): 1152-1155, 1998.

210. Tanaka H, Reiling MJ, Seals DR. Regular walking increases peak limb vasodilatory capacity of older hypertensive humans: implications for arterial structure. *Journal of Hypertension* 16 (4): 423-428, 1998.

211. Thailer SA, Friedman R, Harshfield GA, Pickering TG. Psychological differences between high-normal-,normal-, and low-renin hypertensives. *Psychosomatic Medicine* 47: 294-297, 1985.

212. Toft I, Bonaa KH, Jenssen T. Insulin resistance in hypertension is associated with body fat rather than blood pressure. *Hypertension* 32: 115-122, 1998.

213. Tremblay A, Despres J-P, LeBlanc C, Craig CL, Ferris B, Stephens T. Effect of intensity of physical activity on body fatness and fat distribution. *American Journal of Clinical Nutrition* 51: 153-157, 1990.

214. Trovati M, Carta Q, Cavalot F, Vitali S, Banaudi C, Lucchina PG, Fiocchi F, Emanuelli G, Lenti G. Influence of physical training on blood glucose control, glucose tolerance, insulin secretion, and insulin action in non-insulin-dependent diabetic patients. *Diabetes Care* 7 (5): 416-420, 1984.

215. Tuck ML. Obesity, the sympathetic nervous system, and essential hypertension. *Hypertension* 19: I67-I77, 1992.

216. U.S. Departmen of Health and Human Services. *Physical Activity and Health: A Report of the Surgeon General*. Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease and Prevention and Health Promotion, 1996.

217. Urata H, Kiyonaga A, Ikeda M, Tanaka H, Shindo M, Arakawa K. Antihypertensive and volume-depleting effects of mild exercise on essential hypertension. *Hypertension* 9: 245-252, 1987.

218. Van Dale D, Saris WHM, Hoor FT. Weight maintenance and resting metabolic rate 18-40 months after a diet/exercise treatment. *International Journal of Obesity* 14: 347-359, 1990.

219. Vitaliano PP, Russo J, Bailey SL, Young HM, McCann BS. Psychosocial factors associated with cardiovascular reactivity in older adults. *Psychosomatic Medicine* 55 (2): 164-177, 1993.

220. Waked EG, Jutai JW. Baseline and reactivity measures of blood pressure and negative affect in borderline hypertension. *Physiolology and Behavior* 47 (2): 265-271, 1990.

221. Ward KD, Sparrow D, Landsberg L, Young JB, Vokonas PS, Weiss ST. Influence of insulin, sympathetic nervous system activity, and obesity on blood pressure: the Normative Aging Study. *Journal of Hypertention* 14: 301-308, 1996.

222. Weinsier RL, Norris DJ, Birch R, Berstein RS, Wang J, Yang M, Pierson RN, Jr., Van Itallie TB. The relative contribution of body fat and fat pattern to blood pressure level. *Hypertension* 7: 578-585, 1985.

223. Westererp KR, Goran MI. Relationship between physical activity related energy expenditure and body composition: a gender difference. *International Journal of Obesity* 21: 184-188, 1997.

224. Whithurst M, Menendez E. Endurance training in older women. *Physician and Sportsmedicine* 19 (6): 95-103, 1991.

225. Williams PT. High-density lipoprotein cholesterol and other risk factors for coronary heart disease in female runners. *New England Journal of Medicine* 334: 1298-1303, 1996.

226. Williams PT. Relationship of distance run per week to coronary heart disease risk factors in 8283 male runners. The National Runners' Health Study. *Archives of Internal Medicine* 157 (2): 191-198, 1997.

227. Wood WG, Elias MF, Schultz NR, Pentz CA. Anxiety and depression in young and middle aged hypertensive and normotensive subjects. *Experimental Aging Research* 5 (1): 15-30, 1979.

228. Wu Z, Wu X, Zhang Y. Relationship of menopausal status and sex hormones to serum lipids and blood pressure. *International Journal of Epidemiology* 19 (2): 297-302, 1990.

229. Yamanouchi K, Shinozaki T, Chikada K, Nishikawa T, Ito K, Shimizu S, Ozawa N, Suzuki Y, Maeno H, Kato K, Oshida Y, Sato Y. Daily walking combined with diet therapy is a useful means for obese NIDDM patients not only to reduce body weight but also to improve insulin sensitivity. *Diabetes Care* 18 (6): 775-778, 1995.

APPENDICES

APPENDIX A

Informed Consent Form

INFORMED CONSENT

TITLE OF STUDY: The Dose Effects of Walking on Blood Pressure in Post-Menopausal Women

PURPOSE

You are invited to participate in a research project examining the effects of a 24 week walking program on several measures of health and fitness. This study will begin in the Summer of 1998 and will conclude in the Winter of 1998 or Spring of 1999. This study will provide information on the relationship between walking and specific health benefits.

PROCEDURES

If you choose to participate in this study you will report to the Applied Physiology Laboratory, room 310 in the Health, Physical Education and Recreation (HPER) building on three separate occasions: baseline, 12 weeks, and 24 weeks. On each occasion, you should expect to spend between 1-2 hours. Prior to any testing you will complete a questionnaire regarding your medical history, exercise training habits, and menstrual cycle history. These forms will be used to insure that the testing is safe for you. You will be asked to report to the laboratory in a rested condition not having consumed food 10-12 hours prior to testing. You will also be asked to refrain from alcohol consumption and exercise for 24 hours prior to the test.

On each of your visits, you will report to the laboratory between 6:30 and 8:00 a.m. after a 10-12 hour overnight fast. Your blood pressure and heart rate will be measured three times while you are sitting in a chair after you have rested quietly for five minutes. A needle will then be inserted into your forearm and a 5-ml (about 1 teaspoon) blood sample will be taken to be later analyzed for glucose and insulin levels. Your body composition will be assessed by measuring your height, weight, waist and hip circumferences, and body fat percentage. Your height and weight will be measured on scales like those typically found in a doctor's office. Next your body fat will be measured as you sit for approximately 3 minutes in a sealed chamber. While in the chamber you will be able to breathe normally and see your surroundings. Following the body fat test, you will perform an exercise test on a motorized treadmill to evaluate your cardiorespiratory fitness. During the exercise test you will breathe through a mouthpiece while wearing noseclips. The treadmill test will begin at a low level of exertion and the speed and incline will be increased every three minutes for 9 minutes.

You will be given an electronic pedometer to wear throughout the day and record the number of steps shown on the pedometer output for two weeks. This information will allow us to determine your current average weekly walking distance. You will also be given psychological and physical activity questionnaires, and a three day dietary recall form and will be asked to record the food and drink that you consume for the three days. This information will allow us to measure psychological variables related to blood pressure as well as physical activity status and dietary intake.

WALKING PROGRAM

After completion of all the baseline testing measures, you will be randomly assigned to one of three groups: (1) walking 16 km/week (10 miles/wk), (2) walking 32 km/week (20 miles/wk), or (3) inactive control group. Supervised walking sessions will be held 5 days/week at the University of Tennessee's outdoor track or on a premeasured level course. Initially, both walking groups will walk 16 km /week. The 32 km/week group will increase their walking distance by 3.2 km/week (2 miles/wk) until they reach their prescribed distance by the fifth week. You will be asked to walk at a self-selected, comfortable pace. Your heart rate will be monitored by counting your pulse and occasionally with a heart rate watch. Prior to and following your walking session, you will do warm-up and cool-down exercises to help prevent any muscle or joint injury and soreness. You will be encouraged to attend at least 3 supervised sessions/week, and will be given a target number of steps to walk daily and weekly in order to meet your prescribed walking distance. Those in the 16 km/week group will need to walk an additional 20,000 steps/week, and those in the 32 km/week group will need to walk approximately 40,000 more steps/week above your baseline average weekly steps. Your walking duration, distance, and heart rate will be recorded in a daily log book, along with additional physical activities that you may have done that day. Those in the inactive control group will be asked not to make any changes to your current lifestyle habits. You will be given a pedometer to wear for one week each month, and will record the number of steps taken each day in a log book.

POTENTIAL RISKS OF PARTICIPATION

The potential risks that may occur while participating in this study are those associated with any type of exercise: injury to muscles and / or joints, dizziness, headache, and in rare instances heart attack (one in 10,000). However, these risks are minimal in healthy individuals, and the investigators are trained to conduct these types of experiments. You may experience some local bruising, tenderness and / or an infection at the area where blood samples were taken. Trained technicians will perform the blood sampling technique and sterile procedures will be used to minimize this risk. There is also the risk of your blood pressure response to become exaggerated during walking since you already have high blood pressure. However, exercise is recommended as the initial treatment of mild to moderate high blood pressure due to the exercise-related health benefits. Also, you will be walking at a self-selected pace which would not put you at any more risk than your normal every day walking. In the unlikely event that physical injury occurs as a result of participating in this study, financial compensation is not automatically available and medical treatment will not be provided free of charge. If physical injury should occur through the course of the study immediately notify Kerrie Moreau or Dr. Thompson (see below for phone and office numbers).

BENEFITS OF PARTICIPATION

This study will provide information on the dose-response relationship between walking and specific health benefits. You will benefit by acquiring valuable information on your current health status including: blood lipid profile, glucose tolerance, insulin sensitivity, body composition, fitness level, and resting and exercise blood pressure responses. If you participate in the walking programs, you may obtain some health and fitness benefits, and after completion of the study you will be given information to help improve or maintain your current level of health and conditioning. Those who are assigned to the sedentary control group will be able to participate in a walking program at the end of the 24 week study.

CONFIDENTIALITY

Only you and members of the research team will have access to your results. All data will be coded by a subject number rather than by name and will be kept in a locked file cabinet by Kerrie Moreau. The results of the research will be published; however, no publication will contain information that will allow you to be identified.

RIGHT TO ASK QUESTIONS AND/OR TO WITHDRAW FROM THIS INVESTIGATION

If you have any questions or concerns at any time during the course of this investigation or after you complete this study, you may contact Kerrie Moreau or Dr. Thompson at (423) 974 - 1271. Kerrie Moreau and Dr. Thompson's offices are located in the HPER Building in rooms 305 and 347, respectively. If at any time during this study events occur that will impact your ability to participate (e.g., you sustain a training injury, you are ill etc.), you should inform Kerrie Moreau or Dr. Thompson immediately. As a volunteer in this study, it is your right to withdraw from this investigation at any time. If you decide to withdraw from this study, you will in no way be penalized.

CONSENT

By signing this form, I am indicating that I have read the above information and agree to participate in this project. I have had the procedures and potential risks explained to me and my questions have been answered to my satisfaction. I understand that I will receive a copy of this consent to keep.

Participant's Signature____________________________ **Date**______________

Investigator's Signature____________________________ **Date**______________

APPENDIX B

Health History Questionnaire

SURVEY OF EXERCISE TRAINING, MEDICAL HISTORY AND MENSTRUAL HISTORY

NAME ______________________________ DATE ____________

DATE OF BIRTH __________

ADDRESS ____________________________

____

PHONE #s (home)________(work)__________

Please answer the following questions. This information will only be used for research purposes and will not be made public. Please answer these questions based on physical exercise in which you regularly engage. **This should not include daily work activities such as walking from one office to another.**

1. Do you regularly engage in exercise? _____ If yes, please describe.

____

____

2. On the average, how many times per week do you engage in exercise training?
   0___ 1___ 2___ 3___ 4___ 5___ 6___ 7___

3. On the average, how long are your workouts?
   0-19 minutes ____ 20-40 minutes _____ more that 40 minutes _____

4. Describe the type of exercise or physical activities in which you participate on a weekly basis.

____

____

5. How long have you been exercising at this level?
   less than 6 months _____
   6 - 12 months _____
   1-2 years _____
   3 or more years _____

MENSTRUAL HISTORY

1. At approximately what age did you begin menstruating? __________

2. Do you currently have regular menstrual cycles (i.e., regularly spaced periods of menstrual bleeding)?
____

If you answered NO to question #2:

a. Have your menstrual periods stopped completely? _____

b. When did you have your last period? ______

c. If you still occasional have menstrual bleeding, describe the pattern.

____

____

3. Are you taking hormone replacement medication such as estrogen or progesterone?______

MEDICAL HISTORY

Past History:

Have you ever been diagnosed with the following conditions? Please check the appropriate column.

	Yes	No	Don't Know
Free or easy bleeding (hemophilia)	( )	( )	( )
Rheumatic fever	( )	( )	( )
Heart murmur	( )	( )	( )
High blood pressure	( )	( )	( )
Any heart problem	( )	( )	( )
Varicose veins	( )	( )	( )
Lung disease	( )	( )	( )
Seizures	( )	( )	( )
Irregular heart beat	( )	( )	( )
Bronchitis	( )	( )	( )
Emphysema	( )	( )	( )
Diabetes	( )	( )	( )
Asthma	( )	( )	( )
Kidney Disease	( )	( )	( )
Liver Disease	( )	( )	( )
Severe Allergies	( )	( )	( )
Orthopedic problems	( )	( )	( )
Hyper- or Hypothyroidism	( )	( )	( )
Hepatitis	( )	( )	( )
AIDS	( )	( )	( )
Heparin Sensitivity	( )	( )	( )

Present Symptoms Review:

Have you recently had any of the following symptoms? Check "X" if yes.

Chest pain	( )	Frequent urination	( )
Shortness of breath	( )	Blood in urine	( )
Heart palpitations	( )	Burning sensations	( )
Leg or ankle swelling	( )	Severe headache	( )
Coughing of blood	( )	Blurred vision	( )
Low blood sugar	( )	Difficulty walking	( )
Feeling faint or dizzy	( )	Weakness in arm	( )
Leg numbness	( )	Significant emotional problem	( )

Do you smoke? ____ If yes, how many per day?_______

Are you taking any medications? ______If yes, please describe:

OTHER INFORMATION

Whom should we notify in case of an emergency?

Name ____

Address ______________________________________

Phone # ______________________________________

I have been given the opportunity to ask questions about any of the above items that were unclear, and I have answered all questions completely and truthfully to the best of my knowledge.

SIGNATURE __________________________ DATE _____________

APPENDIX C

Paffenbarger Physical Activity Questionnaire

Physical Activity Questionnaire

Name________________________________

1. How many city blocks or their equivalent do you normally walk each <u>day</u>? _________ Blocks/ day (Let 12 blocks = 1 mile)

2. What is your usual pace of walking? (Please check one.)
   a. ____ Casual or strolling (less than 2 mph)   b. ____Average or normal (2 to 3 mph)
   c. ____ Fairly brisk (3 to 4 mph)   d. ____Brisk or striding (4 mph or faster)

3. How many flights or stairs to you climb <u>up</u> each <u>day</u>? ____ flights/day (Let 1 flight = 10 steps.)

4. List any sports or recreation you have actively participated in during the past <u>month</u>?

Sport, Recreation, or Other Physical Activity (other than walking)	Number of Times/month	Average Time/Episode Hours	Minutes	Years Participation
a.				
b.				
c.				
d.				

5. Which of these statements best expresses your view? (Please check one.)
   a.___I do enough exercise to keep healthy. b. ____I ought to do more exercise. c. ____Don't know

6. At least once a week, do you engage in regular activity akin to brisk walking , jogging, bicycling, swimming, etc. long enough to work up a sweat, get your heart thumping, or get out of breath?
   ___ No   Why not? ________________
   ___ Yes   How many times per week? ______   Activity: ______________________

7. When you are exercising in your usual fashion, how would your rate your level of exertion (degree of effort)? (please circle one number.)

0	0.5	1	2	3	4	5	6	7	8	9	10
Normal	Very very weak (just noticeable)	Very weak	Weak	Moderate	Somewhat strong	Strong (heavy)		Very strong		Very very strong (almost maximal)	Maximal

8. On a usual weekday and a weekend day, how much time do you spend on the following activities? Total for each day should add to 24 hours.

	Usual Weekday Hours/Day	Usual Weekend Day Hours/Day
a. Vigorous activity (digging in the garden, strenuous sports, jogging, aerobic dancing, sustained swimming, brisk walking, heavy carpentry, bicycling on hills, etc.)		
b. Moderate activity (housework, light sports, regular walking, golf, yard work, lawn mowing, painting, repairing, light carpentry, ballroom dancing, bicycling on level ground, etc.)		
c. Light activity (office work, driving car, strolling, personal care, standing with little motion, etc.)		
d. Sitting activity (eating, reading, desk work, watching TV, listening to radio, etc.)		
e. Sleeping or reclining		

APPENDIX D

Psychological Surveys

PSYCHOLOGICAL SURVEY

Subject#____

Below is a list of the ways you might have felt or behaved. In the space before each item, write the number that corresponds with how often you have felt this way during the past week.

1 = Rarely or None of the Time (Less than 1 Day)
2 = Some or a Little of the Time (1-2 Days)
3 = Occasionally or a Moderate Amount of Time (3-4 Days)
4 = Most or All of the Time (5-7 Days)

<u>During the past week:</u>

_____ 1. I was bothered by things that usually don't bother me.
_____ 2. I did not feel like eating; my appetite was poor.
_____ 3. I felt that I could not shake off the blues even with help from my family or friends.
_____ 4. I felt that I was just as good as other people.
_____ 5. I had trouble keeping my mind on what I was doing.
_____ 6. I felt depressed.
_____ 7. I felt that everything I did was an effort.
_____ 8. I felt hopeful about the future.
_____ 9. I thought my life had been a failure.
_____ 10. I felt fearful.
_____ 11. My sleep was restless.
_____ 12. I was happy.
_____ 13. I talked less than usual.
_____ 14. I felt lonely.
_____ 15. People were unfriendly.
_____ 16. I enjoyed life.
_____ 17. I had crying spells.
_____ 18. I felt sad.
_____ 19. I felt that people dislike me.
_____ 20. I could not get "going".

Please answer the following questions by circling T for true or F for false.

T F It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.

T F Some of my family have habits that bother and annoy me very much.

T F People often disappoint me.

T F I am not easily angered.

T F There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.

T F When someone does me wrong, I feel I should pay them back if I can, just for the principle of the thing.

T F I don't blame anyone for trying to grab everything they can get in this world.

T F I can be friendly with people who do things which I consider wrong.

T F I do not blame a person for taking advantage of someone who lays himself open to it.

T F I would certainly enjoy beating a crook at their own game.

T F I have at times had to be rough with people who were rude or annoying.

T F I am often inclined to go out of my way to win a point with someone who opposed me.

T F I do not try to cover up my poor opinion or pity of a person so that they won't know how I feel.

T F I strongly defend my own opinion as a rule.

APPENDIX E

Physical Self Perception Profile and

The Percieved Importance Profile

THE PHYSICAL SELF PERCEPTION PROFILE (PSPP)

WHAT AM I LIKE?

These are statements which allow people to describe themselves.
There are no right or wrong answers since people differ a lot.

First, decide which one of the two statements best describes you.

Then, go to that side of the statement and check if it is just "sort of true" or "really true" FOR YOU.

	Really True for Me	Sort of True for Me		EXAMPLE		Sort of True for Me	Really True for Me
	☐	☐	Some people are very competitive	BUT	Others are not quite so competitive	☒	☐

REMEMBER to check only ONE of the four boxes

1.	☐	☐	Some people feel that they are not very good when it comes to playing sports	BUT	Others feel that they are really good at just about every sport	☐	☐
2.	☐	☐	Some people are not very confident about their level of physical conditioning and fitness	BUT	Others always feel confident that they maintain excellent conditioning and fitness	☐	☐
3.	☐	☐	Some people feel that compared to most, they have an attractive body	BUT	Others feel that compared to most, their body is not quite so attractive	☐	☐
4.	☐	☐	Some people feel that they are physically stronger than most people of their sex	BUT	Others feel that they lack physical strength compared to most others of their sex	☐	☐
5.	☐	☐	Some people feel extremely proud of who they are and what they can do physically	BUT	Others are sometimes not quite so proud of who they are physically	☐	☐
6.	☐	☐	Some people feel that they are among the best when it comes to athletic ability	BUT	Others feel that they are not among the most able when it comes to athletics	☐	☐

	Really True for Me	Sort of True for Me				Sort of True for Me	Really True for Me
7.	☐	☐	Some people make certain they take part in some form of regular vigorous physical exercise	BUT	Others don't often manage to keep up regular vigorous physical exercise	☐	☐
8.	☐	☐	Some people feel that they have difficulty maintaining an attractive body	BUT	Others feel that they are easily able to keep their bodies looking attractive	☐	☐
9.	☐	☐	Some people feel that their muscles are much stronger than most others of their sex	BUT	Others feel that on the whole their muscles are not quite so strong as most others of their sex	☐	☐
10.	☐	☐	Some people are sometimes not so happy with the way they are or what they can do physically	BUT	Others always feel happy about the kind of person they are physically	☐	☐
11.	☐	☐	Some people are not quite so confident when it comes to taking part in sports activities	BUT	Others are among the most confident when it comes to taking part in sports activities	☐	☐
12.	☐	☐	Some people do not usually have a high level of stamina and fitness	BUT	Others always maintain a high level of stamina and fitness	☐	☐
13.	☐	☐	Some people feel embarrassed by their bodies when it comes to wearing few clothes	BUT	Others do not feel embarrassed by their bodies when it comes wearing few clothes	☐	☐
14.	☐	☐	When it comes to situations requiring strength some people are one of the first to step forward	BUT	When it comes to situations requiring strength some people are one of the last to step forward	☐	☐
15.	☐	☐	When it comes to the physical side of themselves some people do not feel very confident	BUT	Others seem to have a real sense of confidence in the physical side of themselves	☐	☐
16.	☐	☐	Some people feel that they are always one of the best when it comes to joining in sports activities	BUT	Others feel that they are not one of the best when it comes to joining in sports activities	☐	☐

	Really True for Me	Sort of True for Me				Sort of True for Me	Really True for Me
17.	☐	☐	Some people tend to feel a little uneasy in fitness and exercise settings	BUT	Others feel confident and at ease at all times in fitness and exercise settings	☐	☐
18.	☐	☐	Some people feel that they are often admired because their physique or figure is considered attractive	BUT	Others rarely feel that they receive admiration for the way their body looks	☐	☐
19.	☐	☐	Some people tend to lack confidence when it comes to their physical strength	BUT	Others are extremely confident when it comes to their physical strength	☐	☐
20.	☐	☐	Some people always have a really positive feeling about the physical side of themselves	BUT	Others sometimes do not feel positive about the physical side of themselves	☐	☐
21.	☐	☐	Some people are sometimes a little slower than most when it comes to learning new skills in a sports situation	BUT	Others have always seemed to be among the quickest when it comes to learning new sports skills	☐	☐
22.	☐	☐	Some people feel extremely confident about their ability to maintain regular exercise and physical condition	BUT	Others don't feel quite so confident about their ability to maintain regular exercise and physical condition	☐	☐
23.	☐	☐	Some people feel that compared to most, their bodies do not look in the best of shape	BUT	Others feel that compared to most their bodies always look in excellent physical shape	☐	☐
24.	☐	☐	Some people feel that they are very strong and have well developed muscles compared to most people	BUT	Others feel that they are not so strong and their muscles are not very well developed	☐	☐
25.	☐	☐	Some people wish that they could have more respect for their physical selves	BUT	Others always have great respect for their physical selves	☐	☐
26.	☐	☐	Given the chance, some people are always one of the first to join in sports activities	BUT	Other people sometimes hold back and are not usually among the first to join in sports	☐	☐

	Really True for Me	Sort of True for Me				Sort of True for Me	Really True for Me
27.	☐	☐	Some people feel that compared to most they always maintain a high level of physical conditioning	BUT	Others feel that compared to most their level of physical conditioning is not usually so high	☐	☐
28.	☐	☐	Some people are extremely confident about the appearance of their body	BUT	Others are a little self-conscious about the appearance of their bodies	☐	☐
29.	☐	☐	Some people feel that they are not as good as most at dealing with situations requiring physical strength	BUT	Others feel that they are among the best at dealing with situations which require physical strength	☐	☐
30.	☐	☐	Some people feel extremely satisfied with the kind of person they are physically	BUT	Others sometimes feel a little dissatisfied with their physical selves	☐	☐

HOW IMPORTANT ARE THINGS TO YOU?

	Really True for Me	Sort of True for Me				Sort of True for Me	Really True for Me
1.	☐	☐	Some people feel that being good at sports is vitally important to them	BUT	Others feel that being good at sports is not so important to them	☐	☐
2.	☐	☐	Some people do not feel that maintaining a high level of physical conditioning is very important to them	BUT	Others feel that maintaining a high level of physical conditioning is extremely important to them	☐	☐
3.	☐	☐	Some people believe that having an attractive physique or figure is vitally important to them	BUT	Others believe that having an attractive physique or figure is not all that important in their lives	☐	☐
4.	☐	☐	Some people believe that being physically strong is not so important to them	BUT	Others feel that it is extremely important to them to be physically strong	☐	☐
5.	☐	☐	Some people feel that having very good sports ability and skill is not so important to them	BUT	Others feel that having a high level of sports ability is really important to them	☐	☐
6.	☐	☐	Some people feel that maintaining regular vigorous exercise is vitally Important to them	BUT	Others feel that keeping up regular vigorous exercise is not of prime importance to them	☐	☐
7.	☐	☐	Some people do not feel it so important to them to spend a lot of time and effort maintaining an attractive body	BUT	Others think that it is vitally important to spend time and effort maintaining an attractive body	☐	☐
8.	☐	☐	Some people feel that being strong and having well developed/toned muscles is vitally important to them	BUT	Others feel that being strong and having well developed/toned muscles is not so important to them	☐	☐

VITA

Kerrie Lynn Moreau was born in Superior, Wisconsin on October 17, 1971. She attended Central Junior High School and graduated in June 1989 from Superior Senior High School in Superior, Wisconsin. She went on to attend the College of St. Scholastica in Duluth, Minnesota where she received her bachelor of arts in exercise science in August 1993. After taking a year off from her studies, she went on to pursue her masters at Ball State University, Muncie, Indiana where she specialized in adult fitness and cardiac rehabilitation. In May 1996 she graduated from Ball State with a master of science degree in exercise physiology. She then ventured down to Knoxville, Tennesse where she served as a graduate teaching associate for two years in the exercise science unit at the University of Tennessee, Knoxville while pursuing her doctor of philosophy. She graduated in August 1999 with a Ph.D in Education and went on to The University of Colorado, Boulder, to pursue a post-doctoral fellowship in the Human Cardiovascular Research Laboratory / Department of Kinesiology.