

To the Graduate Council:

I am submitting herewith a dissertation written by William John Duey entitled "Racial Comparison of Hemodynamic Responses to Dynamic Exercise and Their Relationship to Muscle Fiber Composition". I have examined the final copy of this dissertation and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctorate of Philosophy, with a major in Education.

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RACIAL COMPARISON OF HEMODYNAMIC RESPONSES
TO DYNAMIC EXERCISE AND THEIR RELATIONSHIP
TO MUSCLE FIBER COMPOSITION

A DISSERTATION
PRESENTED FOR THE
DOCTOR OF PHILOSOPHY
DEGREE
THE UNIVERSITY OF TENNESSEE, KNOXVILLE

WILLIAM JOHN DUEY

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DEDICATION

To my wife, Renee and son, Zachary

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ABSTRACT

The purpose of this study was to examine the relationship between blood pressure responses to dynamic exercise and muscle fiber composition, and compare hemodynamic mechanisms in a biracial group. 30 normotensive, males (15 black, 15 white) completed a dynamic exercise test at 20%, 40%, and 60% of VO_{2peak} and a muscle biopsy. Blood pressure (BP), heart rate (HR), cardiac index (CI), and oxygen uptake (VO_2) were measured during dynamic exercise and systemic vascular resistance (SVR) was calculated. Blacks demonstrated greater systolic ($p = .01$) diastolic ($p = .002$) and mean arterial blood pressure responses ($p = .002$) to dynamic exercise. No racial difference was present for HR or CI; however, SVR ($p = .02$) was higher in the blacks. The percentage of type II fibers was greater in blacks ($p = .05$) and a significant positive correlation ($r = .54$, $p = .003$) was found between the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and the percentage of type II fibers. SVR at 60% of VO_{2peak} was also positively correlated to the percentage of type II fibers ($r = .47$, $p = .01$). The results suggests that racial differences in muscle fiber composition may contribute to the greater blood pressure response and higher SVR values in blacks during dynamic exercise.

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CHAPTER I

INTRODUCTION

The American Heart Association has estimated that over 61 million Americans have high blood pressure (1). A consistent finding in the literature, dating back to 1932, is that American blacks have an increased prevalence of hypertension (2). Recently, the Joint National Committee has reported that the prevalence of hypertension in blacks is 38% compared to 29% in whites (3). Coronary heart disease (4,5) and cerebrovascular disease (6,7) mortality rates are higher in the U.S. black population.

The greater prevalence of hypertension in blacks has been attributed to both genetic and environmental factors (8-11). Specifically, racial differences in sodium sensitivity (12-17), cation transport (18-22), endocrine factors (19,23-27), autonomic nervous system function (28-30), cardiac structure (21,31,32), dietary intake of calcium and potassium (33-35), and socioeconomic stress (36,37) may contribute to the racial disparity in the prevalence of hypertension.

Recently, studies have shown that blacks exhibit exaggerated blood pressure responses to dynamic exercise (38-43). The significance of these results is that exaggerated blood pressure responses to exercise have been shown to successfully predict future hypertension in normotensive

individuals (44-50).

It appears that the exaggerated blood pressure response to exercise in blacks is due to an increased peripheral resistance (43). However, the precise mechanisms responsible have yet to be elucidated. Researchers have speculated that the exaggerated blood pressure response could be due to an enhanced sympathetic drive, an augmented alpha-adrenergic sensitivity, and/or a structural alteration of the peripheral vasculature (51,52). Recently, data collected in our laboratory suggest that young, normotensive blacks have a greater resistance to blood flow than whites when the blood vessels are maximally dilated (53). This lends support to the hypothesis that the peripheral vasculature may be altered in blacks (i.e. fewer or smaller peripheral blood vessels) which could potentially contribute to the development of hypertension.

The purpose of this study was to: 1) evaluate the blood pressure responses to a graded dynamic exercise test in black and white normotensive males 2) assess the systemic hemodynamic mechanisms responsible for any observed racial blood pressure differences (i.e. cardiac output, peripheral resistance) 3) analyze the relationships between blood pressure responses to dynamic exercise, muscle fiber composition, and capillary density.

Hypotheses

Blacks exhibit exaggerated blood pressure responses to a variety of physical stressors (38-43,51,54-59). The following hypotheses will be tested in an attempt to reconfirm the reported racial differences in blood pressure response to dynamic exercise and to elucidate the systemic hemodynamic mechanisms underlying these racial differences. In addition, relationships between blood pressure response to dynamic exercise, fiber type, and capillary density will be analyzed.

Hypothesis 1: Heart rates measured at rest are not different in black and white normotensives.

Statistical analysis: Unpaired Student's t-test (Race)

Hypothesis 2: Systolic and diastolic blood pressures during rest are not different in black and white normotensives.

Statistical analysis: Unpaired Student's t-test (Race)

Hypothesis 3: Systolic blood pressures during a graded dynamic exercise test at 20%, 40%, and 60% of VO_{2peak} are not different in black normotensives compared to white normotensives.

Statistical analysis: Two factor repeated measures ANOVA

(Race * Intensity)

Post hoc analysis: Test of simple main effects

Hypothesis 4: Diastolic blood pressures during a graded dynamic exercise test at 20%, 40%, and 60% of VO_{2peak} are not different in black normotensives compared to white normotensives.

Statistical analysis: Two factor repeated measures ANOVA
(Race * Intensity)

Post hoc analysis: Test of simple main effects

Hypothesis 5: Cardiac indices at rest are not different in black and white normotensives.

Statistical analysis: Unpaired Student's t-test (Race)

Hypothesis 6: Cardiac indices during a graded dynamic exercise test at 20%, 40%, and 60% of VO_{2peak} are not different in black normotensives compared to white normotensives.

Statistical analysis: Two factor repeated measures ANOVA
(Race * Intensity)

Post hoc analysis: Test of simple main effects

Hypothesis 7: Systemic vascular resistance measures at rest are not different in black and white normotensives.

Statistical analysis: Unpaired Student's t-test (Race)

Hypothesis 8: Systemic vascular resistance measured during a graded dynamic exercise test at 20%, 40%, and 60% of VO_{2peak} are not different in black normotensives compared to white normotensives.

Statistical analysis: Two factor repeated measure ANOVA

(Race * Intensity)

Post hoc analysis: Test of simple main effects

Hypothesis 9: The percentage of type II fibers is not different in black and white normotensives.

Statistical analysis: Unpaired Student's t-test (Race)

Hypothesis 10: Capillary densities are not different in black and white normotensives.

Statistical analysis: Unpaired Student's t-test (Race)

Hypothesis 11: Resting blood pressures are positively correlated with the percentage of type II fibers.

Statistical analysis: Linear regression analysis

Hypothesis 12: Blood pressure responses to a graded dynamic exercise test are positively correlated with the percentage of type II fibers.

Statistical analysis: Linear regression analysis

Hypothesis 13: Blood pressure responses to a graded dynamic exercise test are negatively correlated with capillary density.

Statistical analysis: Linear regression analysis

Significance

Numerous investigators have reported that an exaggerated blood pressure response to various physical stressors is predictive of future hypertension (44-50,60-64). Early detection of individuals at a greater risk for developing hypertension (i.e. blacks, borderline hypertensives, and normotensive children of hypertensive parents) would allow for appropriate preventative measures to be taken. This study may contribute to the identification of the mechanisms responsible for an exaggerated blood pressure response during exercise, which could help to clarify the link between this phenomenon and the development of hypertension.

Delimitations of the study

This study is delimited to:

- 1) Thirty, apparently healthy, males (15 blacks and 15 whites) ages 18-30 years
- 2) Participants who are normotensive (defined by a resting supine blood pressures < 140/90 mmHg on three separate occasions)
- 2) Participants who are of average fitness ($40-50 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) and not involved in varsity athletics
- 3) Participants who are not involved in high intensity aerobic training
- 4) Participants who are not involved in weight-lifting workouts on a regular basis

Limitations of the study

This study is limited as follows:

- 1) The subjects will not be randomly selected but recruited via posters, announcements, and "word of mouth".
However, there will be no intentional selection bias.

CHAPTER II

REVIEW OF LITERATURE

The purpose of this literature review will be to: 1) define and classify hypertension 2) outline the primary determinants of blood pressure 3) present prevalence, mortality, and morbidity data related to hypertension 4) discuss factors related to the racial difference in the prevalence of hypertension 5) examine the predictive value of an exaggerated blood pressure response to various physical stressors for the subsequent development of hypertension 6) present studies which examine the blood pressure response to dynamic exercise in blacks and whites 7) examine the relationship between muscle fiber type and blood pressure.

Hypertension

Hypertension is a condition in which an individual's blood pressure is chronically elevated above a level judged to be normal (65). The American Heart Association's definition of hypertension is a systolic blood pressure >140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg (1). The World Health Organization defines normal resting blood pressure as $<140/90$ mmHg and hypertension as $>160/95$ mmHg. Individuals with blood pressure between $140/90$ mmHg and $160/95$ mmHg are classified as borderline or mild hypertensives (66).

Hypertension can be divided into two subgroups: primary hypertension and secondary hypertension (67). Primary hypertension develops without an apparent cause and is commonly referred to as essential or idiopathic hypertension. Approximately 95% of all cases of hypertension are of an unknown cause (68,69). Secondary hypertension is caused by a specific abnormality and accounts for a small percentage of all hypertensive cases. Secondary causes of hypertension include: renal or endocrine dysfunction, coarctation of the aorta, pregnancy-induced hypertension, neurological disorders, and acute stress (67).

Primary determinants of blood pressure

Blood pressure is the pressure exerted by the blood on the wall of any artery during the cardiac cycle (65). Each cardiac cycle has two distinct phases 1) systole, the phase during ventricular contraction and 2) diastole, the phase during ventricular relaxation. Generally, systolic blood pressure is indicative of the force of left ventricular contraction and the compliance of the arteriole bed, and diastolic blood pressure is determined by the resistance to blood flow, pressure at the end of systole, blood volume, and heart rate (70). The two primary determinants of blood pressure are cardiac output and total peripheral resistance. Cardiac output is the flow of blood, usually expressed, in liters per minute, and total peripheral resistance is the

impediment to mean flow through the blood vessels (71).

Prevalence

Hypertension is the most prevalent of the cardiovascular diseases (1). The 1984 Joint National Committee combined and estimated hypertension prevalence figures for adults aged 18-74, institutionalized persons, military personal, elderly populations, and children (3). Their revised prevalence figures are based on data from the National Health and Nutrition Examination Survey II and the Systolic Hypertension in the Elderly Program. Individuals had to meet one of the following criteria in order to be classified as hypertensive: 1) diastolic pressure ≥ 90 mmHg; 2) systolic blood pressure ≥ 140 mmHg; or 3) currently taking antihypertensive medication. Based on 1983 population data, the Joint National Committee estimated that 58 million Americans are at an increased risk of morbidity and premature mortality associated with high blood pressure (3). Most recently, the American Heart Association estimated that over 61 million children and adults have hypertension (1).

A consistent finding in the literature, dating back to 1932, is that American blacks have an increased prevalence of hypertension. Adams (2) reported on the blood pressure readings of 14,000 individuals, 8,000 whites and 6,000 blacks, examined between 1920 and 1930. The blood pressure measurements were a part of a routine pre-employment or annual

physical examination. Systolic blood pressure and diastolic blood pressure were higher at all ages for blacks compared to whites. In addition, the mean systolic blood pressure differences between blacks and whites increased with age.

Comstock (72) conducted the first study in which racial differences in blood pressure were compared in a representative community based sample. The sample consisted of 1,162 individuals who were selected from the 1946 population census data of Muscogee County, Georgia. Mean systolic blood pressure and diastolic blood pressure were higher for blacks compared to whites at all ages, except for systolic blood pressure in black males under 15 years of age.

McDonough et al. (73) examined blood pressure levels in a biracial group residing in Evans County, Georgia. Individuals were grouped by age (10 year age groups, aged 15-74 years), sex, and race. Black males and females exhibited higher systolic and diastolic blood pressures for every age-sex group. In addition, the racial differences in blood pressure increased with advancing age.

More recently, one million persons were screened as part of the Community Hypertension Evaluation Clinic Program (74). Individuals were grouped by age, sex, and race based on their resting blood pressure values. Blacks had higher systolic and diastolic blood pressures compared to whites in all but one age-sex group. In addition, blacks exhibited much greater variability in their blood pressure. Hypertension prevalence

data, based on a diastolic blood pressure of ≥ 90 mmHg, was also obtained. Black males and females age 30 and over had a 43% and 90% higher prevalence, respectively. Prevalence data was also computed using a diastolic blood pressure cutoff of ≥ 95 mmHg and ≥ 110 mmHg. Black males and females age 30 and over had a 78% and 122% greater prevalence with ≥ 95 mmHg as the cutoff and 192% and 253% with ≥ 110 mmHg.

The Hypertension Detection and Follow-up Program Cooperative Group (75) assessed blood pressure by age, sex, and race in 14 communities. 158,906 individuals, aged 30 to 69, were examined to identify persons with an elevated diastolic blood pressure, i.e., ≥ 95 mmHg. Consecutive casual seated blood pressure readings were taken. If the participant's mean diastolic blood pressure was ≥ 95 mmHg or if antihypertensive medication was currently being taken, the individual was classified as being hypertensive. Diastolic blood pressures were higher for blacks in each of the age-sex groups. Blacks exhibited a higher prevalence, 37%, compared to whites, 18%. Severe hypertension, defined as a diastolic blood pressure of ≥ 115 mmHg, was approximately 6 times greater in blacks.

The 1984 Joint National Committee reported revised hypertension prevalence figures based on the results of the 1976-1980 National Health and Nutrition Examination Survey (3). Mean systolic and diastolic blood pressures were grouped by age, sex, and race. Hypertension was defined as a systolic

blood pressure of ≥ 140 mmHg and/or a diastolic blood pressure of ≥ 90 mmHg. The prevalence of hypertension in blacks was 38.2% compared to whites 28.8%. In addition, blacks exhibited two times the rate of moderate hypertension and severe hypertension was three times greater in blacks.

Most recently, the National Health and Epidemiologic Follow-up Study (76) examined a cohort of 14,235 individuals who had participated in the National Health and Nutrition Examination Survey. Several analysis were performed on this cohort group that were followed up for approximately 9.5 years. Of particular interest was an analysis of participants that had normal blood pressure levels at baseline and became hypertensive at follow-up. The incidence, the number of new cases of definite hypertension (defined as a systolic blood pressure ≥ 160 mmHg and/or a diastolic blood pressure of ≥ 95 mmHg) was categorized by sex, race, and age. Twenty-three percent of the black men and 22% of the black women had developed definite hypertension at follow-up compared to 12% and 10% of the white men and women, respectively.

Morbidity and mortality

Cardiovascular disease accounted for 45% of the total annual mortality in 1988 (1). Cardiovascular diseases include: coronary artery disease, hypertension, stroke, congestive heart failure, peripheral vascular disease, congenital heart defects, valvular heart disease, and

rheumatic heart disease (77). Hypertension is the most prevalent of the cardiovascular diseases (1). The following literature suggest that morbidity and mortality rates related to hypertension are elevated in the black population.

Stone and Vanzant examined 915 patients who presented definite evidence of heart disease between 1920 and 1926 (78). Heart disease was reportedly 1.8 times more common in blacks compared to whites. All cases were classified according to etiology and 436 of the 915 patients (47.7%) were hypertensive. Hypertension was approximately two times more frequent in blacks. Age group data revealed that blacks had a greater frequency of hypertension between the ages of 40 and 50 years and whites exhibited a greater frequency between the ages of 60 and 70 years.

The Medical College of South Carolina performed 2,066 consecutive autopsies in order to study the distribution of disease in the South (79). Twenty-five percent of the cases were directly or indirectly the result of cardiovascular disease. The relative incidence of various cardiovascular disease was grouped by race and sex. Black males and females exhibited a greater incidence of hypertensive vascular disease, 63% and 52%, respectively, compared to white males, 34%, and females, 33%. In addition, blacks died at a much earlier age than did the whites.

Stamler et al. (80) reported the cardiovascular-renal mortality rates of Chicago residents aged 25-64 years in 1951.

Specifically, death rates for hypertensive disease and hypertensive heart disease were categorized by age (10 year age groups), race, and sex. Blacks exhibited higher death rates due to hypertensive disease for every age group. The ratio of black to white death rates ranged from a minimum of 2.6 to more than 10.0.

McDonough et al. (73) examined racial differences in hypertensive disease among blacks and whites in Evans County, Georgia. Left ventricular hypertrophy and cardiac enlargement were significantly elevated in the blacks.

More recently, the Multiple Risk Factor Intervention Trial (81) screening program examined all-cause and cause-specific mortality after five years among 23,490 black males and 325,384 white males. All cause mortality was higher for blacks compared to whites. Cause-specific mortality data revealed that the death rates from cerebrovascular disease were almost three times higher for black males.

Neaton et al. (6) followed participants in the MRFIT screening program an additional five years and reported the following: 1) coronary heart disease death rates were lower for black hypertensives (DBP ≥ 90 mmHg) compared to white hypertensives, 2) coronary heart disease death rates were higher for black normotensives compared to white normotensives, and 3) both normotensive and hypertensive blacks had higher death rates for cerebrovascular disease compared to white normotensives and hypertensives.

Factors related to racial differences in hypertension

Skin color

Although it is difficult to quantify, investigators have suggested that genetics may be responsible for the racial differences in prevalence of hypertension. A study conducted in Charleston, South Carolina utilized skin color as a genetic marker to study black and white differences (82). Initially, it appeared that darker skin was associated with higher blood pressures; however, when controlling for social class the relationship did not exist (83).

Renal physiology/sodium

Epidemiological studies have reported a similar or lower dietary intake of sodium in blacks compared to whites (33,34). However, the literature strongly supports the contention that blacks are more salt sensitive than whites (12-17). Salt sensitivity is characterized by a reduced ability to excrete a sodium load. Luft et al. (12) examined racial differences in urinary excretion of sodium after volume expansion. Black males excreted less sodium (328 mEq) than whites (361 mEq) over a 24-hour period. The delay in sodium excretion persisted for 12-hours following sodium loading. Additionally, Weinberger et al. (14) reported a greater prevalence of sodium sensitivity in both normotensive and hypertensive blacks. The blacks who exhibited an increase in blood pressure following a sodium load also demonstrated a

delay in sodium excretion.

The release of renin by the juxtaglomerular cells of the kidney will result in an eventual increase in blood pressure. Therefore, it is possible that racial differences in blood pressure may be related to elevated renin levels in blacks. However, it has been reported that normotensive blacks have lower plasma renin level than whites (19,84). In addition, Wisenbaugh et al. (85) reported that 36-62% of black hypertensives had suppressed plasma renin activities as compared to 19-55% of the white hypertensives.

Cation transport

Canessa et al. (18) demonstrated that the sodium-potassium-chloride cotransport mechanism is less active in hypertensive blacks and normotensive blacks with a parental history of hypertension. Generally, this transport mechanism is present in red blood cells, and epithelial cells of the kidney. This decreased sodium transport has been used as a predictor of sodium sensitivity.

Cardiac structure

Dunn et al. (32) examined cardiac structure in 30 black and 30 white patients with essential hypertension. Left ventricular wall thickness measurements were not different; however, blacks had a greater left ventricular mass index. In addition, blacks exhibited greater left ventricular

hypertrophy in the Evans County, Georgia Study (73) and the Hypertension Detection and Follow-up Program (86).

Dietary factors

A greater daily intake of sodium could theoretically contribute to the racial disparity in the prevalence of hypertension. However, studies analyzing dietary intake and 24-hour urinary sodium excretion have reported no racial differences (33,87-90). Additionally, it has been reported that the dietary intake of calcium and potassium is lower in blacks (33-35). The significance of this finding is that the ratio of urinary sodium-to-potassium excretion has been positively correlated to blood pressure (35).

Blood pressure response as a predictor of hypertension

Researchers have hypothesized that an exaggerated blood pressure response to stress is a marker for the subsequent development of hypertension (91). In 1921, K. Fahrenkamp (77) suggested that persons who develop hypertension have a prehypertensive condition that may be manifested by transient increases in blood pressure. Longitudinal studies examining blood pressure responses have suggested that an exaggerated blood pressure response to the cold pressor test (61,62,64,92,93), static (60,61), and dynamic exercise (44-49,60) is predictive of future hypertension. The

following sections will outline the various studies that have examined blood pressure responses to stress as a predictor of hypertension.

Cold pressor

Hines and Brown (94) developed the cold pressor test for measuring blood pressure responses in normotensive and hypertensive individuals. Follow-up studies (95) on the original cohort prompted Hines to conclude the following: 1) the cold pressor test is an index of vascular reactivity, 2) vascular hyperactivity, as measured by the cold pressor test, is an important etiologic factor in essential hypertension, 3) the vascular hyperactivity of normotensive individuals represents an antecedent or latent phase of hypertension, and 4) 38% of the original normotensive hyperreactors had developed hypertension within 6 years, whereas none of the normal hyporeactors had developed hypertension.

In 1961, Barnett et al. (62) examined 207 individuals, ages 33 to 46 years, who had a cold pressor test in 1934 (96). Forty of 207 subjects were classified as hyperreactors and 167 of 207 were classified as normoreactors based on their blood pressure response to the cold pressor test. The current blood pressures of the subjects were taken from their personal physician or by blood pressure readings obtained at the Mayo Clinic. After 27 years, hypertension had developed in 4 of 40 hyperreactors and in none of the 167 normoreactors. Barnett

concluded from these results that "vascular hyperactivity is an antecedent to rather than a result of hypertension."

Recently, Wood et al. (63) reported the results of a 45 year follow-up study of 142 subjects who were studied by Hines in 1937 (96) and Barnett et al. (62) in 1961. Subjects had been classified as normoreactors or hyperreactors to the cold pressor test based on their maximal blood pressure responses recorded in 1934 and 1961. Hypertension, defined as a blood pressure, $> 140/90$ mmHg, developed in 34 of 48 (71%) of the hyperreactors and 18 of 94 (19%) of the normoreactors. Wood concluded that the cold pressor test is a potential predictor of future hypertension.

Menkest et al. (64) examined cardiovascular reactivity to the cold pressor test in 910 white medical students between 1948 and 1964. Subjects were subdivided into three groups based on their systolic blood pressure response to the cold pressor test (low ≤ 6 mmHg, moderate 7-17 mmHg, and high ≥ 18 mmHg). The incidence rates of hypertension at 44 years of age were 2.4%, 3.0%, and 6.7% for the low, moderate, and high group, respectively. This association persisted after adjusting for entry age, quetelet index, cigarette smoking, pretest systolic blood pressure, and parental history of hypertension.

The association between blood pressure responses to the cold pressor task and future blood pressure levels in children were analyzed as part of the Bogulusa Heart Study (61).

Simple correlations, adjusting for initial resting blood pressure, showed that the maximal blood pressure response to the cold pressor test was correlated with resting systolic blood pressure ($r = .53$) measured during the fourth year of the study and resting diastolic blood pressure ($r = .31$).

The five studies cited above concluded that the cold pressor test is a useful predictor of subsequent hypertension. However, four studies have shown no relationship between the blood pressure response to the cold pressor test and the subsequent development of hypertension. Harlan et al. (97) examined a group of 385 naval flight students. The subjects performed a cold pressor test and were classified as hyporeactors or hyperreactors. Ninety-six percent of the original cohort participated in a follow-up study 18 years later. No relationships were observed between the blood pressure response to the cold pressor test and the subsequent blood pressure level or the development of hypertension.

Eich et al. (98) examined vascular reactivity to the cold pressor test as a predictor of hypertension in medical students. One hundred and seventy three students performed a cold pressor test and were classified a hypo- or hyperreactors. The authors concluded, based on the 9-10 year follow-up data, that the blood pressure response to the cold pressor test did not accurately predict future resting blood pressure levels.

Thomas and Duszynski (99) examined the fate of the cold pressor test as a predictor of hypertension in 78 male medical students who had developed hypertension. Statistically, no relationships were found between the response to the cold pressor test and the subsequent development of hypertension.

A longitudinal study conducted by Thomas et al. (100) investigated the cold pressor test as a predictor of hypertension in black physicians. Three hundred and fifteen participants performed a cold pressor test. After 23 and 30 years, the participants were reexamined and a resting blood pressure level was obtained. At this time subjects were classified as normotensive, defined as a blood pressure <140/90 mmHg, or hypertensive. Statistical analysis performed on the data revealed no relationship between the blood pressure response to the original cold pressor test and follow-up blood pressure levels. The authors concluded that the cold pressor test had no value in predicting the future occurrence of essential hypertension

Static exercise

Parker et al. (61) examined the association between the blood pressure response to isometric handgrip exercise and future resting blood pressure levels in children. Simple correlations between blood pressure response to isometric handgrip exercise were correlated to the systolic ($r = .60$) and diastolic ($r = .45$) resting blood pressures taken during

the fourth year of the study.

Chaney and Eyman (60) examined prospectively 100 office normotensive patients who had blood pressure measurements taken during maximal isometric exercise. Sixteen patients developed hypertension within a 14 year follow-up period and 84 patients remained normotensive. A multivariate statistical analysis revealed that the highest diastolic blood pressure recorded correctly classified 69% of the hypertensives and 73% of the normotensives. The highest systolic blood pressure measured correctly classified 63% of the hypertensives and 73% of the normotensives. The authors concluded that transient blood pressure increases during maximal isometric exercise can be predictive of future hypertension.

Dynamic exercise

Dahms et al. (48) investigated the prognostic value of combined rest-exercise blood pressures patterns. Two hundred and thirty men aged 25-65 were separated into four groups: 1) normotensive rest/normotensive exercise, 2) hypertensive rest/normotensive exercise, 3) hypertensive rest/hypertensive exercise, and 4) normotensive rest/hypertensive exercise. A five year follow up revealed that 27 of 30 hypertensive rest/normotensive exercise men were now normotensive at rest and 19/26 normotensive rest/hypertensive exercise men were now hypertensive at rest. The authors concluded that blood pressures taken during a maximal treadmill exercise test can

identify individuals who are predisposed to hypertension.

Wilson et al. (44) evaluated the early prediction of hypertension using blood pressure measures taken during maximal treadmill exercise in 3,395 men and 425 women. A normal resting blood pressure was defined as $<140/90$ mmHg and an exaggerated exercise blood pressure response was defined as $\geq 225/90$ mmHg. Individuals with a normal resting blood pressure and a high exercise blood pressure had a relative risk of 2.28 for the subsequent development of hypertension. The relative risk for women (3.3) exceeded that of men (2.17). Wilson concluded that exercise blood pressures can be used as a means for earlier identification of persons who are prone to hypertension.

Davidoff (49) investigated postexercise blood pressure as a predictor of hypertension. Seven hundred and twenty-one healthy males performed a cycle ergometer exercise test at 70% of their maximal predictive heart rate. A positive blood pressure response to exercise was defined as a systolic blood pressure >200 mmHg or a raised diastolic blood pressure taken 30 seconds post exercise. 236 of 721 participants became hypertensive, e.g., resting blood pressure $>149/90$ mmHg, during the follow-up which ranged from 12-170 months. Of the 236 individuals who became hypertensive, 17% exhibited a positive systolic response and 17% exhibited a positive diastolic response. Of the 485 individuals who remained normotensive, 5% exhibited a positive systolic response and

12% exhibited a positive diastolic response.

Dlin et al. (47) investigated 75 individuals who were normotensive at rest and displayed an exaggerated blood pressure response to exercise and compared them with a matched control group. An exaggerated exercise blood pressure response was defined as a systolic blood pressure ≥ 200 mmHg and/or a diastolic blood pressure increase of 10 mmHg or a diastolic blood pressure of >90 mmHg during a submaximal cycle ergometer test. Individuals were followed-up for an average of 5.8 years. Eight of the subjects with an exaggerated blood pressure response to exercise were found to be hypertensive, e.g., resting blood pressure $\geq 140/90$ mmHg, and none of the control group were hypertensive. Of all the other parameters reviewed in this study (i.e. family history, resting heart rate, resting systolic and diastolic blood pressure, fitness level, age, smoking) exercise blood pressure was the best predictor of future hypertension.

Jackson et al. (46) examined 4,856 patients to determine whether exercise hypertension was predictive of future resting hypertension. Exercise hypertension was defined as a systolic blood pressure of ≥ 230 mmHg and/or a diastolic blood pressure of ≥ 110 mmHg during a treadmill or cycle ergometer test to 90% of age-predicted maximal heart rate. Fifty-three patients were found to be normotensive at rest but hypertensive during exercise. Twenty-three of 53 patients who exhibited an exaggerated blood pressure response to exercise returned to

the clinic and 12 of 23 (51%) had developed hypertension. A control group who were normotensive at rest and exercise were matched for age. 14 of 91 patients (15%) were found to be hypertensive upon follow-up. These results support the hypothesis that elevated blood pressure during exercise can be useful predictor of future hypertension.

Chaney and Eyman (60) examined 100 patients who had their blood pressure taken during a maximal treadmill test utilizing a modified Ellestad protocol. Sixteen patients developed hypertension within a 14 year follow-up period and 84 remained normotensive. A multivariate statistical analysis revealed that maximal treadmill diastolic blood pressures correctly identified 81% of the hypertensives and 68% of the normotensives. Maximal treadmill systolic blood pressures correctly identified 63% of the hypertensives and 73% of the normotensives.

Tanji et al. (45) examined the usefulness of blood pressure recovery curves following submaximal exercise as a predictor of hypertension. Twenty six males performed a Harvard Step Test to differentiate between a hypertensive and normotensive response to exercise. Blood pressure was taken immediately post exercise and every 20 seconds for a period of three minutes. A hypertensive response to exercise was defined as a systolic blood pressure >200 mmHg immediately post exercise or a systolic blood pressure of >135 mmHg at three minutes post exercise. Nine of the 10 individuals who

demonstrated a hypertensive response to exercise were diagnosed as hypertensive at the ten-year follow-up. Eleven of 12 individuals who demonstrated a normotensive response to exercise remained normotensive.

Racial differences in blood pressure response to exercise

Currently, eight studies have investigated blood pressure responses to dynamic exercise in blacks and whites. Specifically, Table 1 outlines 6 studies which investigated the effect of race on the blood pressure response to exercise in normotensive adults. Alpert et al. (38) examined blood pressure responses to dynamic exercise in healthy black and white children. Four hundred and five children, 184 black and 221 white, performed a continuous graded maximal cycle ergometer stress test. Systolic blood pressure was measured at every stage and immediately following exercise. The highest blood pressure measured was defined as peak systolic blood pressure. No racial differences were reported for resting systolic or diastolic blood pressure. Peak exercise blood pressures were higher in black females compared to white females when grouped by body surface area. Black males had higher exercise blood pressure than white males when classified by age and body surface area.

Hohn et al. (87) examined 141 children aged 10-17 years. The children were grouped by race and parental history of hypertension. Each child performed an exercise stress test on

Table 1. Effect of Race on Blood Pressure Response to Exercise in Normotensive Adults

Source	Stressor	Subjects	Gender	SBP	DBP
Duey (55)	IHG 30%MVC 3 minutes	15 BL 15 WH	M	ND	^BL
McAdoo (54)	IHG 30%MVC 2 minutes	47 BL 70 WH	M&F	ND	^BL
Ekelund (39)	Treadmill	83 BL 2548 WH	M	^BL	---
Thomas (40)	Double Masters	433 BL 573 WH	M&F	^BL	--- *
Tischenkel (42)	Cycle ergometer 65% age adjusted heart rate	44 BL 39 WH	M&F	^BL	--- **
Walker (43)	Cycle ergometer Graded test	15 BL 15 WH	M	^BL	^BL

Abbreviations: SBP (systolic blood pressure), DBP diastolic blood pressure, IHG (isometric hand grip), MVC (maximal voluntary contraction), BL (black), WH (White), M (male), F (female), ND (no difference)

* Peak pressure (SBP * HR) was compared

** Black females only exhibited a greater SBP

a treadmill. The protocol consisted of walking at a constant rate (3.5 mph) with the grade increasing 2% each minute. Systolic and diastolic blood pressures were significantly higher in the black children with a parental history of hypertension compared to white children with a positive parental history. However, no difference were reported between black and white children without a parental history of hypertension.

Thomas et al. (40) measured blood pressure responses in 433 black medical students and 573 white medical students as part of the Meharry Cohort study. Resting systolic and diastolic blood pressures were higher in black males compared to white males. Black females had higher resting systolic blood pressures compared to white females with no reported difference in resting diastolic blood pressure. Maximum pulse pressure during exercise was significantly higher in black males and females compared to white males and females.

Arensman et al. (52) investigated hemodynamic variables during a maximal supine graded exercise test in a biracial group of 10 year old boys. Blood pressure, cardiac output, and systemic vascular resistance were measured in 19 blacks and 31 whites. No racial differences were observed in preexercise, maximum, or postexercise blood pressures. The white males had higher cardiac indexes during preexercise, maximum exercise, and recovery. Conversely, black males exhibited higher systemic vascular resistance during

preexercise, maximum exercise, and postexercise. It was suggested by the authors that the greater systemic vascular resistance observed in blacks may serve as a potential marker for the subsequent development of hypertension.

Treiber et al. (41) reported racial differences in the blood pressure response to maximal dynamic exercise for children aged 4 to 6 years. Seventy-five children (51 white and 24 black) performed a maximal graded exercise treadmill test with blood pressures and heart rates measured prior to exercise, every minute during exercise, and for 5 minutes of recovery. Black children had significantly lower preexercise and peak exercise heart rates and a significantly higher peak systolic blood pressure response to the exercise bout. No racial differences were reported for diastolic blood pressure or heart rate during the treadmill test.

Tischenkel et al. (42) examined blood pressure reactivity to submaximal exercise as a function of race and sex. Each participant rode a cycle ergometer for a total of 6 minutes. Two minutes were allowed to increase the heart rate to the specified level (65% of age-adjusted heart rate) and that workload was maintained for the remaining 4 minutes. Systolic blood pressure was measured every minute and the highest blood pressure was recorded. Diastolic blood pressures were not analyzed because the instrument that was used was not reliable during exercise. Black women had the greatest systolic blood pressure response (171 mmHg), followed by white men (150

mmHg), black men (141 mmHg), and white women (134 mmHg).

Racial differences in the blood pressure response to dynamic exercise were investigated as part of the Lipid Research Clinics Program Prevalence Study (39). Eighty-three black men and 2,548 white men and 113 black women and 1,1519 white women aged 20-69 year participated in the study. Resting blood pressures and blood pressure responses to a modified Bruce protocol treadmill test were obtained. There were no significant differences in resting systolic and diastolic blood pressures between black and white men. However, resting diastolic blood pressures were significantly higher in black women compared to white women. Systolic blood pressures were significantly greater in black men (174 mmHg) during stage 2 of the treadmill test compared to white men (166 mmHg). This racial difference persisted after controlling for a number of baseline characteristics (i.e. age, weight, resting systolic blood pressure, smoking, low density lipoprotein cholesterol, physical activity, and alcohol intake). Exercise blood pressures were not significantly different for black and white women.

Walker et al. (43) investigated the hemodynamic responses to dynamic exercise in a group of normotensive college-age blacks and whites. All subjects performed graded exercise on a cycle ergometer at 25, 50, and 75% of VO_2 peak. Blood pressure and cardiac output were measured at each stage. Blacks exhibited greater systolic and diastolic blood pressure

responses at 50 and 75% of VO_{2peak} . In addition, blacks had an elevated systemic vascular resistance while whites had higher cardiac index values, across all conditions.

Muscle fiber types and distribution

Human skeletal muscle is comprised of a mixture of two fiber types: type I and type II fibers. Type II fibers can be further subdivided into type IIa and type IIb. A number of histological techniques have been used to distinguish between skeletal muscle fiber types. Consequently, several different classifications have been used to differentiate between fiber types. Table 2 provides a summary of the more frequently used nomenclature to designate between muscle fiber types (101).

Type I and type II human skeletal muscle fibers are metabolically and physiologically distinct. Table 3 outlines some of the characteristics of type I and type II fibers. Recently, investigators have reported on the muscle fiber type composition of human skeletal muscle. Thorstensson et al. (102) examined 10 sedentary men and reported a mean value of 56% for type II fibers. Gollnick et al. (103) biopsied 14 men and reported a mean value of 55% type II fibers. Saltin (101) biopsied 45 females and 70 males and reported a normal distribution with a mean of 50% type II fibers. Hedberg et al. (104) biopsied 115 young men and women aged 16 years. The mean percentage of type II fibers was 48%.

Table 2. Muscle Fiber Type Nomenclature

Type I	Type IIa	Type IIb
Slow twitch (ST)	Fast Twitch (FTa)	Fast Twitch (FTb)
Red	Intermediate	White
High Oxidative	Oxidative	Low oxidative
Low Glycolytic	Glycolytic	High Glycolytic

Table 3. Characteristics of Type I and Type II Muscle Fibers

Characteristic	Type I	Type II
Force per cross-sectional area	low	high
Myofibrillar ATPase activity	low	high
Intramuscular ATP stores	low	high
Intramuscular PC stores	low	high
Contraction speed	slow	fast
Relaxation time	slow	fast
Glycolytic enzyme activity	low	high
Endurance	high	low
Intramuscular glycogen stores	no difference	
Intramuscular triglyceride stores	high	low
Myoglobin content	high	low
Aerobic enzyme activity	high	low
Capillary density	high	low
Mitochondrial density	high	low

An important physiological distinction between type I and type II fibers is their degree of capillarization. Andersen (105) reported that type I fibers have a greater capillary/muscle fiber ratio than type II fibers. Additionally, Nygaard and Goricke (106) reported a 4.2 capillary per fiber ratio for type I fibers and a 3.2 capillary per fiber ratio for type II fibers in sedentary men. The difference is greater for endurance trained men 5.9 per type I fibers and 4.3 for type II fibers.

Recently, Ama et al. (107) examined muscle fiber type composition in sedentary black and white males. Twenty-three black male African students and 23 white males were matched on the basis of age, height, body weight, and body mass index. Muscle biopsies were obtained from the vastus lateralis muscle. The results indicated that whites had a higher percentage of type I fibers 41% compared to blacks 33%. In addition, whites had a lower percentage of type IIa fibers 42% compared to blacks 49%.

Muscle fiber type and resting blood pressure levels

A relatively unexplored phenomenon is the relationship between muscle fiber type and resting blood pressure. In 1979, Juhlin-Dannfelt et al. (108) performed muscle biopsies in the vastus lateralis muscle of 22 normotensives and 19 untreated hypertensives. Resting blood pressures were determined by auscultation and intra-arterial measures. The

percentage of type I fibers was significantly lower in the hypertensive group (35%) compared to the normotensive group, (40%). Consequently, a significant positive correlation ($r = .68$ hypertensives , $r = .69$ normotensives) was found between the percentage of type II fibers and resting blood pressure levels.

Frisk-Holmberg et al. (109) demonstrated a positive relationship between fiber type composition and resting blood pressure in 17 hypertensives and 17 age and sex matched controls. Mean resting blood pressure for the hypertensives was 156/105 mmHg compared to a mean resting blood pressure of 124/83 for the normotensives. The percentage of type II fibers were higher for hypertensives, 52%, compared to normotensives, 43%. A significant positive correlation was reported between the percentage of type II fibers and mean arterial blood pressure in the supine position for hypertensives ($r = .68$) and normotensives ($r = .47$).

Recently, Henrich et al. (110) examined capillary rarefaction in 12 normotensives and 15 hypertensives. Muscle biopsy samples were obtained from autopsy specimens of the vastus lateralis and the pectoralis major muscles. The capillary density (capillaries per 2.5 mm^2) of the vastus lateralis muscles was 442 in normotensives compared to 277 in hypertensives. The pectoralis major muscle had a capillary density of 477 in normotensives and 232 in hypertensives. Rarefaction of the capillaries in the quadriceps and

pectoralis major muscle was 37% and 51%, respectively.

Muscle fiber type and the exaggerated blood pressure response to exercise

Two studies have examined the relationship between muscle fiber type and the blood pressure response to exercise (109,111). Frisk-Holmberg et al. (109) investigated muscle fiber composition and blood pressure responses to isometric exercise. Seventeen hypertensive and 17 normotensive individuals performed isometric exercise at 33% of their maximal voluntary contraction for 60 seconds. Blood pressure was measured via auscultation before, during, and for three minutes after exercise. A muscle biopsy was performed on the vastus lateralis muscle the day after the isometric exercise test. The results revealed a significant positive correlation between the maximal change in mean arterial blood pressure and the percentage of fast twitch fibers for hypertensive ($r = .62$) and normotensive ($r = .47$) subjects. In addition, it was noted that the hypertensives with the highest percentage of fast twitch fibers, >55%, displayed the greatest maximal change in blood pressure during isometric exercise.

Fitton (111) indirectly examined the relationship between blood pressure responses to dynamic and static exercise and muscle fiber composition. Nine sprinters and 9 distance runners performed a submaximal cycle ergometer exercise test and a static handgrip exercise test. It has been well

documented that these two groups have divergent populations of muscle fibers (102,103,112). The results of the investigation revealed that male sprinters exhibited a greater systolic and diastolic blood pressure response to static exercise. In addition, the diastolic blood pressure response was significantly greater in sprinters during dynamic exercise. The author speculated that muscle fiber type appears to be closely linked to the blood pressure response to exercise.

CHAPTER III

METHODS

Subjects

Thirty healthy male university students (15 blacks, 15 whites) were recruited to participate in this study. The subjects were screened for normal resting blood pressure (resting supine BP <140/90 mmHg on three separate occasions). A health history questionnaire and an informed consent form were obtained at the initial screening. Following 20 minutes of supine rest, systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were measured using a Colin STPD 780 automated blood pressure monitor (Colin Medical Instruments, Plainfield, NJ). The Colin monitor has previously been validated against auscultation and intra-arterial recordings during rest and exercise (113). Height, weight, and a three-site skinfold measurement (chest, tricep, and subscapular) were also obtained (114).

Peak oxygen uptake ($\text{VO}_{2\text{peak}}$) test

Each subject performed a maximal graded dynamic exercise test on a cycle ergometer. The subject began exercising at 30 watts, and the work rate increased by 30 watts each minute until the subject could no longer maintain the pedal cadence. Expired oxygen and carbon dioxide were measured throughout the graded exercise test. A computerized system connected to

Applied Electrochemistry S3-A O₂ and CD-3A CO₂ analyzers provided minute by minute gas analysis during the test. The analyzers were calibrated against known gases analyzed by the Scholander technique (115). Oxygen uptake and carbon dioxide production were calculated using the Haldane transformation of the Fick equation.

Dynamic exercise

The subject reported to the laboratory in the early morning, following an overnight fast. After 20 minutes of supine rest, baseline blood pressures and heart rates were taken. The subject was then positioned on the cycle ergometer. The dynamic exercise protocol began with 6 minutes of seated rest. The subject's oxygen uptake and carbon dioxide production were measured at 60-second intervals and the values obtained at minutes 4 and 5 were averaged and recorded as the steady state value. At minute 5, cardiac output was measured and completed by minute 6. The cycle ergometer was then set at 20%, 40%, and 60% of the subject's $\text{VO}_{2\text{peak}}$ and the protocol described above was repeated.

Cardiac output was measured non-invasively by the CO₂-rebreathing technique (116). End-tidal pCO₂ was measured through the use of a rapid-response infrared CO₂ analyzer. The 10 end-tidal pCO₂ values prior to a rebreathing trial were averaged, and this value was used to estimate arterial pCO₂ according to the formula of Jones et al. (117).

The subject then rebreathed into a latex bag containing CO_2 and O_2 to permit rapid equilibration with venous pCO_2 . A valid equilibrium during rebreathing was determined by a "plateau" in pCO_2 according to the criteria of Aston and McHardy (118) and Jones et al. (119). This requires that during the first 6-8 seconds there is an inspiration followed by an expiration in which the pCO_2 values are within ± 1 mmHg of each other. In most cases, a plateau was obtained without having to repeat a trial. The "downstream" correction factor of Jones et al. (120) was employed. An automated gas mixing apparatus was used to adjust the initial gas volume and initial pCO_2 in the rebreathing bag, according to the procedures described by Jones (120). If a trial had to be repeated, the end-tidal pCO_2 value was allowed to return to the baseline value before redoing the procedure.

Arterial and venous CO_2 content (CaCO_2 and CvCO_2) were determined using a standard dissociation curve. Cardiac output was calculated from the indirect Fick equation:

$$\text{Cardiac output} = \text{VCO}_2 / (\text{CaCO}_2 - \text{CvCO}_2)$$

Cardiac output values were corrected for body surface area (BSA) by the method of Dubois (121) and expressed as cardiac index (CI) ($\text{L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$). Systemic vascular resistance (SVR) was calculated from the formula: $\text{SVR} = \text{MAP} / \text{CI}$, where SVR is expressed as units, MAP as mmHg, and CI as ($\text{L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$).

Muscle biopsy procedure

The muscle sample was taken from the vastus lateralis with the subject in the supine position. The biopsy site was located 18-20 cm above the patella, 5-6 cm lateral to the midline of the thigh, and 2 cm from the epimysium.

The sampling site was prepared by removing all hair from the skin and surgically cleaning the surrounding area. A local anaesthetic (1.5 cc - xylocaine) was injected near the sampling site.

A scalpel was used to make a 6-7 mm incision through the skin and fascia. Before the biopsy sample was taken the subject was instructed to relax the vastus lateralis muscle. The biopsy sample was then taken via the procedure of Bergstrom (122).

After the biopsy needle was withdrawn, direct pressure was applied to the sampling site. Once the bleeding had stopped, the incision was closed with sterile closure strips and the subject was instructed not to participate in vigorous physical activity for three days. On the fifth day following the biopsy the subject returned to the laboratory to insure that the sampling site was free of infection.

Storage of the muscle sample

Once the muscle sample was removed, it was placed on a moistened sponge. The sample was then placed under a dissecting microscope for proper orientation. Cork sections

containing gum tragacanth (Sigma # G-1128) were used for mounting the sample. After determining the direction of the fibers in the muscle sample, it was carefully placed on the cork section. A stainless steel beaker containing approximately 100 ml of isopentane was immersed in a styrofoam thermos containing liquid nitrogen. The cork section was then immersed, using long forceps, in the isopentane for 15 seconds. The sample was then placed in a plastic bag and was tightly sealed and stored at -74°C until sectioning.

Staining and quantification of fiber types and capillaries

Muscle fiber type composition was determined by the myosin ATPase staining procedure (123). A complete and detailed description of the procedure can be found in Appendix C. A five minute preincubation at a pH of 4.54 was used to delineate between the different fiber types. Type I fibers (slow twitch) stained dark, type IIa (fast twitch - oxidative) stained white, and type IIb (fast twitch glycolytic) stained grey. All stained samples were photographically reproduced to facilitate counting. For each muscle specimen, 200 - 400 fibers were classified and the percentage was calculated.

Capillary density was determined by the periodic acid - Schiff's stain (124). A complete and detailed description of the procedure can be found in Appendix D. Initially, the muscle sections were incubated in a 1% amylase solution for five minutes at 37°C for the removal of glycogen.

Subsequently, the slides were placed in periodic acid followed by fifteen minutes in Schiff's reagent. The Schiff's reagent stained the mucopolysaccharides in the capillary walls a bright pink color. All stained samples were photographically reproduced at a known magnification (10X objective). A .5mm X .5mm square was placed on the photograph and all capillaries within the square were counted. This number was multiplied by four to determine the capillary density (capillaries/mm²).

Statistical analyses

Means and standard errors were calculated for all the variables measured. An unpaired Student's t-test was used to compare the subject's physical characteristics (age, height, weight, body mass index, body surface area, sum of skinfolds, resting heart rate, resting systolic, diastolic, and mean arterial pressure).

An unpaired Student's t-test was used to analyze racial differences for SBP, DBP, MAP, and HR during supine and seated rest. A two-way repeated measures ANOVA (Race * Intensity) was used to analyze the cardiovascular variables (SBP, DBP, MAP, HR, CI, SVR) during the graded exercise test.

Relationships between muscle fiber composition, capillary density, resting blood pressure, and the blood pressure response to exercise were analyzed via linear regression analyses.

CHAPTER IV

RESULTS

Descriptive and physical characteristics

The descriptive and physical characteristics of the thirty subjects, fifteen blacks and fifteen whites, are presented in Table 4. An unpaired Student's t-test revealed significant differences for age, whites were older (22.9 ± 0.6 yrs) than blacks (19.4 ± 0.03 yrs), and weight, whites weighed more (82.5 ± 2.9 kg) than blacks (75.0 ± 1.6 kg). No significant differences were found between the group means for height, body mass index (BMI), body surface area (BSA), sum of skinfolds, resting heart rate, resting systolic, diastolic, or mean arterial blood pressure.

Peak oxygen uptake (VO_{2peak}) test

Group means for the data obtained during the VO_{2peak} test are presented in Table 5. A significant difference was found for absolute VO_{2peak} ($L \cdot min^{-1}$), whites were higher ($3.6 \pm .15$) than blacks ($3.2 \pm .09$); however, no significant difference was found for VO_{2peak} ($ml \cdot kg^{-1} \cdot min^{-1}$) relative to body mass. Maximal heart rate, systolic, and diastolic blood pressure were not different between blacks and whites.

Table 4. Descriptive and Physical Characteristics of Subjects

Variable	Blacks	Whites
Age (years)	19.4 $\pm$.3	22.9 $\pm$.6 *
Height (cm)	179.2 $\pm$ 3	181.2 $\pm$ 2
Weight (kg)	75.0 $\pm$ 2	82.5 $\pm$ 3 *
BMI (kg/m ²)	23.5 $\pm$.7	25.1 $\pm$.6
BSA (m ²)	1.9 $\pm$.03	2.0 $\pm$.04
Sum of Skinfolks (mm)	29.6 $\pm$ 3	32.1 $\pm$ 2
Rest SBP (mmHg)	119.0 $\pm$ 2	117.7 $\pm$ 2
Rest DBP (mmHg)	72.5 $\pm$ 1	71.5 $\pm$.8
Rest MAP (mmHg)	87.8 $\pm$ 1	86.7 $\pm$.9
Rest HR (b·min ⁻¹)	58.8 $\pm$ 2	59.0 $\pm$ 2

Values are listed as mean $\pm$ SE

* significant at $p \leq 0.05$

Table 5. Peak Oxygen Uptake ($\text{VO}_{2\text{peak}}$) Data

Variable	Blacks	Whites
$\text{VO}_{2\text{peak}}$ ($\text{L}\cdot\text{min}^{-1}$)	$3.2 \pm .1$	$3.6 \pm .2 *$
$\text{VO}_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\text{min}^{-1}$)	42.6 ± 1	44.3 ± 1
Peak HR ($\text{b}\cdot\text{min}^{-1}$)	188.3 ± 2	188.9 ± 2
Peak SBP (mmHg)	220.7 ± 4	211.7 ± 3
Peak DBP (mmHg)	90.3 ± 2	86.0 ± 1

Values are listed as mean $\pm$ SE

* significant at $p \leq 0.05$

Graded dynamic exercise test

The cardiovascular measures at rest and during the graded, submaximal, dynamic exercise test are presented in Figures 1-6. No significant differences were found for SBP, DBP, MAP, and HR during supine or seated rest. A significant (Race) main effect was found for SBP ($p = .01$), DBP ($p = .002$), and MAP ($p = .002$), blacks had higher values during exercise (Figures 1-3). No significant differences appeared in the analyses of the HR and CI data; however, blacks exhibited higher SVR ($p = .02$) values during exercise compared to whites (Figures 4-6).

Table 6 presents the mean percentages for each fiber type and capillary density (capillaries/mm²) in the two racial groups examined. A significant racial difference was observed for type I ($p = .05$) fibers; whites had a greater percentage (50.8 ± 2.7) compared to blacks (43.7 ± 2.2). Blacks tended to have a greater percentage of type IIb fibers ($p = .06$); however, no significant difference was observed in the percentage of type IIa fibers.

Capillary density (capillaries/mm²) was determined in ten black and ten white subjects. Blacks demonstrated a lower capillary density 252.6 ± 10.3 (capillaries/mm²) compared to whites 269.6 ± 11.7 (capillaries/mm²); however, this difference was not statistically significant.

Resting MAP was not related to the percentage of type II fibers (Figure 7). A significant positive correlation

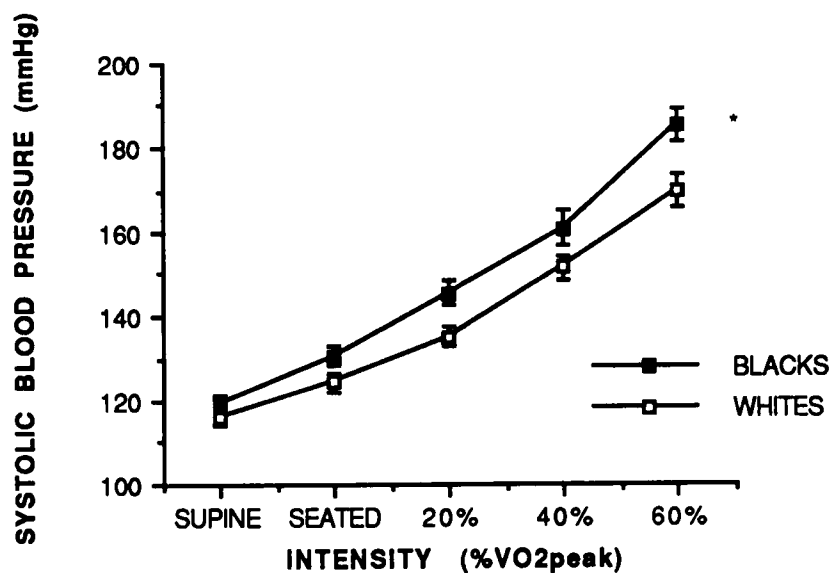


Figure 1. Systolic blood pressure response to dynamic exercise. A significant main effect for (Race) was found ($p = .01$) during exercise. Values are means $\pm$ SE

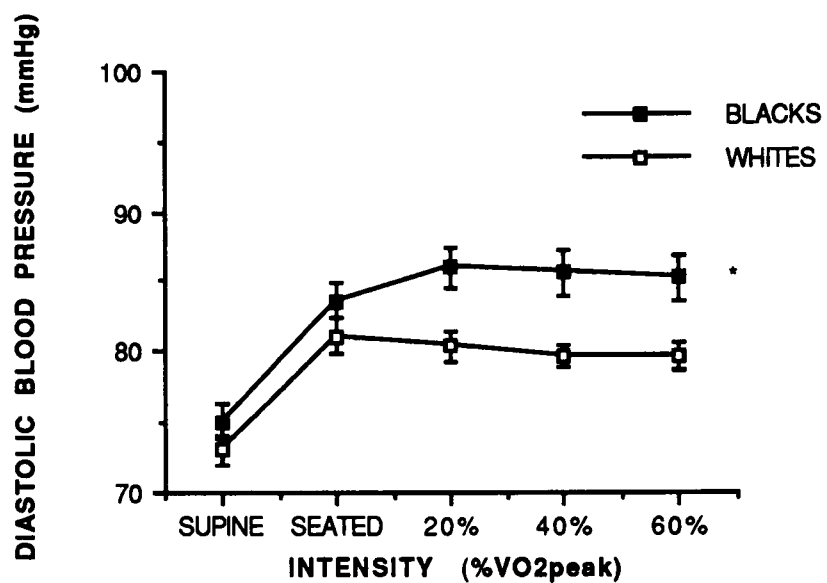


Figure 2. Diastolic blood pressure response to dynamic exercise. A significant main effect of (Race) was found ($p = .002$) during exercise. Values are means $\pm$ SE

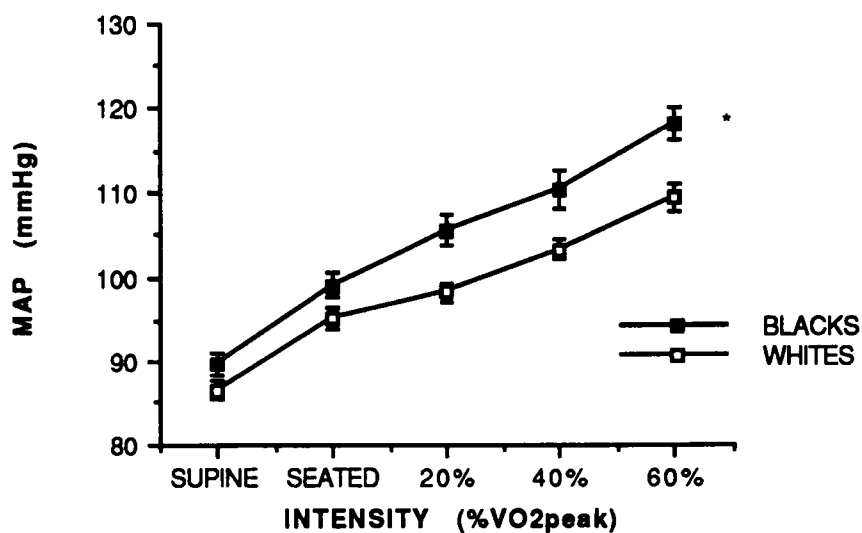


Figure 3. Mean arterial blood pressure response to dynamic exercise. A significant main effect (Race) was found ($p = .002$) during exercise. Values are means $\pm$ SE

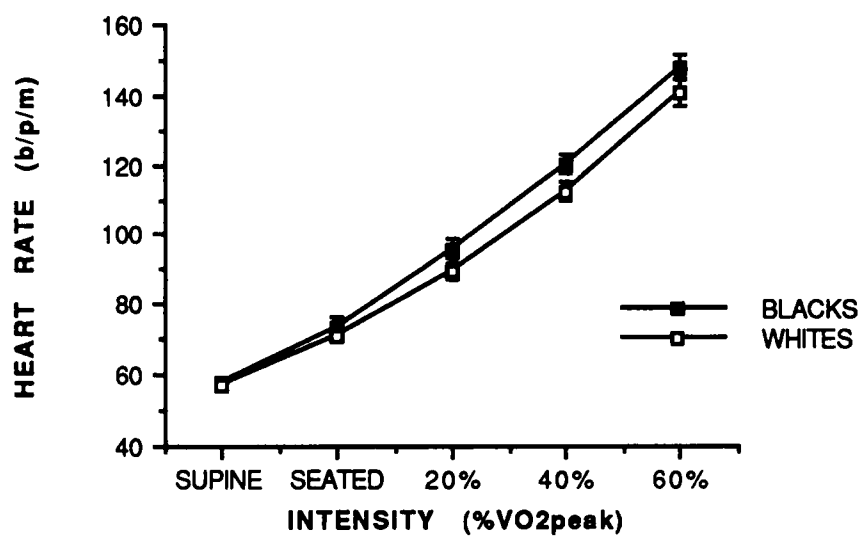


Figure 4. Heart rate response to dynamic exercise.
Values are means $\pm$ SE

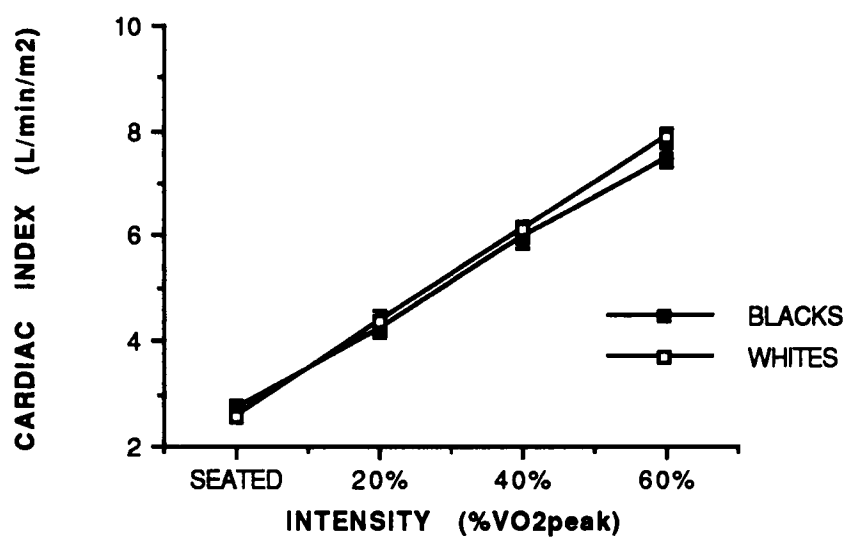


Figure 5. Cardiac index response to dynamic exercise. Values are means $\pm$ SE

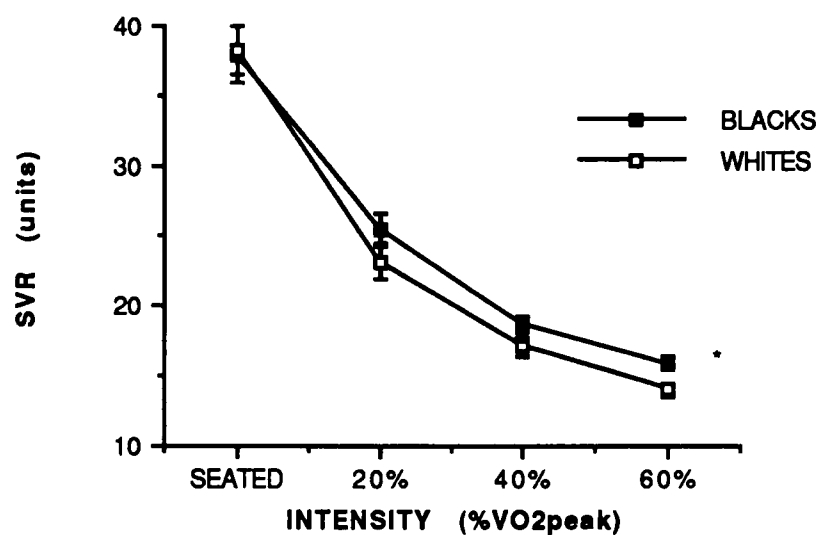


Figure 6. Systemic vascular resistance response to dynamic exercise. A significant main effect (Race) was found ($p = .01$) during exercise. Values are means $\pm$ SE

Table 6. Muscle Fiber Composition and Capillary Density of the Vastus lateralis

Fiber type	Blacks	Whites
type I %	43.7 $\pm$ 2.2	50.8 $\pm$ 2.7 *
type IIa %	34.9 $\pm$ 1.3	33.5 $\pm$ 2.4
type IIb %	22.1 $\pm$ 2.3	15.7 $\pm$ 2.3
capillary density (capillaries/mm ²)	252.6 $\pm$ 10.3	269.6 $\pm$ 11.7

Values are listed as mean $\pm$ SE

* significant at $p \leq 0.05$

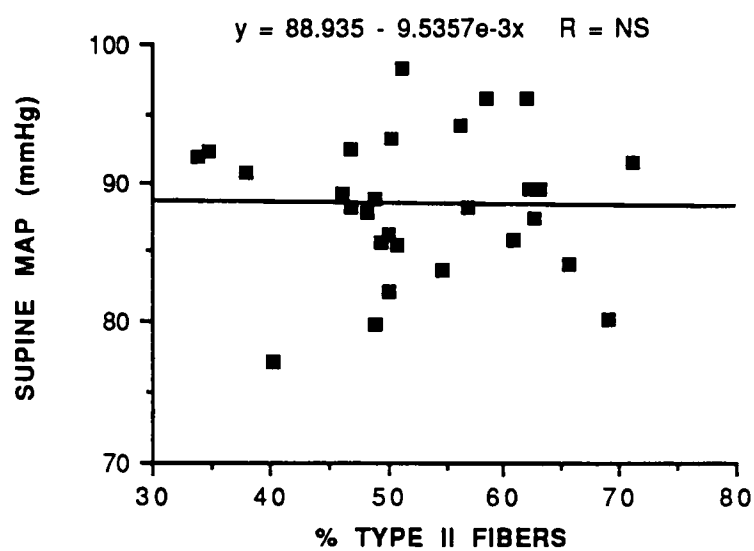


Figure 7. Scatter plot of resting mean arterial blood pressure and the percentage of type II muscle fibers.

($r = 0.54$, $p = .003$) was found between the change in MAP from supine rest to 60% of VO_{2peak} and the percentage of type II fibers (Figure 8). The percentage of type IIb fibers was also correlated to the change in MAP during dynamic exercise ($r = 0.49$, $p = .007$); however, no relationship was found between the percentage of type IIa fibers and the change in MAP during exercise (Figure 9-10). SVR at 60% of VO_{2peak} was also correlated to the percentage of type II fibers ($r = .47$, $p = .01$) (Figure 11). No relationship was found between the maximal change in MAP during exercise and capillary density (Figure 12).

Linear regression analyses between the change in MAP from supine rest to 60% of VO_{2peak} and the percentage of type II fibers were also computed for blacks and whites separately (Figure 13-14). A strong positive correlation ($r = 0.78$, $p = .001$) was found for blacks. This relationship did not exist for whites ($r = 0.27$, $p = 0.4$). Additionally, the relationship between systemic vascular resistance at 60% VO_{2peak} was examined for blacks and whites (Figures 15-16). A very strong positive correlation ($r = 0.87$, $p = .0001$) was found for blacks and no relationship existed for the whites ($r = 0.09$, $p = 0.76$).

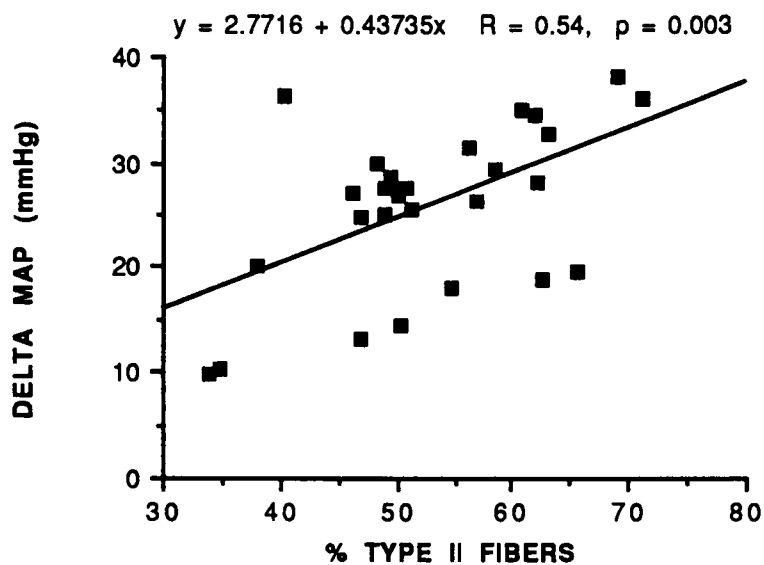


Figure 8. Scatter plot of the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and the percentage of type II fibers.

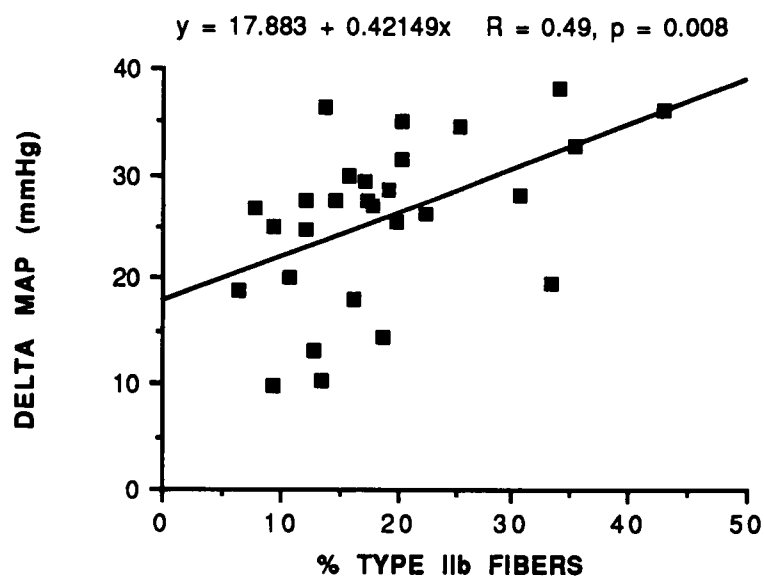


Figure 9. Scatter plot of the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and the percentage of type IIb fibers.

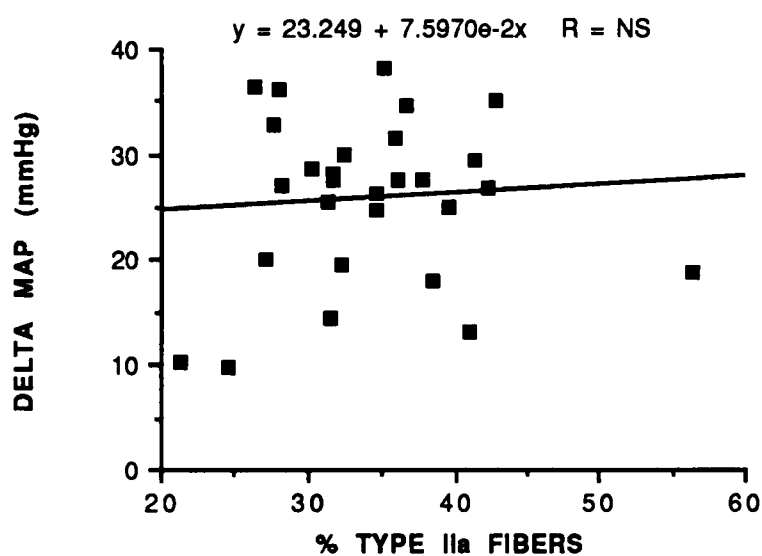


Figure 10. Scatter plot of the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and the percentage of type IIa fibers.

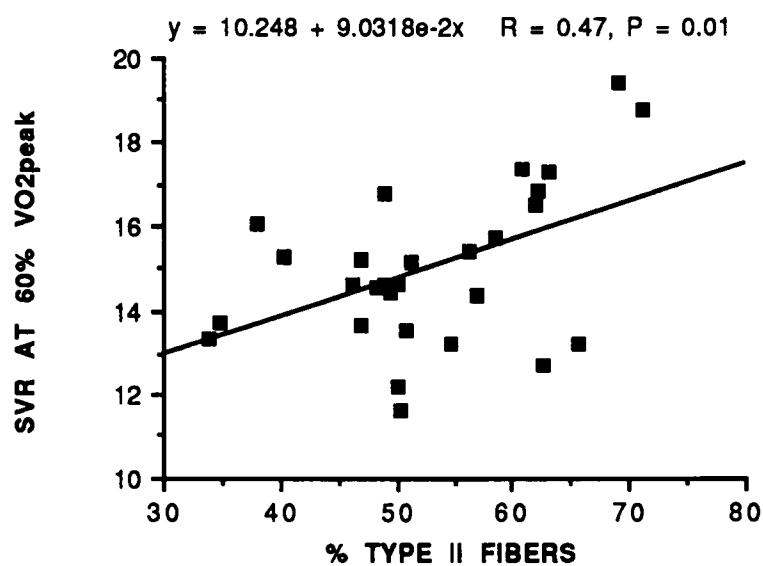


Figure 11. Scatter plot of systemic vascular resistance at 60% of VO_{2peak} and the percentage of type II muscle fibers.

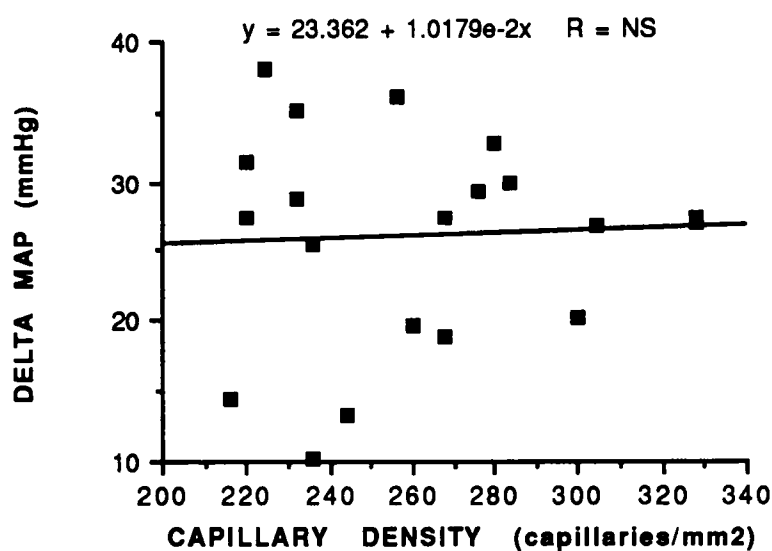


Figure 12. Scatter plot of the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and capillary density (capillaries/mm²).

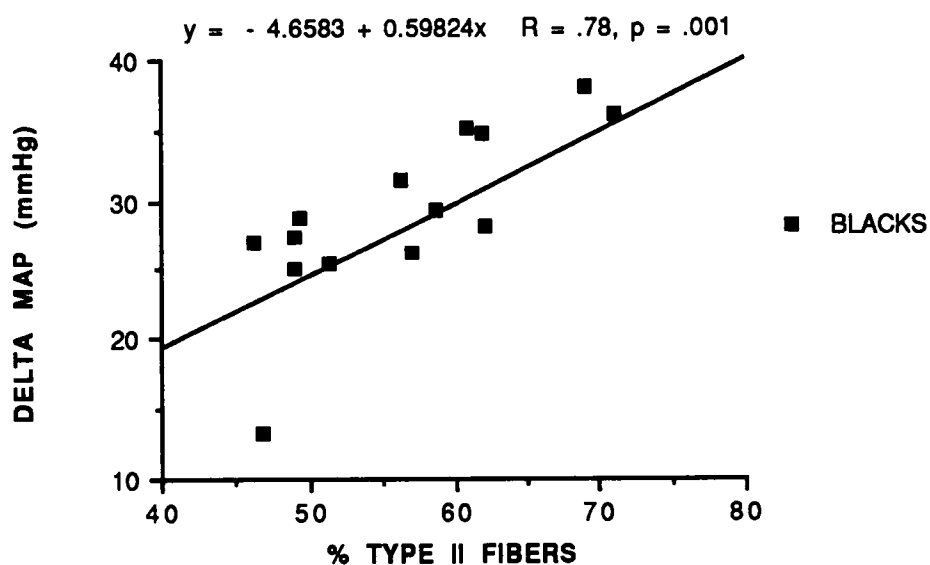


Figure 13. Scatter plot of the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and the percentage of type II fibers for blacks.

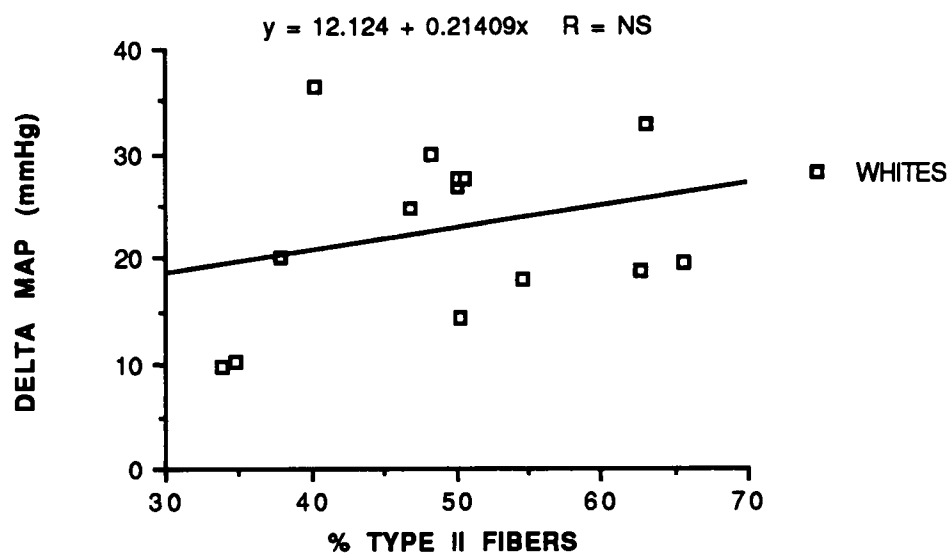


Figure 14. Scatter plot of the change in mean arterial blood pressure from supine rest to 60% of VO_{2peak} and the percentage of type II fibers for whites.

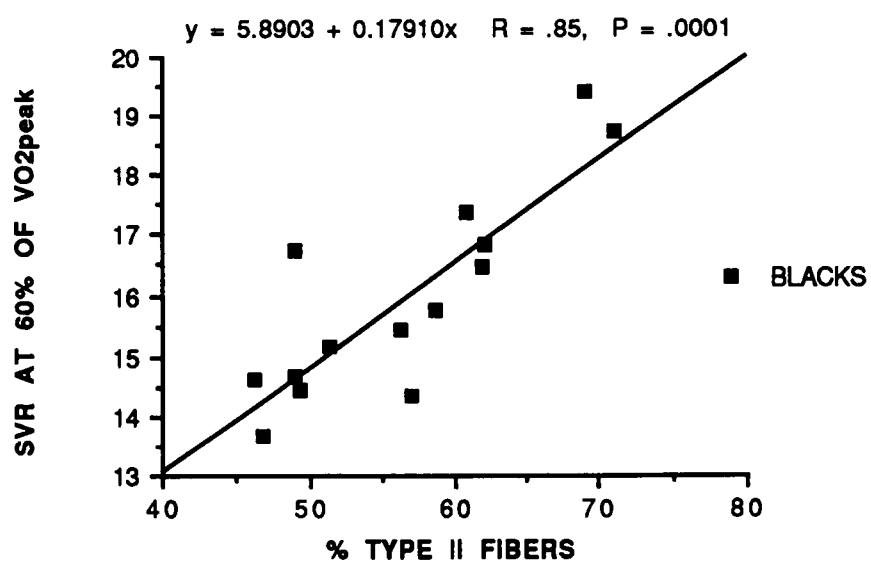


Figure 15. Scatter plot of systemic vascular resistance at 60% of VO_{2peak} and the percentage of type II muscle fibers for blacks.

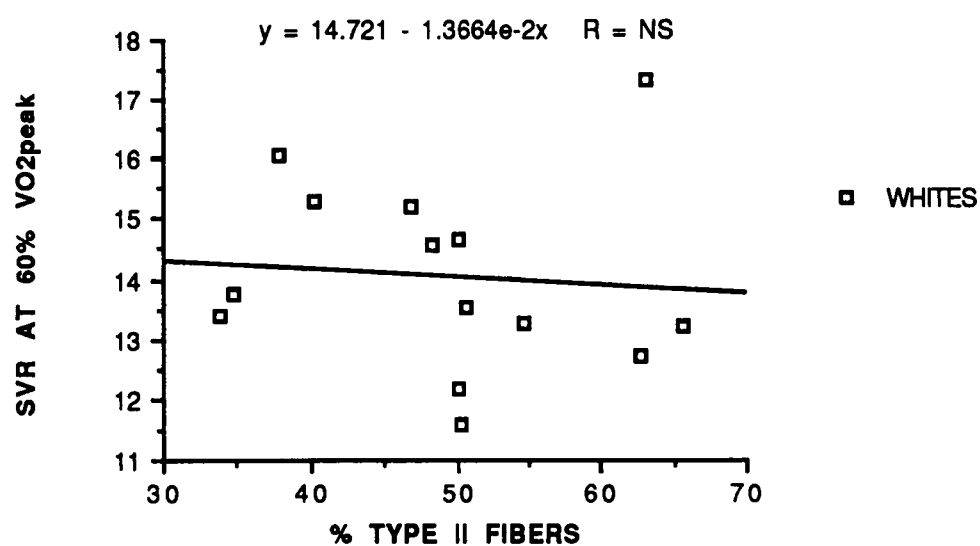


Figure 16. Scatter plot of systemic vascular resistance at 60% of VO_{2peak} and the percentage of type II muscle fibers for whites.

CHAPTER V

DISCUSSION

The purpose of this study was to examine muscle fiber composition and cardiovascular responses during dynamic exercise in a biracial population. 30 normotensive, college-aged males, (15 black, and 15 white) were evaluated. Consistent with the results of other studies on young, normotensive men (40,43,54), a racial difference in the blood pressure response to exercise was obtained in the present study. Systolic, diastolic, and mean arterial blood pressures were greater in blacks during graded, submaximal, dynamic exercise on a cycle ergometer. Ekelund et al. (39) reported similar findings during a modified Bruce protocol treadmill test. Systolic blood pressures were significantly greater in black men (174 mmHg) during stage 2 of the treadmill test compared to white men (166 mmHg). Walker et al. (43) demonstrated similar trends in a biracial population of normotensive males. Thomas et al. (40) measured blood pressure responses during dynamic exercise in a biracial population of 1006 medical students and reported a significantly higher maximum pulse pressure in the black men and women. Additionally, Alpert et al. (125) and Treiber et al. (41) have documented an exaggerated blood pressure response to dynamic exercise in normotensive children.

In this study, the exaggerated blood pressure response of the blacks was the result of an elevated peripheral resistance confirming other studies by Walker et al. (43) and Arensman et al. (52). Falkner (52) and Anderson et al. (51) have speculated that the exaggerated blood pressure response could be due to an enhanced sympathetic drive, an augmented alpha-adrenergic sensitivity, and/or a structural alteration of the peripheral vasculature (51,52). Specifically, one of the factors that may contribute to an elevated resistance are differences in the structural vascularity of skeletal muscle.

Muscle fiber composition and BP response to dynamic exercise

In the current study, a positive relationship was found between the change in MAP (rest to 60% $\text{VO}_{2\text{peak}}$) and the percentage of type II fibers in skeletal muscle ($r = 0.54$, $p = .003$). Type II fibers are less highly vascularized compared to type I fibers due to their decreased reliance on oxidative metabolism (101). Frisk-Holmberg et al. (109) reported similar findings during isometric exercise in a white population. The change in mean arterial blood pressure was correlated with the percentage of type II fibers for both hypertensive ($r = 0.62$) and normotensive ($r = 0.47$) individuals. In addition, Fitton et al. (111) reported that caucasian sprinters (a group presumed to have a predominance of type II fibers) exhibited exaggerated blood pressure responses to static and dynamic exercise when compared to

caucasian distance runners; however, muscle fiber composition was not directly quantified.

Muscle fiber composition appeared to be more important in explaining the variation in the blood pressure response of blacks. A strong positive correlation ($r = 0.78$, $p = .001$) was found between the change in mean arterial blood pressure and the percentage of type II fibers for blacks. This relationship was not significant ($r = 0.27$, $p = 0.4$) in the whites.

In the present study, systemic vascular resistance at 60% VO_{2peak} was correlated to the percentage of type II muscle fibers ($r = 0.47$, $p = 0.01$) for both blacks and whites. These results imply that muscles with a high percentage of type II fibers have a higher resistance compared to muscles with a high percentage of type I fibers. However, when separating the data by race a different conclusion could be drawn. A very strong positive correlation ($r = 0.85$, $p = .0001$) was found for blacks and no correlation existed for whites ($r = .09$, $p = .78$). Recently, data collected in our laboratory demonstrated that young normotensive blacks exhibit a compromised vasodilatory capacity at rest compared to normotensive whites (53). These findings support Falkner's hypothesis that the peripheral vasculature may be altered prior to racial differences in resting blood pressure (126).

Muscle fiber composition and capillary density

In the present study, blacks had a greater percentage of type II fibers (56%) compared to the whites (49%). Ama et al. (107) reported similar findings in a biracial population of college age males. They performed biopsies of the vastus lateralis muscle in 23 blacks and 23 whites and reported that the percentage of type II fibers was higher in blacks (68%) than the whites (59%). It has been shown that individuals with essential hypertension possess a greater percentage of type II fibers (108,109,127). In 42 subjects, (22 hypertensive and 19 normotensive) the percentage of type II fibers was 60% in the hypertensives and 55% in the normotensives (108). Frisk-Holmberg et al (109) reported similar percentages of type II muscle fibers in 34 subjects (17 hypertensive and 17 normotensives), 52% for hypertensives and 43% for normotensives.

Microvascular rarefaction has been demonstrated in both human (110) and animal hypertension (128-130). Specifically, Henrich et al. (110) reported a decreased capillary density in the quadriceps muscle (37%) and the pectoralis major (51%) in individuals who were clinically defined as having essential hypertension. In the present study, capillary density expressed (capillaries/mm²), was determined in 10 blacks and 10 whites. Blacks demonstrated a decreased capillary density 252.6 ± 10 (capillaries/mm²) compared to whites 269.6 ± 12 (capillaries/mm²); however, this difference was not

statistically significant.

In summary, we observed a higher blood pressure response to dynamic exercise in a group of young, normotensive blacks compared to whites. An elevated total peripheral resistance appeared to be the underlying hemodynamic mechanisms responsible for the observed racial disparity in blood pressure response since cardiac index was the same. The percentage of type II muscle fibers was correlated to the change in MAP (rest to 60% $\text{VO}_{2\text{peak}}$) and systemic vascular resistance at 60% of $\text{VO}_{2\text{peak}}$ in blacks; however, no relationship was found in the whites. Therefore, if racial differences in fiber type do in fact exist this could be a potential factor for the increased prevalence of hypertension in blacks.

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APPENDIXES

APPENDIX A**MECHANISMS OF RACIAL DIFFERENCES IN
BLOOD PRESSURE REACTIVITY TO STRESS****INFORMED CONSENT FORM****Address:**

Dept. of Human Performance and Sport Studies
School of Education
University of Tennessee-Knoxville
1914 Andy Holt Ave./ Knoxville, TN 37996
Phone: 615 974-5111

You are invited to participate in a research study, the purpose of which is to examine racial differences in the control of blood pressure during exercise. The findings are expected to influence our understanding of why blacks have a higher rate of hypertension than whites. A description of the procedures that you will undergo is listed below:

Exercise Bouts.

You will undergo a maximal bicycle test for the determination of maximal oxygen uptake, which will be measured using a computerized oxygen uptake system. On another day, you will report to the laboratory following an overnight fast. After 20 minutes in the supine position, a series of resting measurements will be obtained. You will then perform cycle

exercise at relative intensities of 20, 40, and 60% of their maximum (6 min per stage).

Needle Biopsy.

On a separate day you will report to the laboratory for a needle biopsy procedure. This involves taking 2-3 small samples of muscle from your thigh, each one the size of a small pea. The skin will be shaved and cleaned thoroughly. After injecting a local anesthetic to cause numbness, a small incision (1/4 inch long) will be made. A biopsy needle will be inserted into the muscle and the tissue sample removed. Afterwards, a butterfly bandaid will pull the incision together and a sterile bandage will be applied. You may experience a momentary sensation of pressure or pain when the sample is taken, followed by a dull ache for one to two days following the procedure. Care should be taken to keep the area clean and dry for several days until the skin heals. You will return to the laboratory 3-5 days after the muscle biopsy to be evaluated for any signs of infection.

The risks involved with exercise include abnormal blood pressure responses, fainting, musculoskeletal injury, disorders of the heart, and in very rare instances heart attack. Additional risks for the muscle biopsy procedure include infection and internal bleeding. However, there is only a minor chance that serious complications will occur. In the event that physical injury occurs as a result of the study, the University of Tennessee does not automatically

provide reimbursement for medical care or other compensation. If physical injury is suffered in the course of research, or for more information, please notify the investigators in charge, Dr. David Bassett or Dr. Ed Howley, at 974-5111. Benefits to be expected include financial payment (\$100) and an assessment of your fitness. Your total time involvement would be 1-2 hours/day on 4 separate days.

The information in this study will be provided to you. Data from this study may be used in research reports or presentations; however, your name and other identity will not be disclosed.

You are free to decide whether or not to participate in this study and are free to withdraw from the study at any time.

Before you sign this form please ask questions about any aspects of the study which are unclear to you

AUTHORIZATION:

I, _____, have read the above and decided to participate in the research project described above. My signature also indicates that I have received a copy of this consent form.

Participant's signature

Date

Witness

Date

APPENDIX B

PERSONAL HISTORY FORM

Name _____ Age _____

Date _____

Check X if yes

Past History

Have you ever had?

Free or easy bleeding	()	()	()
(hemophilia)			
Rheumatic fever	()	()	()
Heart murmur	()	()	()
High blood pressure	()	()	()
Any heart problems	()	()	()
Varicose veins	()	()	()
Lung disease	()	()	()
Seizures	()	()	()
Irregular heart beat	()	()	()
Bronchitis	()	()	()
Emphysema	()	()	()
Diabetes	()	()	()
Asthma	()	()	()
Kidney disease	()	()	()
Liver disease	()	()	()
Hay fever	()	()	()

Present Symptoms Review:

Have you recently had?

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 or Leg	()
		Numbness	()

Do you smoke?

How many per day?

APPENDIX C**CONTRAINDICATIONS TO EXERCISE TESTING**

1. Recent acute myocardial infarction
2. Unstable angina
3. Uncontrolled ventricular dysrhythmia
4. Uncontrolled atrial dysrhythmia which compromises cardiac function
5. Congestive heart failure
6. Severe aortic stenosis
7. Suspected or known dissecting aneurysm
8. Active or suspected myocarditis
9. Thrombophlebitis or intracardiac thrombi
10. Recent systemic or pulmonary embolus
11. Acute infection
12. Third degree heart block
13. Significant emotional distress (psychosis)
14. A recent significant change in the resting ECG
15. Acute pericarditis

RELATIVE CONTRAINDICATIONS TO EXERCISE TESTING

1. Resting diastolic blood pressure over 120 mmHg or resting systolic blood pressure over 200 mmHg
2. Moderate valvular heart disease
3. Digitalis or other drug effect
4. Electrolyte abnormalities
5. Fixed rate artificial pacemaker
6. Frequent or complex ventricular irritability
7. Ventricular aneurysm
8. Cardiomyopathy including hypertrophic cardiomyopathy
9. Uncontrolled metabolic disease (diabetes, thyrotoxicosis, myxedema, etc.)
10. Any serious systemic disorder (mononucleosis, hepatitis, etc.)
11. Neuromuscular, musculoskeletal, or rheumatoid disorders which would make exercise difficult

APPENDIX D

SUBJECT: _____

FAMILY HISTORY OF HIGH BLOOD PRESSURE

IT IS IMPORTANT FOR US TO KNOW IF EITHER OF YOUR PARENTS HAVE HIGH BLOOD PRESSURE BECAUSE FAMILY HISTORY OF HIGH BLOOD PRESSURE MAY EXPLAIN SOME OF THE INDIVIDUAL VARIATION WE ARE SEEING IN THE BLOOD PRESSURE RESPONSES TO EXERCISE . THE NEXT TIME YOU TALK TO YOUR PARENTS, PLEASE ASK THEM THE FOLLOWING QUESTIONS:

- 1) HAS YOUR MOTHER EVER BEEN DIAGNOSED AS HAVING HYPERTENSION
(HIGH BLOOD PRESSURE)?
YES _____ NO _____ DO NOT KNOW _____
- 2) IS YOUR MOTHER CURRENTLY TAKING BLOOD PRESSURE MEDICATION?
YES _____ NO _____ DO NOT KNOW _____
- 3) HAS YOUR FATHER EVER BEEN DIAGNOSED AS HAVING HYPERTENSION
(HIGH BLOOD PRESSURE)?
YES _____ NO _____ DO NOT KNOW _____
- 4) IS YOUR FATHER CURRENTLY TAKING BLOOD PRESSURE MEDICATION?
YES _____ NO _____ DO NOT KNOW _____

ALL INFORMATION WILL BE KEPT CONFIDENTIAL!

THANKS

APPENDIX E

AMERICAN HEART ASSOCIATION: HYPERTENSION STUDY
MUSCLE FIBER COMPOSITION AND
BLOOD PRESSURE RESPONSE EXERCISE

DAY 1: SCREENING DATA SHEET

NAME _____ DATE _____

PHONE # _____ SS# _____

ADDRESS:

AGE _____ RACE _____ HEIGHT (cm) _____ WEIGHT (kg) _____

1) HEALTH HISTORY _____

2) INFORMED CONSENT _____

3) PARENTAL HISTORY QUESTIONNAIRE _____

4) RESTING BLOOD PRESSURE _____/_____
_____/_____
_____/_____ MEAN _____/_____

5) RESTING HEART RATE _____

_____ MEAN _____

6) SKINFOLDS CHEST _____ MEAN _____
SUBSCAP _____ MEAN _____
TRICEP _____ MEAN _____

SUM OF SKINFOLDS _____

MEAN RESTING BLOOD PRESSURE FOR THREE DAYS

SCREENING _____/_____

MAX TEST DAY _____/_____

GRADED DAY _____/_____

MEAN _____/_____

AMERICAN HEART ASSOCIATION: HYPERTENSION STUDY
MUSCLE FIBER COMPOSITION AND
BLOOD PRESSURE RESPONSE TO EXERCISE

DAY 2: MAX TEST DAY

NAME _____ DATE _____

WEIGHT (kg) _____ BAROMETRIC PRESSURE _____ TEMP _____

RELATIVE HUMIDITY _____ SEAT HEIGHT _____

RESTING BLOOD PRESSURE / MEAN /
 /
 /

RESTING HEART RATE _____ MEAN _____

EXERCISE BLOOD PRESSURES/HEART RATES

UNLOADED CYCLING	MINUTE		
0.5 KP	1	<u> </u> / <u> </u>	<u> </u>
1.0 KP	2	<u> </u> / <u> </u>	<u> </u>
1.5 KP	3	<u> </u> / <u> </u>	<u> </u>
2.0 KP	4	<u> </u> / <u> </u>	<u> </u>
2.5 KP	5	<u> </u> / <u> </u>	<u> </u>
3.0 KP	6	<u> </u> / <u> </u>	<u> </u>
3.5 KP	7	<u> </u> / <u> </u>	<u> </u>
4.0 KP	8	<u> </u> / <u> </u>	<u> </u>
4.5 KP	9	<u> </u> / <u> </u>	<u> </u>
5.0 KP	10	<u> </u> / <u> </u>	<u> </u>
5.5 KP	11	<u> </u> / <u> </u>	<u> </u>
6.0 KP	12	<u> </u> / <u> </u>	<u> </u>
6.5 KP	13	<u> </u> / <u> </u>	<u> </u>
7.0 KP	14	<u> </u> / <u> </u>	<u> </u>
	15	<u> </u> / <u> </u>	<u> </u>

MAX HEART RATE _____

MAX BLOOD PRESSURE /

MAX VO₂ (L/MIN) _____

MAX VO₂ (ML/KG/MIN) _____

AMERICAN HEART ASSOCIATION: HYPERTENSION STUDY
MUSCLE FIBER COMPOSITION AND
BLOOD PRESSURE RESPONSE TO EXERCISE

DAY 3: GRADED DAY

NAME _____ DATE _____

WEIGHT (kg) _____ BAROMETRIC PRESSURE _____ TEMP _____

RELATIVE HUMIDITY _____ SEAT HEIGHT _____

RESTING BLOOD PRESSURE _____/_____
_____/_____
_____/_____ MEAN _____/_____

RESTING HEART RATE _____

_____ MEAN _____

BIKE REST:

BP _____/_____
HR _____ VO2 _____ VCO2 _____

CARDIAC OUTPUT _____ TPR _____

BIKE 20%: WORKLOAD _____ PREDICTED VO2 _____

BP _____/_____
HR _____ VO2 _____ VCO2 _____

CARDIAC OUTPUT _____ TPR _____

BIKE 40% WORKLOAD _____ PREDICTED VO2 _____

BP _____/_____
HR _____ VO2 _____ VCO2 _____

CARDIAC OUTPUT _____ TPR _____

BIKE 60% WORKLOAD _____ PREDICTED VO2 _____

BP _____/_____
HR _____ VO2 _____ VCO2 _____

CARDIAC OUTPUT _____ TPR _____

APPENDIX F**HISTOCHEMISTRY - MUSCLE FIBER COMPOSITION****REAGENT PREPARATION:****Alkaline Stock Solution**

Add 2.253 g glycine, 2.40 g CaCl_2 , 1.755 g NaCl, 1.08 g NaOH and bring to a final volume of 400 ml with distilled water. Adjust to pH 9.4

Alkaline Preincubation Solution

Remove 135 ml of the alkaline stock solution and readjust to pH 10.3 using 1N NaOH or 1N HCl as needed

Acid Preincubation Stock Solution

This is a 0.2 M stock barbitol acetate buffer. Add 1.94 g NaOAc, 2.94 g sodium barbitol to 100 ml of distilled water.

Acid Preincubation Solution

Add 31.25 ml acid preincubation stock solution, 62.5 .1 M HCl, and 50 ml distilled water in each of three Columbia jars. Adjust each to its respective pH (4.3, 4.54, 4.6).

Incubation Solution

Add 200 mg ATP to 135 ml of the alkaline stock solution and adjust to pH 9.4 with 1N HCl or 1N NaOH as needed. Label this solution acid incubation. Add 20 mg ATP to 135 ml of the alkaline stock solution and adjust to pH of 9.4. Label this solution alkaline incubation.

PROCEDURE:**Preincubation****Alkaline preincubation**

Incubate labeled slides in the alkaline preincubation solution for fifteen minutes. Agitate slightly at room temperature.

Acid preincubation

Incubate labeled slides in the acid preincubation solution for five minutes. Agitate slightly at room temperature.

Incubation

Rinse preincubation solution from slides with distilled water

Incubate labeled slides in their respective incubation solution in the shaker bath for 45 minutes at 37°C.

Rinse twice with distilled water

Incubate in 1% CaCl_2 for three minutes

Rinse twice with distilled water

Incubate in 1% CoCl_2 for three minutes

Rinse twice with distilled water

Incubate in 1% $(\text{NH}_4)_2\text{S}$ for one minute

Rinse three times with distilled water and allow to dry

APPENDIX G

HISTOCHEMISTRY - CAPILLARY DENSITY

REAGENT PREPARATION:

Schiff's Reagent

1. Place 1.25 g Pararosaniline (Sigma p-3750) in 200 ml boiling distilled water and dissolve
2. Cool to 50° and filter
3. Add 20 ml 1 M HCL
4. Cool to room temperature and add 1 g sodium metabisulphite then leave in the dark overnight in refrigerator
5. Add 300 mg activated charcoal (C-4386), shake for 2 minutes and filter (Whatman 1 filter paper)
6. Add a further 5 ml 1 M HCL and .25 g sodium metabisulphite and store in the dark. (The final solution should be colorless)

Carnoy's Fixative

Mix in the ratio of Ethanol:Chloroform:Glacial Acetic Acid
 6 3 1

1% Periodic Acid

To every 150 ml H₂O add 1.5g Periodic acid (Sigma p-7875) make fresh daily

1% Amylase Solution

To every 150 ml H₂O add 1.5g Amylase (Sigma A-3176) warm up in water bath to 37°

STAINING PROCEDURE

1. Place slides with muscle section in a Columbia jar containing Carnoy's fixative at room temperature for 10 minutes
2. Rinse with distilled water 3 times
3. Place in 1% amylase solution in shaking water bath for 5 minutes at 37°
4. Rinse with distilled water 3 times
5. Place in 1% periodic acid for 10 minutes
6. Rinse with distilled water 3 times
7. Place in Schiff's reagent (pour enough amount into a 50 ml jar and warm up to room temperature) for 15 minutes
8. Rinse in tap water for 10 minutes
9. Dehydrate in ascending alcohols
10. Mount coverslip on slide with paramount

APPENDIX H

Cardiovascular Measures at Rest and
During Graded Dynamic Exercise

Variable	Rest		%VO ₂ PEAK		
	Supine	Bike	20%	40%	60%
SBP (mmHg)					
Blacks	119.3 ± 2.0	130.5 ± 2.2	145.3 ± 3.0	160.7 ± 4.2	185.1 ± 3.8
Whites	116.1 ± 1.8	124.4 ± 2.2	135.1 ± 2.1	151.3 ± 3.0	169.5 ± 4.0
DBP (mmHg)					
Blacks	75.1 ± 1.3	83.6 ± 1.3	86.0 ± 1.5	85.6 ± 1.6	85.2 ± 6.0
Whites	72.7 ± 0.8	81.1 ± 1.2	80.4 ± 1.1	79.7 ± 0.8	79.7 ± 1.0
MAP (mmHg)					
Blacks	89.7 ± 1.4	99.1 ± 1.5	105.6 ± 1.8	110.4 ± 2.3	118.1 ± 2.0
Whites	87.0 ± 1.0	95.3 ± 1.2	98.3 ± 1.2	103.3 ± 1.1	109.4 ± 1.6
HR (b·min ⁻¹)					
Blacks	57.7 ± 1.8	73.6 ± 2.5	96.1 ± 2.9	119.8 ± 3.7	147.8 ± 3.4
Whites	57.3 ± 2.2	71.3 ± 1.4	89.2 ± 2.3	112.2 ± 2.6	140.8 ± 3.9
% VO ₂ (L·min ⁻¹)					
Blacks	-----	.34 ± .01	.80 ± .03	1.34 ± .04	1.96 ± .04
Whites	-----	.38 ± .02	.84 ± .05	1.51 ± .07	2.27 ± .10
CI (L·min ⁻¹ ·m ⁻²)					
Blacks	-----	2.7 ± .17	4.2 ± .19	6.0 ± .23	7.5 ± .18
Whites	-----	2.6 ± .11	4.4 ± .19	6.1 ± .17	7.9 ± .17
SVR (units)					
Blacks	-----	38.0 ± 2.1	25.5 ± 1.1	18.7 ± .62	15.9 ± .43
Whites	-----	38.2 ± 1.7	23.1 ± 1.2	17.1 ± .63	14.0 ± .40

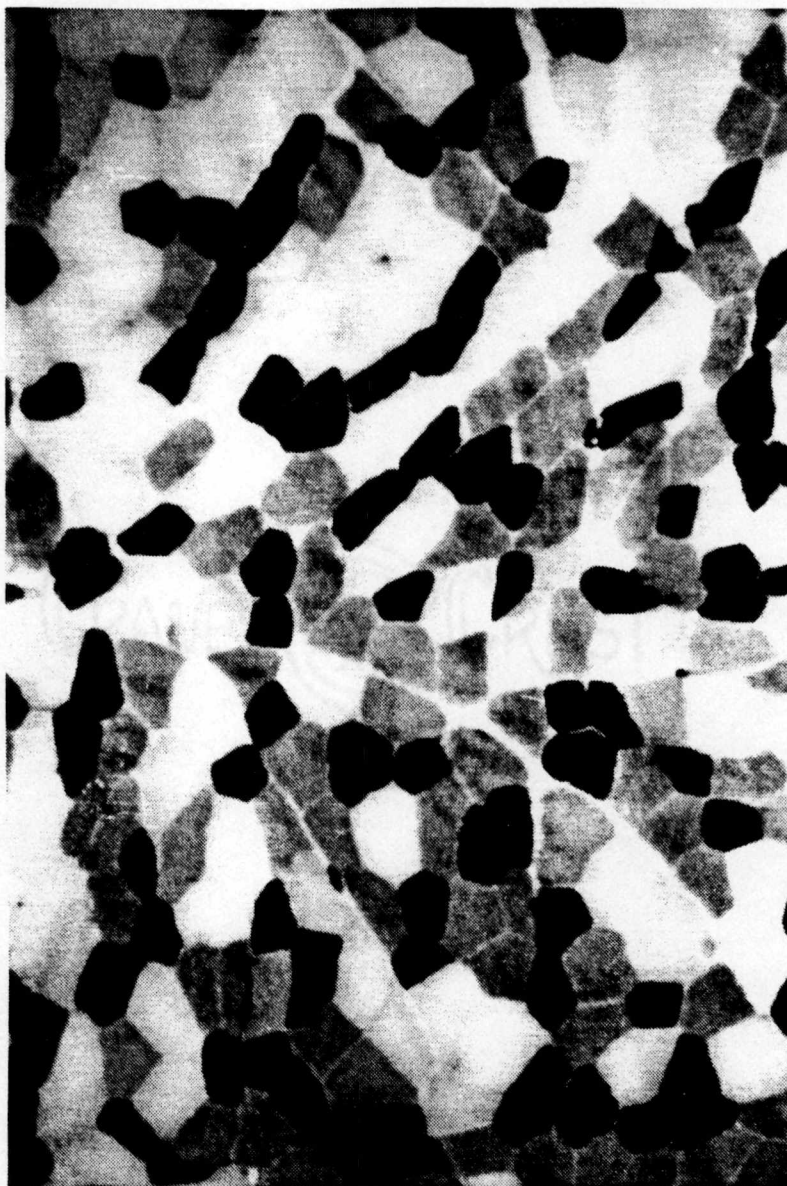
APPENDIX I

Subject With Primarily Type I Fibers



APPENDIX J

Subject With Primarily Type II Fibers



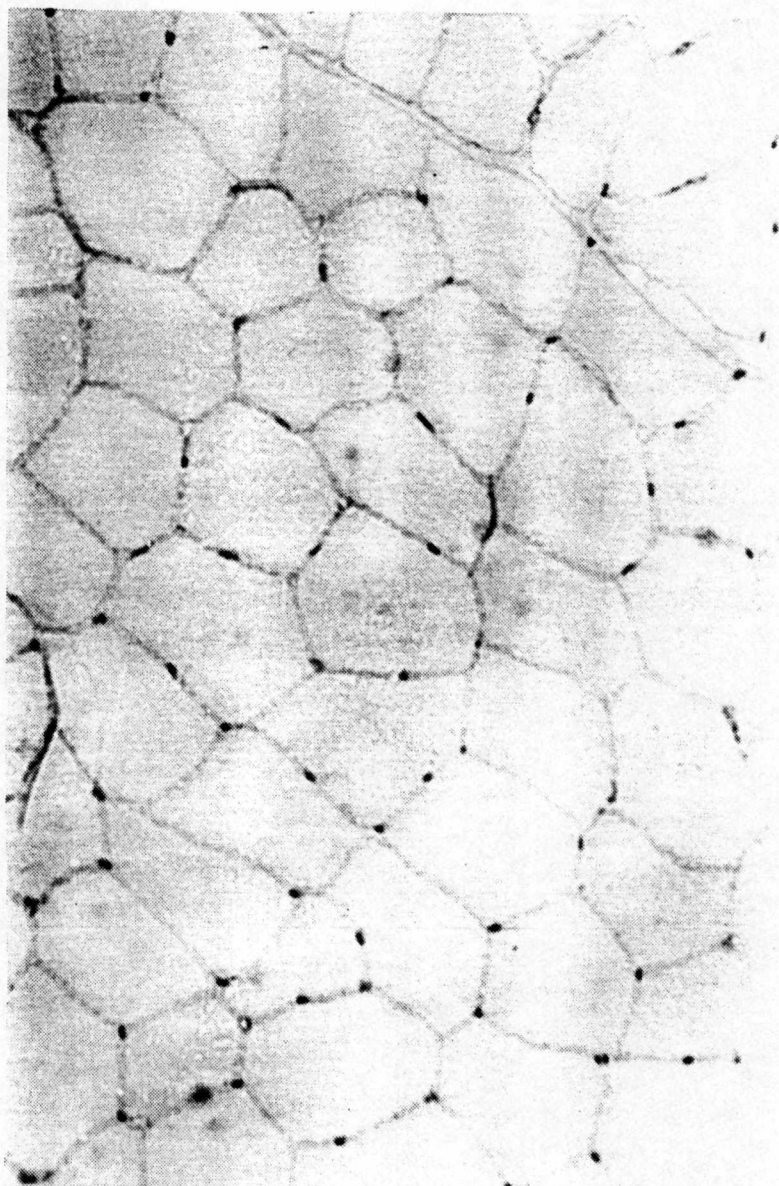
APPENDIX K

Subject With a High Capillary to Fiber Ratio



APPENDIX L

Subject with a Low Capillary to Fiber Ratio



VITA

William John Duey was born in Dallas, Texas on December 27, 1965. In 1969 he moved to Mattoon, Illinois where he attended the public schools. He graduated in May 1984 from Mattoon High School. He entered Concordia University in August of 1984 and transferred to Eastern Illinois University in January of 1987 where he received his Bachelor of Science Degree in physical education in May 1988.

In August 1988 he entered the Graduate School of the University of Tennessee, Knoxville and received a Masters of Science Degree in May of 1990. In 1990, he continued his studies at the University of Tennessee as a doctoral student in the Department of Human Performance and Sports Studies. Following graduation in August of 1992, he will begin teaching at Alabama State University in Montgomery, Alabama as an assistant professor in exercise physiology.