CARDIOVASCULAR RISK FACTORS IN AMERICAN INDIAN AND AFRICAN AMERICAN WOMEN OF CHILD BEARING AGE AND THE RELATIONSHIP OF THESE FACTORS TO BLOOD LEPTIN CONCENTRATION, INSULIN RESISTANCE AND WAIST CIRCUMFERENCE

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1999

Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
MASTER OF SCIENCE
December, 2006

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ACKNOWLEDGMENTS

I wish to express my deep appreciation to my major advisor Dr.Maria Spicer for her excellent guidance, encouragement and patience during every phase of my research. Without her guidance this research would not have been completed. I would like to extend my gratitude to her calm responses and valuable suggestions, understanding and friendship for the last two years.

My sincere appreciation extends to my committee members Dr.Bahram Arjmandi for his valuable advice and suggestions and Dr. Nancy Betts. I would also like to thank the Department of Nutritional Sciences for the financial support provided during my academic career at Oklahoma State University.

I would also like to thank Ms Fanta Toure and other friends for cheering me up whenever I put my head on my table to cry with all the corrections in my thesis.

I am grateful to my family and wish to thank my parents K.P.Krishnan and Omana Ellath, my brother Anoop Ellath and my husband, Ajith Kumaran. They have stood beside me providing me love and encouragement. Their moral support and understanding has made me get to where I am today. Their belief in my abilities helped me make this goal a reality. I wish to dedicate my work to my parents, brother and my husband.

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LIST OF ABBREVIATIONS

CDC	Centers for Disease Control and Prevention
AHA	American Heart Association
NIH	National Institute of Health
BMI	Body Mass Index
HDL-C	High Density Lipoprotein Cholesterol
LDL-C	Low Density Lipoprotein Cholesterol
CVD	Cardiovascular Disease
WHR	Waist Hip Ratio
WC	Waist Circumference
AI	American Indian
AA	African American
CRP	C Reactive Protein
TNF α	Tumor Necrosis Factor Alpha
IL-6	Interleukin 6
mTG	Intramuscular Triglycerides
HOMA IR	Homeostasis Model Assessment Insulin Resistance
CRF	Cardiorespiratory Fitness
CAC	Coronary Artery Calcification

Thesis Format

This thesis contains 5 chapters: the Introduction, literature review, methodology, a chapter in a journal article format, and summary, conclusions and recommendations. The bibliography and journal article are written in the format required by the Journal of the American Medical Association.

CHAPTER I

INTRODUCTION

Cardiovascular disease is a major public health concern in the United States[1] with an estimated health care cost of over $300 billion annually due to disability and death [2, 3] .

According to the American Heart Association, cardiovascular disease mortality accounted for up to 60% of all mortality[1]. According to CDC, heart disease and stroke are the principal components of cardiovascular disease and account for up to 40% of all deaths in the nation[4]. According to American Heart Association, the death rate due to cardiovascular disease accounted for 37.3% of all deaths in 2003[1]. It is estimated that over 71,300,000 adults live with some form of cardiovascular disease in the nation and 1 in 3 adults has some form of cardiovascular disease[1].

Cardiovascular disease is the leading cause of death in both genders among racial and ethnic groups[2]. There is disproportion in death and disability due to cardiovascular disease in minority and low income populations in the nation[3]. Among African Americans 9.9% have heart disease, 5.3% have coronary heart disease, 31.6% have hypertension and 3.5% have had a stroke[5]. Among American Indians 13.8% have heart disease, 8.2 % have coronary heart disease, 23.9% have hypertension and 3.1% have had a stroke[5].

It is estimated that nearly twice as many women die from heart disease compared to all forms of cancer[6]. According to the AHA, cardiovascular disease caused death every minute among women in 2003 in the United States[1]. Over 480,000 American women live with some form of cardiovascular disease every year[1]. Heart disease is the leading killer of minority women in the United States[7]. Among American Indians aged 18 years or older, 61.4% of women have one or more risk factors such as high blood pressure, current cigarette smoking, cholesterol, obesity and diabetes for heart disease[8].

According to the U.S Department of Health and Human Services, African American women had the highest age-adjusted death rate due to major cardiovascular disease and stroke when compared to all American females in 1997-1999. The death rate was more than 395.5 per 100,000 women[9]. High blood pressure and smoking rates are higher in African American women than all groups of women. African American women of 20 years of age or older have higher blood pressure levels (36.4 and 36.0) than white non-Hispanic women (19.7)[10].

Oklahoma ranks third highest in the nation for the prevalence of cardiovascular disease and claimed 317/100,000 lives in 1999[11]. According to the state department of health, cardiovascular disease is the leading cause of death in American Indian and African American populations in Oklahoma in 2003[12]. Heart disease accounted for up to 31% of Oklahoma's deaths in 2001[13]. Coronary heart disease accounted for 1 in 5 deaths in women and was the leading cause of death among females in Oklahoma in 2003 [3].

The likelihood of a woman in Oklahoma to die from heart disease is 50% more than cancer[14]. Minority women from racial and ethnic groups face tremendous social, economic, and cultural barriers to achieving optimal health[15]. Minority women face

lower levels of education, higher levels of unemployment and lack public health insurance. It is estimated that nearly 13 million women live in households with income below the Federal poverty level[16]. Thus minority women have a high risk of death and disability from heart disease, stroke, diabetes, and chronic obstructive pulmonary disease[15]. Thus it is evident that minority women in rural Oklahoma also live below the poverty line and is at higher risk for heart disease.

In Oklahoma, the prevalence of health disparities varies according to race and ethnic minority groups. It was estimated that the annual age adjusted death rate due to heart disease among African Americans accounted for up to 582 and the death rate among American Indians accounted for up to 278 of all deaths between 1991-1995[17].

Obesity is associated with a spectrum of cardiovascular disorders[18]. One of the ways by which adiposity could contribute to the development of cardiovascular disease is through increased leptin production[19-21]. Leptin, the ob-gene product is a protein hormone expressed in the adipose tissue[22]. Leptin correlates with body fat mass in obese and lean subjects[23, 24]. Leptin levels are elevated in obese compared to lean subjects[25].

Insulin is a key hormone involved in glucose metabolism and induces vasodilation. Resistance to the utilization of insulin in obese subjects results in increased insulin production to maintain normal rate of glucose uptake. Thus insulin resistance can adversely affect the cardiovascular system[26].

Waist circumference is a measure of abdominal obesity specifically visceral fat[27]. Abdominal obesity is associated with increased cardiovascular disease in men and women[28]. Risk factors of cardiovascular disease such as high blood pressure, increased triglycerides, total cholesterol, HDL cholesterol and LDL cholesterol in abdominally

obese women has been associated with a waist circumference greater than 88cms in women[27].

However, there is a lack of sufficient data regarding the prevalence of cardiovascular disease in Oklahoma among ethnic minority groups.

Therefore, the following hypotheses were developed

1. There will be a difference between leptin levels, insulin resistance and waist circumference in American Indian and African American women.

2. Blood leptin concentration will be positively correlated with cardiovascular risk factors in American Indian and African American women.

3. Insulin resistance will be significantly correlated with cardiovascular risk factors in American Indian and African American women.

4. Waist circumference is a predictor of cardiovascular risk in American Indian and African American Women.

5. Waist circumference is positively correlated to insulin resistance in American Indian and African American women.

Based on the hypotheses, the following objectives were developed

1. To determine if there is an ethnic difference between leptin levels, insulin resistance and waist circumference in American Indian and African American Women

2. To determine the correlation of blood leptin concentration with cardiovascular risk factors in American Indian and African American Women

3. To examine the relationship of insulin resistance to cardiovascular risk factors in American Indian and African American Women

4. To investigate whether waist circumference is a predictor of cardiovascular risk in American Indian and African American Women.

5. To determine the correlation of Waist circumference and Insulin resistance in both ethnic groups

CHAPTER II

REVIEW OF LITERATURE

CARDIOVASCULAR DISEASE

Global Prevalence Of Cardiovascular Disease

Cardiovascular disease is a cluster of disorders of the heart and blood vessels which includes coronary heart disease, cereberovascular disease, hypertension, peripheral vascular disease, heart failure, rheumatic heart disease, congenital heart disease and cardiomyopathies [29]. It is the most common cause of death worldwide in both men and women. According to The World Health Report 2003 by the World Health Organization, cardiovascular disease accounted for up to 16.7million deaths globally with 7.2 million due to ischemic heart disease, 5.5 million due to cerebrovascular disease and 3.9 million due to hypertension and other heart conditions. Cardiovascular disease deaths accounts for one third of global deaths in low and middle-income countries[30]. In 1999 cardiovascular disease deaths in the low and middle-income countries accounted for approximately 80% of global cardiovascular disease deaths[30].

Cardiovascular disease will be the leading cause of death in developing countries by 2010. In developed countries it is the leading cause of death[31]. According to World Health Report on Violence and Health, 2002, the leading causes of death among the WHO member states in 2000 were ischemic heart disease and cereberovascular disease.

Ischemic heart disease accounted for up to 12.4% of total deaths and cereberovascular disease accounted for up to 9.2% of all deaths. Cardiovascular disease risk poses a global threat in developing countries and industrialized nations.

Prevalence Of Cardiovascular Disease In United States

Cardiovascular diseases are the leading cause of death among men and women of all racial and ethnic groups in the country[32]. According to CDC, it is estimated that almost 70.1 million Americans live with some form of cardiovascular disease[33]. The principal causes of cardiovascular disease death among men and women in the United States are coronary heart disease and stroke[34]. According to the CDC, heart disease and stroke are the principal components of cardiovascular disease and accounts for up to 40% of all deaths in the nation[4].

Health Disparities Among Minority Populations

Health disparities among minority populations can be categorized by race, ethnicity and socioeconomic status in the United States[35]. Racial and ethnic minority groups are growing rapidly in increasing proportions in the United States. According to the US Census Bureau 2000, 1 of every 4 U.S residents belongs to a racial and ethnic minority group[36]. It is estimated that, in 2010 the number of racial and ethnic minorities will be 1 out of 3 U.S. residents. By 2050, the proportion will continue to increase and minority population will account for 50% of the total U.S population[37].

According to the 2004, National Healthcare Quality Report and National Healthcare Disparities Report, women experience socioeconomic disparities in addition

to gender and racial/ethnic disparities. It is estimated that over 53 percent of all African American women receive poorer quality care than whites [38]. They have access to worse care by 29%. Minority women face lower levels of education, higher levels of unemployment and lack public health insurance[15, 39]. It is estimated that nearly 13 million women live in households with income below the Federal poverty level[16].

According to the CDC.1993-2004 report, it is estimated that the prevalence of cardiovascular disease among non-Hispanic white men and women is 66%. Cardiovascular disease rate among non-Hispanic black men and women is 85.8% [40] and 58.5% in Mexican American men and women.

According to the 2000 US Census Bureau report, African Americans accounted for up to 12.9% of total population. Cardiovascular disease is the leading killer of African Americans in the United States. According to the CDC, cardiovascular disease deaths among African Americans accounts for up to 36.4% of all the other causes of deaths every year. In 2001, the death rates among African Americans due to heart disease were 30% higher than among whites. The death rates due to stroke in African Americans were 41% higher than among whites[34]. Of all the minority groups, African Americans tend to develop high blood pressure at a younger age[10, 41]. They are also less likely to engage in physical activity [42].

According to the 2000 US Census Bureau, American Indians accounted for up to 1.5% of the overall population with a total of 4.1 million. Heart disease is the leading cause of death among American Indians and Alaska Natives. In a study conducted in Montana from 1991-1995 and 1996-2000 to calculate the heart disease and stroke

mortality rate of American Indians and Caucasians, it was shown that the death rates due to heart disease and stroke were higher in American Indian men and women compared to white men and women thus suggesting that American Indians are at a higher risk of developing cardiovascular disease compared to whites[43].

According to National Vital Statistics, in 2002, the death rate among African Americans due to cardiovascular disease accounted for up to 33.3% and death rate due to cardiovascular disease among American Indians accounted for up to 24.5% of all deaths. The prevalence of diabetes mellitus, a risk factor for heart disease was higher in American Indians which accounted for up to 6% followed by African Americans which accounted up to 4.4%[44] (National Vital Statistics Reports, Vol 53, No 17, March 7, 2005). Smoking increases the risk of cardiovascular disease. According to CDC, it is estimated that in Oklahoma, the smoking rate among racial/ethnic groups in adolescents range from 13% among African Americans to 26% among American Indians[45].

Ethnicity And Obesity

Obesity and overweight, a risk factor for cardiovascular disease in the U.S population is at a higher rate among racial/ethnic minority populations such as African Americans and American Indians compared with whites. The prevalence of obesity is high among African Americans, particularly African American women. According to CDC, between 1994 and 2000, obesity among African American men increased from 21.3% of American adults to 28.8% American adults. Between 1994 and 2000, obesity among African American women increased from 39.1% of American adults to 50.8% of American adults[46]. When compared to non-Hispanic white women, 69% of AA women

are overweight or obese[47]. Data obtained from National Health and Nutrition Examination Survey (NHANES) have shown that the prevalence of obesity is twice when compared to European American women[48].

American Indians suffer from obesity and overweight at younger ages. This is due to an excess accumulation of fat in childhood among American Indians[49]. According to CDC, in 1999 the prevalence of overweight among children and adolescents between ages 5 to 17 was 39 percent of males and 38 percent females[49, 50]. The 1990 American Indian School children Height and Weight Survey, conducted by the Indian Health Service, showed that 40% of 5-18 year old American Indians were overweight[51]. Data from other studies have observed that 22% of children were at risk for overweight among American Indians between 5- 18 years of age, while 41% of the sample were overweight[52]. The increasing prevalence of obesity and overweight among younger people poses a health challenge for American Indian communities which increased the risk of cardiovascular disease in this population.

Cardiovascular Disease And Women

Cardiovascular disease particularly coronary heart disease and stroke is the leading killer of women in the United States[53-55]. Cardiovascular disease causes about half a million deaths among women every year[56]. According to the CDC, it is estimated that in 2002, 696,947 Americans died due to heart disease and this accounted for up to 51% of women[57].

Minority women experience a number of health problems with shorter life expectancy, higher incidence of chronic diseases and higher maternal and infant

mortality. Increased poverty rate, lack of education and limited medical care have an additional effect on overall health status of minority women[58].

The cardiovascular disease is linked with a number of risk factors in minority women. Cardiovascular disease is influenced by behavioral, social, cultural and economic factors. Elevated blood pressure, cigarette smoking, hypercholesterolemia, excess body weight, sedentary lifestyle and diabetes increase the likelihood of developing the disease[59].

Cardiovascular Disease In Oklahoma

The prevalence of death due to CVD in Oklahoma is the second highest in the nation with 391.6 deaths per 100,000 populations[11]. According to officials at the Oklahoma State Department of Health (OSDH) each day 30 Oklahomans die of heart disease[60]. Coronary heart disease accounts for one in five deaths in women and was also the leading cause of death among females in Oklahoma in 2003[60]. The age-adjusted mortality rates for diseases of the heart and stroke are higher in Oklahoma compared to U.S [60]. Heart disease accounted for approximately 32% of the state's deaths in 2002[61]. An overall, cardiovascular disease claims 14,500 lives in Oklahomans. This accounts for 44% of all deaths in Oklahoma[62]. The associated risk factors for cardiovascular disease in Oklahoma were higher than those in United States overall in many areas[62].

Diseases of the heart were the leading rankable causes of deaths in Oklahoma among African Americans and American Indians in 2003. This accounted for up to 3,205 deaths in Oklahoma and 2012 deaths per 100,000 which is more than the death caused by malignant neoplasm and cereberovascular diseases[63].

In 1999, heart disease claimed 5,869 lives of women of Oklahoma. The likelihood of women in Oklahoma to die from heart disease is 50% more than cancer[14]. Smoking contributes to 25% of heart disease deaths among women in Oklahoma. Obesity accounts for 32% of heart disease deaths. And sedentary lifestyle accounts for 35% of deaths annually. High blood pressure contributes to 29% of heart disease deaths among Oklahoma women[14]. The use of oral contraceptives has also been found to increase a woman's risk of developing heart disease. In 2000,6% of Oklahoma adult women had diabetes and these women have a two times greater risk of coronary heart disease[14].

CARDIOVASCULAR RISK FACTORS

Risk factors that increase the likelihood of developing cardiovascular disease are high blood pressure, high blood cholesterol, Type2 diabetes, overweight and obesity[30]. According to CDC, it is estimated that 65 million Americans aged 20 or older have high blood pressure. It is also estimated that 107 million Americans aged 20 or older have elevated blood cholesterol[64]. It is estimated that 30% of American adults are obese which accounts up to 60 million[34]. According to NIH Statistics, it is estimated that 20.6 million Americans aged 20 or older have diabetes[65].

Framingham Risk Score

The Framingham Risk Score, developed by the National Cholesterol Education Program Adult Treatment Panel (III) procedures, is a multivariate statistical model that

uses age, sex, high density lipoproteins cholesterol, total cholesterol, systolic blood pressure, and smoking. It predicts a person's risk of having coronary heart disease over a period of 10 years. The risk score calculator includes points for each risk factor. The 10-yr risk for coronary heart disease is high if the points are equal or greater than 30[66].

Systolic Blood Pressure

Epidemiologic studies have demonstrated the relationship between SBP and cardiovascular risk. The Framingham Study was a 14 yr biennial follow up study of 5,127 men and women. Systolic and diastolic blood pressure data was compared to the risk of developing coronary heart disease in this cohort of men and women. There was a strong association of SBP with coronary heart disease risk compared to diastolic blood pressure[67]. Another study conducted in the Framingham Heart Study participants concluded that SBP was a strong predictor of congestive heart failure when compared to diastolic blood pressure[68].

The Brisighella Heart Study, a population based European study examined SBP, diastolic blood pressure, pulse pressure and its relationship to coronary heart disease. The study included men and women between the ages of 14-84. There was a 44% increased risk at SBP of 120-139 mm Hg, 76% increased risk at SBP readings of 140-159 mm Hg and 109% increased risk at SBP greater or equal to 160mm Hg. The risk increased with increasing levels of SBP but not diastolic blood pressure[69].

In a 15 yr cohort study, two independent cross-sectional random samples were investigated for subjects who participated in baseline surveys in 1972 and 1977. Men and women aged 25-64 free of myocardial infarction were studied. Isolated systolic

hypertension was defined as SBP >=160 mm Hg. The incidence of heart disease increased with an increase in SBP. In women, isolated systolic hypertension was significantly higher and increased the risk of myocardial infarction[70].

He et al.[71] conducted a meta-analysis of prospective cohort studies, epidemiologic studies and randomized controlled trials to determine the association between SBP and risk of coronary heart disease and stroke. SBP was more strongly associated with coronary heart disease than diastolic blood pressure. A reduction of 12-13 mm Hg in SBP readings was associated with 21% reduction in coronary heart disease, 37% reduction in stroke and 25% reduction in total cardiovascular mortality[71, 72].

In the Copenhagen City Heart Study, men and women were examined to estimate the influence of blood pressure on the risk of stroke incidence. Again, SBP was more strongly associated with stroke risk than diastolic blood pressure. SBP was the best single predictor of cardiovascular events in this study[73].

Total Cholesterol, HDL-Cholesterol, Diabetes

Epidemiologic studies indicate that high levels of serum total cholesterol are related to coronary heart disease. The report on diet and health by the National Research Council-National Academy of Sciences cited these studies in showing a stronger link between total cholesterol and coronary heart disease[74]. The most prominent studies include The Framingham Heart Study, Multiple Risk Factor Intervention Trial, Brown and Goldstein's research on low-density lipoprotein (LDL) receptors, Coronary Primary Prevention Trial and The Helsinki Heart Study.

The Framingham Heart Study investigated men and women for total cholesterol and coronary heart disease risk over a period of 14 years. The total serum cholesterol levels were between 150 and 300 mg/dl. A positive correlation between total serum cholesterol and coronary heart disease rates was found. There was an increase in coronary heart disease rate with an increase in total serum cholesterol[75].

The Mulitiple Risk Factor Intervention Trial (MRFIT) was a randomized trial on high risk middle aged men. The effects of coronary disease risk factors were tested in this cohort. There was a strong association between total serum cholesterol and coronary heart disease deaths over 6 years. The mortality rate was high for individuals with serum cholesterol levels as low as 180mg/dl[76]. A reduction in serum total cholesterol by 1% decreased the risk of coronary heart disease by 2%. The Helsinki Heart Study reported that patients treated with drugs to lower the levels of total cholesterol also reduced the incidence of heart disease[77, 78].

A population based Italian study was conducted with a 10-yr follow up from 1983-2002. Men and women were examined and followed for elevated blood pressure, smoking, diabetes, and total cholesterol/HDL ratio and the incidence of stroke. The risk factors were independently related to incidence of stroke risk. It was shown that 80% of the population was at a high risk of stroke incidence with elevated levels of all the risk factors. In participants with only one unfavorable (not high) risk factor, the stroke rate incidence was 76% lower than high risk participants with more than one risk factor[79]. These results are consistent with earlier findings which examined the relationship of lipids and risk of ischemic heart disease in middle-aged women[80-82] which found that

there was an increased risk of myocardial infarction and coronary heart disease associated with obesity, elevated blood lipids and apo lipoproteins.

In an 8-yr follow up of the MONICA Augsburg cohort study from 1984-1992, men and women were examined and followed for hypertension, total cholesterol/HDL ratio and smoking. Men had a higher systolic (137 vs.135 mm Hg) when compared to women. Smoking more than 20 cigarettes per day increased the risk of myocardial infarction by 70-80 %. The total cholesterol/ HDL cholesterol ratio greater or equal to 5.5 was a risk factor for myocardial infarction. These risk factors contributed to 65% risk for non-fatal and fatal myocardial infarction in this Augsburg population[83].

HDL-Cholesterol is an independent and inverse predictor of coronary heart disease as well. In the Framingham study, men and women were evaluated for lipids and lipoprotein values between 1969-1971. The major lipid risk factor was HDL- C in those subjects who developed coronary heart disease after a period of time. There was an inverse association of HDL-C with coronary heart disease[84]. Further analysis of the Framingham data[85] to investigate the incidence of coronary heart disease and lipoprotein cholesterol levels found that at the 12-year follow up study, 50% of the participants with higher HDL-C were at lower risk of coronary heart disease than those with lower HDL-C. In the 12-yr follow up of the Prospective Cardiovascular Munster (PROCAM) study, men and women of mean age 15-64 years were evaluated for the incidence of coronary heart disease and its relationship with HDL-C levels. Individuals with HDL-C levels <35mg/dL had a 4-fold increased risk of coronary heart disease within 6 years when compared with levels >35mg/dL[86].

In the 10 yr follow up of the Atherosclerosis Risk Communities (ARIC) Study, men and women free of coronary heart disease were investigated. A strong association of increased coronary heart disease risk and total cholesterol, LDL-C, and triglycerides was shown. There was a decreased risk of coronary heart disease in participants with elevated HDL-C levels. High levels of lipoproteins were also associated with relative risks for heart disease in both sexes[87].

Evidence from the Coronary Primary Prevention Trial (CPPT) and Multiple Risk Factor Intervention Trial (MRFIT) showed that a 1-mg/dl increase in HDL-C was associated with a decrease in cardiovascular risk by 3% in women and 2% in men. Hence an inverse relation of HDL-C and coronary heart disease was observed in these studies.

Obesity

Obesity is a major risk factor for cardiovascular disease[88]. Measurement of body mass index (BMI) has been used to define the population as normal weight (18.5 – 24.9 Kg/m^2), overweight (25.0-29.9Kg/m^2) and obese ($\geq$30 Kg/m^2)[89]. Obesity is associated with accelerated coronary atherosclerosis in young adults. In the Pathobiological Determinants of Atherosclerosis Youth Study (PDAY), BMI in obese young men aged 15-34 years was significantly correlated with increased fatty streaks and raised lesions in the right coronary artery. The effect of BMI on the right coronary artery was higher in men with thicker subcutaneous abdominal fat. It was also observed that the direct effects of obesity such as HDL-cholesterol concentrations, smoking levels, hypertension, and

glycohemoglobin concentrations were significantly higher in this group and they accounted for up to 15% of the effect of obesity in coronary atherosclerosis[90].

The Healthy Women Study, conducted in a sample of premenopausal women from 1983-1985, assessed the relationship between BMI, insulin and cardiovascular risk factors such as blood pressure, triglycerides and HDL-C and fasting glucose. A significant correlation of BMI and blood levels of insulin with all the risk factors except cholesterol and apolipoproteins A-I and apolipoproteins A-II was observed. The interaction between insulin and BMI was not significant. Thus it was suggested that at a given level of insulin, an increase in BMI may be associated with elevated SBP and triglycerides and apolipoprotein-B[91].

McLaughlin et al.[92] conducted a study in a cohort of normal, overweight, and obese individuals to determine the relationship of body fat and cardiovascular disease risk. Adiposity (BMI) and cardiovascular risk factors such as blood pressure, plasma glucose, fasting plasma lipid, lipoprotein concentrations and fasting insulin levels were measured. Elevated BMI contributed to higher low-density lipoprotein concentrations. Insulin resistance, which may not be obvious in people with elevated BMI, contributes to the development of cardiovascular disease.

Another study found that adiposity as measured by waist hip ratio (WHR) is a predictor of coronary heart disease. Waist circumference which is a measure of abdominal obesity is a marker of coronary heart disease in women. Women with a WHRof 0.76 in were twice more likely to develop coronary heat disease and women with a WHR greater than 0.88 were 3 times more likely to develop coronary heart disease. Waist circumference and WHP were independently associated with coronary heart

disease after controlling BMI. This suggests that abdominal obesity is a major risk for heart disease[18].

LEPTIN

Leptin is an important regulator of food intake and energy expenditure. It is a protein hormone produced by the adipocytes which was identified in December 1994 by Friedman and co workers. It was first isolated in mice models with the sequencing of the obese gene[22]. The mouse ob gene now called the leptin gene derived its name from the Greek word leptos meaning thin. Leptin is a 16KDa protein product encoded from a 4.4 kilo base mRNA[22] and is synthesized in white and brown adipose tissue[93-95]. Leptin plays an important role in the regulation of food intake and energy expenditure[96-98]. When recombinant leptin is administered to lean mice, it results in a substantial loss of body weight and fat, reduced food intake and increased energy expenditure[97, 99].

Martin et al.,[100] observed a positive correlation of leptin levels with the energy expenditure when the percentage body fat was controlled in cohort of normal weight women. Leptin levels were associated with percent body fat, fat mass and body mass index[101].

Mechanism Of Action

The action of leptin takes place in the hypothalamus in the central nervous system[102, 103] where leptin binds to its receptors inhibiting food intake and decrease body

weight. It has been identified that there are three distinct steps by which the leptin acts as a regulator of food intake. First, leptin is produced by the adipose cells in to the blood stream and it monitors the levels of energy stores in the body. Secondly, leptin signals the adipose stores to the hypothalamus through leptin receptors. Finally, the sympathetic nervous system controls the intake of energy and energy expenditure and maintains a balance in energy. This feedback to the hypothalamus plays an important role in maintaining body weight[104, 105].

Leptin receptors (OB-R) mRNA are expressed in the hypothalamus and in other tissues such as kidneys, lungs, liver, heart, small intestine, pancreas, adipose tissue, spleen, testes, and ovaries[106]. Leptin receptor gene expression has been found to be an important regulator of leptin in the hypothalamus. The expression of receptor mRNA was higher in ob mice which do not produce leptin when compared to lean mice. The administration of recombinant murine leptin in ob type mice decreases the intake of food associated with reduced levels of receptor mRNA in arcuate region of the hypothalamus[105]. This suggested that brain acts as an important site for the action of leptin.

Decreased circulating soluble leptin receptor levels have been associated with obesity in humans. Ogier et al.[107] observed that obesity in humans is associated with a decrease in circulating soluble leptin receptor levels. This study was conducted in obese and lean men and women to determine the soluble leptin receptor levels, it was observed that the soluble leptin receptor levels were lower in obese and overweight individuals when compared to lean subjects. An inverse correlation of soluble leptin receptor levels with percent body fat and leptin was observed. The ratio of circulating leptin to soluble

leptin receptor levels correlated strongly with percent body fat. After a 3-month low calorie diet, soluble leptin receptor levels in obese subjects increased with a decrease in body fat. The leptin levels were higher in women when compared to men and strongly correlated with body fat. Thus, this study suggested that soluble leptin receptor levels increase when there is a decrease in fat mass and weight loss in lean subjects.

Leptin And Obesity

Human obesity is characterized by an increase in serum leptin concentrations associated with an increase in leptin mRNA concentration in adipose tissue as well as an increase in total body fat. Serum leptin concentrations and leptin mRNA content of adipocytes are twice as high in obese subjects as in normal subjects. Serum leptin concentrations are positively correlated with percent body fat[25]. A number of studies[23, 24] conducted in humans has shown that the amount of body fat is the principal determinant of the circulating levels of leptin in humans.

Leptin And Body Fat Distribution

In human obesity, serum leptin concentrations are correlated to subcutaneous adiposity[24, 108] but not to intra-abdominal fat[109, 110]. Subcutaneous fat may be the major contributor of increased leptin concentration. Minocci et al.,[24] determined that subcutaneous fat and preperitoneal visceral fat was assessed. They found that the subcutaneous fat thickness, abdominal fat index and the waist hip ratio correlated independently with the serum leptin concentrations and were higher in women than men. There was no significant relationship between the preperitoneal visceral fat and leptin

concentrations. These results are in agreement with those reported by Vanessa and colleagues[111] in an earlier study. In a group of obese and non obese women, the secretion rate of leptin from subcutaneous tissue was two or three times higher than visceral adipose tissue.

In human studies, serum leptin concentrations have been reported to correlate with the percentage of body fat [25, 112]. A study conducted in normal and obese men and women assessed the percentage of body fat by bioelectric impedance analysis. This showed that elevated serum leptin concentrations were associated with an increase of percent body fat in obese individuals. Decreased serum leptin was also observed due to reduction in body weight. This study also found that the amount of ob mRNA in adipocytes was higher in obese subjects when compared to normal subjects.

Leptin And Systolic Blood Pressure

In rats, high doses of leptin have been shown to increase mean arterial pressure and hypertension [113, 114]. It was suggested that a possible mechanism that may be involved in elevated arterial pressure may be due to the activation of central nervous system. These results are in agreement with a later study conducted by Marcelo and his colleagues [115] who showed a positive correlation of leptin with arterial pressure. But the effect of leptin administration on blood pressure has not yet been studied in humans.

Guagnano et al.[116] have shown the association of serum leptin levels with casual blood pressure and 24-hour ambulatory blood pressure in obese Italian women. In a cohort of 40 women with android type obesity and 30 women with gynoid type obesity, ambulatory blood pressure was monitored for 24 hours with an interval of 15 minutes

during the day and 30 minutes during the night and compared with 20 nonobese healthy women. Casual blood pressure was measured 3 times at 5 minute intervals in the morning. Waist hip ratio (WHR) was used to differentiate between android (abdominal) and gynoid (peripheral) fat distribution. The WHR greater or equal to 0.86 was defined as android obesity and a WHR lesser or equal to 0.86 was defined as gynoid obesity[117]. It was found that the serum leptin levels were significantly higher in women with android obesity than women with gynoid obesity. There was a strong positive correlation between leptin levels and 24-hr ambulatory blood pressure in women with android obesity.

Schutte et al.[118] conducted a study in a cohort of hypertensive obese/overweight African and normotensive obese/overweight women. The effect of leptin on blood pressure and arterial compliance was investigated. Arterial compliance was measured by stroke volume over pulse pressure. Stroke volume is the volume of blood ejected from the ventricle with each beat of the heart. Pulse pressure is the change in blood pressure during the contraction of the heart. It was observed that leptin levels were higher in obese/overweight hypertensive and normotensive women when compared to lean women but similar in the normotensive and hypertensive obese overweight groups. It was observed that leptin positively correlated with SBP and pulse pressure only in the obese/overweight hypertensive group. Leptin levels also correlated negatively with arterial compliance thus suggesting a positive role of leptin in the development of cardiovascular disease.

Suter et al.[119] conducted a study in a group of overweight subjects with hypertension and healthy non-hypertensive subjects. The effect of plasma leptin levels

on blood pressure and heart rate in these subjects was investigated. There was a significant correlation of plasma leptin levels with systolic blood pressure in all women, after adjusting for body weight. Heart rate correlated significantly with leptin levels in all subjects. It was suggested that the relationships were statistically not very strong and may be due to the heterogeneity of study population.

Cardiovascular Disease And Leptin

Leptin is a novel, independent risk factor for the progression of disease of the heart[120]. Soderberg et al.[19] observed that higher leptin levels, total cholesterol and Apo-I predicted acute myocardial infarction in a cohort of obese Swedish men. High leptin levels were positively associated with high BMI, high blood pressure and high insulin levels. In the 5 yr follow up West of Scotland Coronary Prevention Study, leptin levels were examined in subjects who experienced coronary events and compared with controls. It was observed that with an increase in leptin levels, the relative risk of a future coronary event increased[20].

Ciconne et al.[21] showed that the concentration of human plasma leptin is independently associated with the intima-media thickness of the common carotid artery. This was studied in a cross-sectional sample of obese men and women aged 18-45 years of age. Intima media thickness of the common carotid artery was quantified by high resolution B-mode ultrasound imaging was positively correlated with leptin, age, BMI and waist circumference and negatively correlated with insulin sensitivity in men and women.

A cross-sectional study was conducted in a cohort of men and women with type-2 diabetes to determine the levels of plasma leptin and its association with coronary atherosclerosis. Coronary artery calcification (CAC), which is a measure of atherosclerosis, was measured in these subjects using ultra fast computed tomography. Plasma leptin levels were significantly associated with coronary artery calcification after controlling age,gender,BMI and CRP levels suggesting a positive role of plasma leptin levels in the progression of cardiovascular events[121]. In The Atherosclerosis Risk in Communities (ARIC) study[122], the incidence of coronary heart disease was higher at higher carotid intima media thickness. Thus, it is possible that leptin through its effects on calcification and carotid intima media thickness, is a predictor of coronary heart disease incidence.

Schulze et al.[123] conducted a study in a group of 53 patients with chronic heart failure. Serum leptin concentrations and serum concentration of soluble leptin receptor was measured in these subjects. It was shown that the leptin concentrations and concentrations of leptin receptor were higher in patients with congestive heart failure when compared with the healthy controls. It was also found that there is a strong positive correlation of serum leptin and TNF-α in patients with severe exercise intolerance. This study suggests that increased concentrations of leptin and soluble leptin receptor in patients with congestive heart failure may be due to increased levels of proinflammatory cytokines.

Interrelationship Of Inflammation, Cardiovascular Disease And Leptin

Inflammation is involved in the etiology of cardiovascular diseases particularly atherosclerosis, ischemic heart disease and heart failure[124-126]. C-reactive protein, a marker of inflammation and an important indicator of cardiovascular risk[127-131] is associated with a sequence of cardiovascular events in patients with acute coronary artery disease, angina pectoris and myocardial infarction in healthy men and women. Increased levels of leptin are associated with increased CRP[132]. In a random sample of Finnish men and women, associations of CRP, IL-6 and TNF α factor with coronary heart disease incidence events and cardiovascular disease events were analyzed. The cohort was followed up for a period of 10 years. Increased CRP levels and TNF α factor were significantly associated with the risk factors for the incidence of coronary heart disease and cardiovascular disease such as total cholesterol, hypertension and triglycerides in men when compared to women[133].

Inflammation And Atherosclerosis

Atherosclerosis is an inflammatory disease[134]. Coronary atherosclerosis is a disease of the coronary arteries characterized by a combination of changes in the intima of arteries. Fatty granulomatous lesions develop in the arterial wall and lead to the hardening of vessels. The lesions, known as atheroma derived its name from the Greek word meaning “gruel”, are characterized by thickening of the intimal wall, deposition of lipid, deformation and fragmentation of the internal elastic membrane. In advanced cases, it results in fibrosis and calcification. Atheromas have 2 major constituents namely, fat and fibrous tissue which gives rise fatty plaques and pearly plaques. As a

result of the formation of atheroma, it can erode the wall of the artery and diminish the elasticity of the artery. Thus thrombosis or blockage of the arteries takes place. Hence, the symptoms and signs of ischemic heart disease occurs[135]. According to the National Cholesterol Education Program, increased low density lipoprotein (LDL) results in the storage of lipids which are involved in an ongoing inflammatory response[136].

Ross et al.[137] proposed that endothelial dysfunction leads to atherosclerotic lesions. The possible causes of this dysfunction are due to hypertension, cigarette smoking, elevated LDL levels and diabetes mellitus. Endothelial dysfunction results in the alteration of the homeostatic properties of endothelium. At the point of injury, when the inflammatory response does not remove the offending agents effectively, then these responses stimulates the proliferation of the smooth muscle cells that becomes intermixed at the area of inflammation and forms an intermediate lesion. When these responses continue, the formation of lesions leads to further enlargement that thickens the artery wall. The artery wall prevents the thickening by the process of dilation, so that up to a point the lumen remains unaltered. During worse conditions, the lesion may then intrude in to the lumen and alter the flow of blood when the artery wall can no longer dilate.

Van Dielen et al.[138] in a cohort of morbidly obese individuals examined the association of obesity, leptin and the development of an inflammatory state. Inflammatory markers such as soluble TNF-α receptors, acute phase proteins, and lip polysaccharide binding protein, serum amyloid, CRP, and plasminogen activator inhibitor-1 (PAI-1) were measured. Leptin concentrations significantly correlated with BMI in these subjects. It was observed that there was a significant correlation of leptin with TNF α receptors such as TNFr55 and TNFr75. Thus it was concluded that in obese

subjects, an increase in leptin concentrations was associated with the increase in inflammatory markers suggesting a positive role of leptin in the regulation of inflammation[139, 140].

INSULIN RESISTANCE

Insulin Resistance And Body Fat

Insulin resistance is a condition in which the peripheral target tissues are unable to respond properly to normal circulating concentrations of insulin leading to hypersecretion of insulin by the pancreas[141]. The process by which insulin action takes place is by binding to insulin receptors on target cells. This results in the uptake of glucose through the glucose transport mechanism and its subsequent metabolism in insulin sensitive tissues[142, 143]. So in insulin resistant condition, there is a substantial increase of insulin production in the attempt to maintain a normal rate of glucose uptake.

Abdominal body fat is closely associated with insulin resistance. Carey et al.[144] conducted a study in normal and obese women to determine the relationship of insulin resistance and abdominal obesity in these groups. Obese women with abdominal subcutaneous fat as opposed to peripheral non-abdominal fat showed a stronger relationship with insulin sensitivity. These results were consistent with other cross sectional studies conducted in humans that demonstrated a positive correlation of insulin resistance with abdominal adiposity. Researchers observed the association between race, sex, abdominal obesity, hyperlipidemia and fasting insulin levels in black males and

white males with hypertension. The fasting insulin levels were higher in black males when compared to white males. Abdominal obesity and hyperlipidemia significantly correlated with fasting insulin levels in black males when compared to white males. Abdominal obesity was also associated with insulin sensitivity in black males. Hence it was suggested that abdominal obesity or hyperlipidemia doubled the risk of hyperinsulinemia[145]. But the mechanisms of insulin resistance in obesity are not fully understood.

Abdominal fat can accumulate either viscerally or subcutaneously. Many investigators[146-149] have found that excess accumulation of subcutaneous fat is more strongly associated with insulin resistance than visceral fat. Kelley et al.[147] partitioned abdominal subcutaneous adipose tissue in to depots of fat namely, superficial subcutaneous adipose tissue (posterior half of the abdominal wall) and deep subcutaneous adipose tissue (anterior half of the abdominal wall). A cross sectional abdominal computed tomography was performed in a cohort of lean and obese men and women. There was a strong positive correlation between insulin resistance and deep subcutaneous adipose tissue. Insulin resistance was also strongly related to visceral adipose tissue. Similarly, researchers found that central abdominal fat in obese early postmenopausal women is a strong correlate of insulin resistance. Intraabdominal fat and subcutaneous fat was inversely and independently related to insulin sensitivity after adjusting for total fat[150].

The accumulation of visceral fat contributes to insulin resistance. Ross et al.[151] investigated the relationship of insulin resistance between visceral and total abdominal adipose tissue and muscle composition in a cohort of 40 abdominally obese

premenopausal women. Abdominal fat, which includes visceral and subcutaneous adipose tissue, was measured using magnetic resonance imaging. Insulin mediated glucose disposal rate was measured by hyperinsulinemic euglycemic clamp. It was observed that abdominally obese subjects had higher visceral adiposity when compared to abdominal adiposity. There was also a strong association of glucose disposal rate to visceral adipose tissue when compared to abdominal adipose tissue. Additionally, the association of visceral adipose, subcutaneous adipose tissue, total fat mass, physical activity expenditure, and peak VO_2 with glucose uptake in obese postmenopausal women was examined. Visceral adipose tissue was inversely related to glucose uptake with higher visceral adipose tissue levels at lower glucose disposal per Kg lean body mass[152].

Upper body fat is associated with insulin resistance due to its relationship with non-esterified fatty acids from adipose tissue. Several studies suggest that upper body obesity is associated with increased free fatty acids release[153-156]. In a study of upper body obese women, lower body obese women and non obese women. It was shown that insulin resistance was associated more with upper body fat than lower body fat. The difference in body fat distribution was associated with abnormalities in the release of free fatty acids metabolism. Palmitate turnover, which is a measure of lipolysis in adipose tissue, was observed to be higher in upper body obese women than lower body obese women[156]. A decline in glucose uptake is associated with an increase in free fatty acid concentration. Decreased glycogen synthesis and carbohydrate oxidation was also observed with reduced glucose uptake. The possible mechanism for decreased glucose uptake is suggested to be due to lowered glucose transport or phosphorlyation in subjects with increased free fatty acids[157].

Roden et al.[158] observed plasma concentrations of free fatty acids, increased by an infusion of a triglyceride emulsion combined with heparin to activate lipoprotein lipase using carbon-13 and phosphorus NMR spectroscopy techniques in 9 healthy subjects. A decreased rate of muscle glycogen synthesis by 50% and a decreased rate of glucose uptake by the whole body by 46% of control values were observed. Elevated plasma free fatty acids inhibited glucose uptake with a reduction in glucose oxidation rate and muscle glycogen synthesis. This suggested that free fatty acids play an important role in insulin resistance. However, the mechanism for this is uncertain.

A study was conducted in a group of obese and lean Caucasian women to investigate the relationship between insulin sensitivity and intra-muscular triglycerides (mTG) and saturated free fatty acids. There was a negative correlation of mTG and insulin mediated glucose uptake in obese women when compared to controls. Saturated fatty acids were higher in obese women than in controls. Increased mTG with saturated fat decreased the rate of glucose uptake in obese women[159].

Deposition of fat in the thigh region is also a marker of insulin resistance in obese subjects with type-2 diabetes[146]. Mid thigh adipose tissue measured by tomography was divided in to three compartments in obese and lean subjects. They were subcutaneous adipose tissue (SCAT), adipose tissue beneath fascia (SFAT), adipose tissue infiltrating muscle groups. Adipose tissue around and between the skeletal muscle strongly correlated to insulin resistance along with adipose tissue beneath the fascia[160].

Insulin Resistance And Cardiovascular Disease Risk

Insulin resistance measured by euglycemic insulin clamp glucose disposal rate is predictive of congestive heart failure[26]. The severity of coronary heart failure was assessed by peak (VO_{2max}), an index of cardiopulmonary function derived from maximal exercise test in men with chronic heart failure, coronary heart disease and in controls[161]. Chronic heart failure progressed with a marked increase in insulin resistance and reduced peak Vo_2.

In the San Antonio Heart Study which was in conducted in Mexican-Americans and Non-Hispanic white women for a period of four years, HOMA-IR was positively associated with anthropometric measures and cardiovascular risk factors such as HDL, cholesterol, triglycerides, systolic and diastolic blood pressure[162].

In the Strong Heart Study of American-Indian communities, the relation of insulin resistance to echocardiographic markers of cardiovascular disease was studied. Simone et al.[163] measured insulin resistance by HOMA IR, left ventricular mass, left Ventricular functioning, stroke volume/pulse pressure ratio and arterial compliance. Insulin resistance was significantly associated with decreased arterial compliance in both men and women.

The Framingham study of women free of myocardial infarction was examined for the relationship between insulin resistance and echocardiogaraphic left ventricular measurements. It was observed that left ventricular mass and left ventricular wall thickness correlated positively with HOMA-IR in women when compared with men. This is due to an increase in HOMA-IR with increased obesity in women[164].

The study on women was conducted by Folsom et al.[165] in a cohort of middle aged women from four U.S communities. Fasting insulin levels were measured in these groups. The subjects developed coronary heart disease after 4-7 years of their study. There was a positive correlation between coronary heart disease and fasting insulin. Fasting insulin was a risk factor for coronary heart disease in black and non-black middle aged women.

In addition, a study was conducted in a group of 15,000 men and women to investigate the relationship between elevated fasting insulin levels and incidence of ischemic stroke. The subjects were for 6-8 years for the incidence of ischemic stroke. Fasting insulin levels and waist to hip ratios were determined in this cohort. The incidence of ischemic stroke correlated with elevated levels of insulin and higher waist to hip ratios[166]. This study agrees with an earlier report by Laakso [167] who reported that fasting insulin levels is a good marker of insulin resistance in non-diabetic subjects.

Some studies have also not shown any significant correlation of insulin resistance measured by euglycemic/hyperinsulinemic clamp and oral glucose tolerance test with the development of cardiovascular disease[168, 169].

Relationship Between Leptin And Insulin Resistance

In population based studies, serum leptin concentrations have been observed to correlate with insulin sensitivity. De Courten et al.[170] in his study of western Samoans observed that serum leptin concentrations positively correlated with insulin resistance as measured by oral glucose tolerance test clamp, BMI, fasting insulin and mean blood pressure.

Doehner et al.[171] conducted a study to investigate the relationship between insulin resistance, norepinephrine, TNF-alpha and hyperleptinaemia. The study compared patients with chronic heart failure with healthy subjects**.** There was an inverse correlation of insulin sensitivity and leptin levels in subjects with chronic heart failure. Thus it is suggested that elevated leptin levels would directly and independently predict insulin resistance in subjects with ischemic heart disease.

WAIST CIRCUMFERENCE

Waist Circumference

The sex-specific waist circumference cutoff points used by the NIH are a WC of 102 cm (40 inches) in men and 88 cm (35 inches) in women. Patients are categorized according to their cut off points to be normal when WC is below 102 cm and high above 102 cm for men. Women are categorized to be normal with a WC of 88cm and high when the WC is above 88 cm [172].

In the Third National Health and Nutrition Survey(1988-1994), subjects were classified by BMI and waist Circumference in accordance with NIH guidelines, Subjects with WC values greater than the cut off points(WC >102 for men and WC >88cm for women) had hypertension, diabetes, dyslipidemia and metabolic syndrome compared with normal subjects with WC values below the cutoffs. Subjects with increased WC values showed greater health risk for heart disease[173].

Some authors have suggested a graded system for the assessment of health risk using WC. Han et al.[28] determined the frequency of cardiovascular risk factors in a random sample of men and women and proposed that WC values less than 94cm in men and of less than 80 cm in women were at low health risk. Those ranging from 94 to 102 cm in men and 80 to 88 cm in women were of moderate health risk. Those greater than 102 cm in men and greater than 88 cm in women have a health risk for cardiovascular disease. When cardiovascular risk factors such as systolic and diastolic blood pressure, total plasma cholesterol concentration were measured, men and women with waist circumferences exceeding 94 cm and 80 cm respectively had one cardiovascular risk factor.

Waist circumference is correlated with obesity and cardiovascular disease risks more than BMI. Obesity related risks such as low HDL, high LDL, high blood pressure, and high glucose values were more strongly associated with WC than BMI in a cohort of white men and women. A WC of 90 cm for men and 83 cm for women corresponded to an equivalent in risk of BMI of 25, whereas a WC of 100 cm for men and 93 cm for women was equivalent in risk to BMI of 30 [174].

Lofgren and his colleagues[27] conducted a study in a cohort of overweight or obese premenopausal women. The association of biomarkers for coronary heart disease with waist circumference and BMI was evaluated. Plasma biomarkers such as lipids, apolipoproteins, LDL peak diameter, LDL susceptibility to oxidation, glucose, leptin and insulin were measured. A strong positive correlation between BMI and WC was observed. A majority of the subjects who had a BMI < 30 Kg/m^2 had a WC>88 cm (92.6 ± 3.7 cm). The subjects with WC > 88cm had higher diastolic pressure, higher plasma

TG and apo C-III concentrations which are associated with coronary heart disease. Although, BMI also showed a positive correlation with all the risk factors in these subjects, WC in these subjects showed stronger association with coronary heart disease. This suggest that WC can be used as reliable tool to assess the risk for coronary heart disease[27]. This finding is consistent with another study conducted by Janssen and colleagues[175] who found that WC alone was a better predictor of cardiovascular disease. Thus for a given value of Waist circumference in overweight and obese person and normal weight persons, there is an increase risk for cardiovascular disease.

Waist circumference is an indirect measurement of abdominal fat which includes both visceral and subcutaneous fat. In the 1981 Canada Fitness Survey, the relationship of cardiorespiratory fitness (CRF) with waist circumference, sum of trunk skin folds (an index of central adiposity) and sum of five skinfolds, (an index of abdominal adiposity) was examined. Cardio respiratory fitness was measured by maximal oxygen uptake ($VO_{2max)}$ during an exercise test. Higher cardio respiratory fitness was strongly associated with lower waist circumference for a given BMI. Lower sum of trunk skinfolds and sum of five skinfolds were observed in groups with high CRF[176]. Janssen et al.[177] determined the correlation of abdominal and non-abdominal fat to waist circumference and BMI in a cohort of white men and women. Waist circumference was independently correlated with abdominal fat, non-abdominal fat and visceral fat in obese men and women. It was observed that an increase in waist circumference with increasing abdominal fat is specifically contributed by visceral fat. The ability of waist circumference to predict obesity related health risk is explained by visceral fat.

According to Mosca et al.[178] WC can be used as a surrogate measurement for cardiovascular disease risk in women because it is highly correlated with the global Framingham risk score, cardiovascular disease and diabetes. These findings are consistent with the findings of Kato et al.[179], who conducted a study in a cohort of 62 female patients with schizophrenia. An increased waist circumference was associated with cardiovascular risk factors such as hypertension, dyslipidemia, abdominal serum glucose. Waist circumference is a non-invasive, economical and practical tool for the identification of women with risk for cardiovascular risk.

CHAPTER III

METHODOLOGY

Hypothesis

The following hypotheses were formulated for this study:

1. There is an ethnic difference between leptin levels, insulin resistance and waist circumference in American Indian an African American women.
2. Blood leptin concentration is positively correlated with age, systolic blood pressure and total cholesterol in American Indian and African American women.
3. Insulin resistance (Homa-IR) is positively correlated with age, systolic blood pressure and total cholesterol in American Indian and African American women.
4. Waist circumference is positively correlated with age, systolic blood pressure and total cholesterol in American Indian and African American Women.
5. Waist circumference will be positively correlated with Insulin resistance (Homa-IR) in both ethnic groups.

Assumptions And Limitations

Several assumptions have been made in order to proceed with this analysis. The first assumption is that subjects were truthful with the information they provided. The second assumption is that HOMA IR is a valid measure of insulin resistance in AI and and AA women. The third assumption is that the subjects in the study were truly fasting when blood samples were taken. The fourth assumption is that all the subjects were

similar in that they did not have any underlying condition or health problem that might confound the outcomes. The final assumption is that results of lab analysis were accurate.

Potential limitations for this design include missing data because not all values for each subject may have been obtained. In addition, the sample is a convenience sample based on their availability and willingness to participate. Therefore, this is not a random selection of subjects. This sample is a not a representative sample of the whole American Indian and African American population but is a good representation of American Indian and AA women from rural Oklahoma.

Primary Data Collection Methods

Data was collected by researchers and trained staff at local community centers, clinics and tribal complexes between May, 2001 to Spetember, 2002 .

Eighty one women were recruited from the local communities of Binger, Boley, Anadarko, Langston, Wewoka, El reno and Lawton . Local contact persons such as tribal leaders, community center staff, extension specialists and medical clinic workers assisted with distributing information regarding the health study eligibility requirements and with recruiting of participants. Written consent to participate in the study was obtained in accordance with the guidelines of the Institutional Review Board for Human Research at Oklahoma State University and Langston University.

Inclusion And Exclusion Criteria

Women of child bearing age were included in the study. Subjects who were missing more than 25% of data were excluded. After consent was obtained, fasting

venous blood samples were drawn by licensed phlebotomists. Anthropometric measurements include weight, height, triceps skin fold thickness, and bioelectric impedance assessment (BIA) of body compositions were done by the same person to maintain precision. Finally, the women were interviewed by research staff to obtain information on diet, lifestyle habits, and socioeconomic background.

Anthropometric Measurements And Body Composition Determination

Standing barefoot height was obtained for each participant using a portable stadiometer, Seca model 214 Road Rod, (Seca Corporation, Hanover, MD). Weight and body composition were then measured utilizing a bioimpedance scale(Tanita Body Composition Analyzer/Scale Model TBF-310,Tanita Corporation of America,Inc.,Arlington Heights,IL). Body mass Index was calculated as Kg/m^2. Midarm circumference was measured using nonstretch tape on the non-dominant arm midway between the acromion process and tip of olecranon. Triceps skin fold measurement was done on the posterior side of the non-dominant arm. It was measured over the triceps muscle and the midway between the acromion of scapula and olecranon of the ulna. On the lateral side of the arm, the midpoint of acromion and olecranon was marked with the elbow placed at 90 degrees. The skinfold was grasped by the measurer with the thumb and index finger of the left hand. The tip of the caliper was about 1cm or 0.5 in. from the thumb and finger. The caliper was placed perpendicularly to the long axis of the cylinder and 3 readings were noted down from the dial and averaged (Lange skin fold caliper operation manual, Beta technology Inc., Santacruz, CA, U.S.A). Waist

circumference was measured by locating the navel and a non stretch tape was placed around the abdomen without compression on the skin.

Biochemical Analysis

Leptin concentrations were determined using an enzyme linked immunoassay (ELISA) Kit (Linco Research, St.Charles, Missouri). Human leptin molecules from samples were captured on the pretreated plates. These molecules are transferred to a microtiter plate coated by polyclonal rabbit anti-human leptin molecules. Unbound materials from the samples were removed by washing with the buffer. Biotinylated monoclonal antibodies were added to bind the captured human leptin, after which alkaline phosphatase was added to the biotinylated antibodies. The immobilized antibody enzyme conjugates were quantified by monitoring alkaline phosphatase activity in the presence of substrate p-nitrophenyl phosphate. The enzyme activity was measured spectrophotometrically by the increased absorbency at 405 nm. Increased absorbency is proportional to the amount of captured human leptin in the participants' samples.

Serum insulin concentrations were determined by radioimmunoassay (RIA) kit (Linco Inc). A fixed concentration of labeled tracer antigen was added to the samples taken from participants. The antiserum was then added and incubated with a mixture of sample antigen and radio-labeled antigen. Due to limited binding sites on the antibody, the unknown antigen reduces the amount of available tracer antigen that may bind to the antibody. The fractions of bound and free labeled antigen (insulin) were measured to determine the amount of insulin in the unkown sample. In this assay I-labeled human insulin and human insulin anti-serum was used. The calculation for the amount of human

insulin present in each sample was automatically performed by gamma counter at Oklahoma state University's Human Nutrition Laboratory.

Colorimetric assays of fasting serum glucose, total cholesterol, HDL cholesterol, and triglycerides were performed on a Roche Cobas Fara Centrifugal Analyzer (Roche,) using commercially available kits.

Estimation Of Insulin Resistance By HOMA (Homeostasis Model Assessment)

Matthews et al.[180] developed a mathematical model to predict the homeostatic concentrations of glucose and insulin beta-cell deficiency in varying states of insulin resistance. The quantitative assessment of insulin resistance was done utilizing fasting insulin and glucose concentrations (HOMA) using the following mathematical formula:

(HOMA IR) = Ln (Fasting Insulin * Fasting Glucose /22.5)

Fasting Insulin units are in μU/mL and Fasting Glucose in mg/mL.

HOMA has been validated as a reliable predictor of insulin resistance in humans. The estimation of insulin sensitivity by HOMA is significantly and highly correlated with other measures of insulin sensitivity such as hyperglycemic and euglycaemic clamps and intravenous glucose tolerance test. It must be noted that HOMA is merely an estimation of insulin resistant state and not as sensitive or precise as the clamp technique. It is however, less invasive, less time consuming, less costly, requires less personnel and equipment[180-185].

Normal Values

Normal leptin levels for lean women ranges from 7.4±3.7ng/ml. Normal blood pressure is 120/80 mm Hg. HDL-C ranges from 40-60 mg/dL. Normal total cholesterol is expected to be less than 200 mg/dL. Normal fasting insulin levels are expected to be between 6.0-27.0µU/ml and normal fasting glucose levels at 70-110mg/dL. Normal HOMA IR values are expected to range from 1.45-2.8.

Statistical Analysis

Statistical Analysis Software for Windows (SAS 9.1.3 Version, Cary, and N.C) was used to analyze all data. Proc GLM was used to compare differences between American Indian and African American groups. The GLM procedure was used because there are unequal numbers of observations in both groups.

Pearson's Correlation coefficients were used to determine the relationship of cardiovascular risk factors with leptin insulin resistance and waist circumference using PROC CORR procedure of SAS.

The chi-square statistic was used to compare the frequency counts of number of smokers with non smokers in both the groups.

CHAPTER IV

CARDIOVASCULAR RISK FACTORS IN AMERICAN INDIAN AND AFRICAN AMERICAN WOMEN OF CHILD BEARING AGE AND THE RELATIONSHIP OF THESE FACTORS BLOOD LEPTIN CONCENTRATION, INSULIN RESISTANCE AND WAIST CIRCUMFERENCE

Context

Prevalence of health disparities among racial/ethnic groups especially the prevalence of cardiovascular diseases is a major concern in the nation. Leptin concentrations, insulin resistance, and waist circumference are predictors of cardiovascular disease incidence in American Indian and African American women.

Objectives

To determine if there is an ethnic difference in leptin levels, insulin resistance and waist circumference between AI and AA women and to examine the relationship of these variables with cardiovascular risk factors in this cohort.

Design, Setting and Participants

Prospective epidemiologic study of 81 women (48 AI, 33 AA) of child bearing age from rural Oklahoma. Participants were recruited by convenience sampling from the local communities of Andarko, Binger, Boley, El Reno, Langston, Lawton and Wewoka in Oklahoma between May, 2001 to September, 2002.

Main Outcome Measures

Differences in leptin levels, insulin resistance (HOMA IR), and waist circumference in this cohort and correlation of these variables with age, systolic blood pressure, waist circumference and total cholesterol.

Results

Ninety percent of the women were overweight or obese with a mean BMI of 33 Kg/m^2. Of the AI women, 58% were obese and of the AA women, 61% were obese. Leptin levels were significantly different between the two ethnic groups. Leptin concentrations were significantly higher for AA women when compared to AI women. There was no significant difference in waist circumference and insulin resistance measures between these groups. There was a positive correlation of leptin with systolic blood pressure in both groups ($r=0.33$, $p= 0.004$) and waist circumference with systolic blood pressure ($r=0.26$, $p=0.02$) in both groups.

Conclusion

African American women had greater leptin concentrations when compared to AI women. Obesity and overweight were prevalent in this cohort. Blood leptin concentration and waist circumference were correlated with cardiovascular risk factors in AI and AA women of child bearing age. Insulin resistance estimated by HOMA IR was not correlated with cardiovascular risk factors. However HOMA IR correlated with waist circumference and waist circumference correlated with SBP.

Introduction

Cardiovascular disease is major public health concern in the United States with an estimated health care cost of over $300 billion annually due to disability and death[1]. It is estimated that over 71 million adults in the U.S, or 1 in 3 live with some form of cardiovascular disease [1]. According to the CDC, heart disease and stroke are the principal components of cardiovascular disease and accounts for up to 40% of all deaths in the nation[2]. Cardiovascular disease is the leading cause of death in both genders among racial and ethnic groups[3]. Heart disease is the leading killer of minority women in the United States[4]. According to the Framingham risk score, risk factors that increase the likelihood of developing cardiovascular disease are age, gender, systolic blood pressure, HDL cholesterol, total cholesterol, and smoking [5].

Of all the minority groups, African American women tend to develop high blood pressure at a younger age[6]. They are also less likely to engage in physical activity resulting in obesity and diabetes. The prevalence of obesity is high among AA women. A Montana study has shown that American Indians are at a higher risk of developing cardiovascular disease compared to whites[7]. American Indian women develop obesity and overweight at a very earlier stage. This is due to an excess accumulation of fat in childhood among AI's[8].

Cardiovascular disease is the leading rankable cause of deaths in Oklahoma among AI and AA women. Overall, this accounts for 40% all deaths in United States and 44% of all deaths in Oklahoma[9]. In Oklahoma, the age adjusted death rate due to heart disease was higher in African Americans when compared to American Indians between 1991-1995. The likelihood of women in Oklahoma dying from heart disease is 50%

higher than from cancer[10]. Coronary heart disease accounts for one in five deaths in women in Oklahoma and was also the leading cause of death among females in Oklahoma in 2003[11]. In addition, risk factors such as smoking, high blood pressure, obesity and inactive lifestyle are a problem in Oklahoma. It was calculated that of heart disease deaths among women in Oklahoma smoking accounted for 25%, obesity accounted for 32%, sedentary lifestyle accounted for 35% and high blood pressure accounted for 29% of heart disease deaths[12].

Human obesity is characterized by an increase in serum leptin concentrations associated with an increase in leptin mRNA concentration in adipose tissue as well as an increase in total body fat[13-15]. Leptin has been shown to increase arterial pressure and blood pressure in obese subjects[16, 17]. Furthermore, it has been reported that leptin is positively correlated systolic blood pressure (SBP) and pulse pressure in hypertensive obese/overweight African women[18]. In addition, in women with android type of obesity serum leptin levels were significantly higher[19] than women with gynoid obesity. Researchers have proposed that the heredity and genetics influence the propensity for developing heart disease in AI and AA[20].

Insulin resistance is predictive of congestive heart failure and is associated with more severe disease and prognosis[21]. Abdominal body fat is closely associated with insulin resistance and decreased glucose disposal rate in obese patients[22, 23]. Waist circumference which is a measure of abdominal obesity is a predictor of coronary heart disease in women[24, 25].

In this study, it was hypothesized that differences existed in leptin levels, insulin resistance and waist circumference exist between AI and AA women and that these

variables were correlated with cardiovascular risk factors in this cohort of women of child bearing age.

Methods

Design and Study Cohort

A community based sample of AI and AA of child bearing age, were recruited from rural Oklahoma communities of Anadarko, Binger, Boley, El Reno, Langston, Lawton, and Wewoka, Oklahoma. Written consent to participate in the study was obtained in accordance with the guidelines of the Institutional Review Board for Human Research at Oklahoma State University and Langston University. Data was collected by researchers and trained staff at local community centers ,clinics and tribal complexes between May,2001 to September,2002. Inclusion criteria were AI or AA women with age between 22 and 45 with children. Subjects with a BMI greater than 52 (outliers) were excluded in this study and subjects who were missing more than 25% of data were excluded.

Anthropometric Measurements and Body Composition Determination:

Standing barefoot height was obtained for each participant using a portable stadiometer, Seca model 214 Road Rod, (Seca Corporation,Hanover,MD). Weight and body composition were then measured utilizing a bioimpedance scale(Tanita Body Composition Analyzer/Scale Model TBF-310,Tanita Corporation of America,Inc.,Arlington Heights,IL). Body mass Index (Kg/m^2) was calculated. Waist circumference was measured in the location of the navel with a non stretch tape placed around the abdomen without compressing the skin.

Biochemical Analysis:

Fasting blood samples were drawn, placed on ice, transported to the lab, processed, and stored on the same day. Leptin concentrations were determined using an enzyme linked immunoassay (ELISA) Kit (Linco Research, St..Charles, Missouri). Serum insulin concentrations were determined by radioimmunoassay (RIA) (Linco Inc). Colorimetric assays of fasting serum glucose, total cholesterol, HDL cholesterol, and triglycerides were performed using a Roche Cobas Fara Centrifugal Analyzer (Roche) and commercially available kits.

Formula and normal values

The normal fasting range of leptin for lean women ranges with BMI of 18-25 is 7.4±3.7ng/ml (Linco Kit). Normal fasting insulin levels are expected to be between 6.0-27.0μU/ml and normal fasting glucose levels between 70-110mg/dL. Waist circumference cutoff is 88 cm (35 inches) in women[26] (NIH). Normal blood pressure is 120/80 (AHA). Normal HDL- cholesterol level ranges from 40-50mg/dL (AHA). Glucose and insulin concentrations were used in calculating the HOMA-IR, as an index of insulin sensitivity which was calculated as follows[27].

HOMA IR=ln [insulin(μU/mL)*glucose(mg/mL)/22.5]

Normal expected values for HOMA –IR ranges from 1.45-2.8. As HOMA-IR value increase, insulin resistance increases.

Statistical Analysis:

Statistical Analysis Software for Windows (SAS 9.1.3 Version, Cary NC) was used to analyze all data. Proc GLM was used to compare differences between AI and AA women. Pearson's Correlation coefficients were used to determine the relationship of

cardiovascular risk factors with leptin insulin resistance and waist circumference. The chi-square statistic was used to compare smoking frequency between both the groups.

Results

Of the 81 subjects, there were 33 AA and 48 AI women. Table 1 lists values for the variables from each ethnic group. No significant difference was observed between the groups for age, BMI, waist circumference, total cholesterol, HDL cholesterol, systolic blood pressure, HOMA IR, and, percent body fat listed in (Table.2). Mean age was 34 ± 5 years (Table.2).Mean BMI was 33.2 ± 7.3. Mean waist circumference was 107.2 ± 17.3 (centimeters). Mean total cholesterol was 195.9 ± 40.8 mg/dL. Mean HDL cholesterol was 49.9 ± 16.4 mg/dL. Mean SBP was 125.9 ± 12 mmHg. Mean HOMA IR was 5.0 ± 0.9. Mean percent body fat is 42.8 ± 7.5 (Table 1)

Leptin levels were significantly different between the two ethnic groups. Concentrations were significantly higher for AA women when compared to AI women (Table.1). No significant difference was observed in waist circumference and insulin resistance (HOMA IR) between the ethnic groups although fasting glucose levels were significantly higher in the AI women. Leptin concentrations correlated with indices of body fatness including waist circumference in both ethnic groups. More noticeably, HOMA IR and leptin concentration were significantly correlated in both groups (AI, $r=0.56$, $p=<0.01$, AA, $r=0.57$, $p= 0.01$). HOMA IR was also significantly correlated with BMI, waist circumference ($r=0.45$, $p=<0.01$), and percent body fat in both groups but not with systolic blood pressure. HOMA IR significantly correlated with serum triglycerides in both groups ($r= 0.26$, $p=0.02$).

Correlations between leptin concentrations and age, systolic BP, HDL, total cholesterol are in Table.3. In both AI and AA women, leptin concentrations had significant positive correlation with systolic blood pressure (r=0.33, P=0.01) (fig.1). There was no significant correlation of leptin with age, HDL-C and total cholesterol in these women.

No significant differences in waist circumference and insulin resistance were observed between both groups. Table.4 gives the bivariate correlation coefficients for waist circumference and insulin resistance with age, SBP, HDL C and total cholesterol in both groups. There was no significant correlation between HOMA IR and HDL-C or total cholesterol. The relationship of waist circumference with HDL-C and total cholesterol was not statistically significant in both groups. Waist circumference was significantly correlated with systolic blood pressure (r=0.27, p=0.02) (fig.2). Significantly more AI women smoked than AA women. Fifty one percent of AI women and eighteen percent of AA women were smokers.

Comment

This study was designed to determine differences in leptin levels, insulin resistance and waist circumference between AI and AA women of child bearing age from rural Oklahoma and to determine the relationship of these variables to cardiovascular risk factors such as age, SBP, HDL-C and total cholesterol. It must be noted that, the number of smokers in AI women were higher when compared to AA women and places these women at high risk for cardiovascular disease. Serum leptin and insulin concentrations were above normal values for both groups as well.

This study highlights an important difference in the levels of leptin concentration between AI and AA women of Oklahoma. Despite similarities in waist circumference, BMI and percent body fat, the leptin levels were higher for AA women when compared to AI women. Studies have shown an association between serum leptin concentrations with percent body fat as a major contributor to increased leptin concentration[14, 15, 28]. Increased subcutaneous fat[29]is also associated with increased leptin concentrations. In AA women, the midarm circumference and triceps skinfold measurements was also greater compared to AI women. The Tanita scale may not have been sensitive to body fat in these women. In our group, it is possible that AA women had more subcutaneous fat than AI women. We found positive correlations between SBP and leptin in both the groups. But the correlation was low and there was no difference between SBP among AI and AA women. Schutte et al.[18] found a significant relationship between leptin levels and SBP in hypertensive obese African women when compared to normotensive lean controls. Elevated plasma leptin levels have been shown to exist in women with android obesity and positively correlated with ambulatory 24-hour systolic blood pressure[19]. In our study, correlation outcome may be because AI and AA women were more sensitive to the action of leptin in their response to sympathetic nervous system response. The possible mechanism that links leptin levels and SBP in obesity may be the action of leptin on the central nervous system activity[30].

In this study, HOMA IR (insulin resistance) was significantly correlated with indices of body fatness such as BMI, WC, and percent body fat in both groups. Insulin resistance in obesity is characterized by a strong correlation between insulin levels and degree of obesity [31]. Abdominal body fat is closely associated with insulin resistance[23].

Carey et al.[22] have shown that abdominal subcutaneous fat as well as visceral body fat is positively correlated with insulin resistance in obese women. The majority of women in this study had waist circumference greater than 88 cm with a mean WC of 107 cm. Despite differences in leptin concentrations in AI and AA women, HOMA IR and leptin positively correlated in both groups. Doehner et al.[32] observed an inverse correlation of leptin levels and insulin sensitivity in subjects with chronic heart failure. Elevated levels of leptin directly predict insulin resistance in ischemic heart disease[33]. Thus it is suggested that an interrelationship of body fatness, leptin levels and HOMA IR may result in the development of heart disease in our cohort.

There were no associations between insulin resistance and age, SBP, HDL-C and total cholesterol. However there was significant correlation between HOMA IR and serum triglycerides. In a study conducted in obese and lean Caucasian women, elevated triglycerides were negatively correlated with intramuscular triglycerides and insulin mediated glucose uptake in obese women when compared to controls[34].

In the present study, waist circumference (WC) positively correlated with SBP in both groups which are in agreement with the finding of Third National Health and Nutrition Survey (1988-1994)[35]. Han et al.[25] observed women with a WC greater than 80 cm was a predictor of cardiovascular risk factors such as high blood pressure, total cholesterol, high LDL, low HDL and high glucose values than BMI alone. Waist circumference is correlated to obesity and is a strong predictor of cardiovascular risk in obesity[35, 36]. Results of this present study are in agreement with Lofgren et al.[24] who observed a positive association of waist circumference with coronary biomarkers such as high blood pressure and serum triglycerides in overweight or obese premenopausal

women suggested that waist circumference is a more reliable tool to assess risk for coronary heart disease than BMI. However, in our cohort there was no association between waist circumference and age, HDL-C or total cholesterol. Thus, it may be suggested that waist circumference is a non-invasive, economical and practical tool for the identification of subjects with risk for cardiovascular risk.

Strengths And Limitations

This is a study of the differences between two ethnic minority groups for leptin, insulin resistance and waist circumference and the relationship of these variables to cardiovascular risk factors in these groups who face major health challenges. We only examined women of child bearing age with a similar ethnic and socio-economic background within the state of Oklahoma. In addition, this study is a convenience sample based on participant's willingness and availability to participate. It is not a representative sample of the whole American Indian and African American population but is a good representation of AI and AA women from rural Oklahoma.

Conclusions

Ninety percent of women in this cohort were overweight or obese. Leptin concentrations were significantly greater in AA women. But blood leptin concentration was correlated with SBP in both AA and AI women suggesting a phenotypic difference in the etiology of and the involvement of leptin in the development of cardiovascular disease. Mean waist circumference places this cohort at risk for heart disease. Waist circumference was correlated with SBP in both AI women and AA women. As well, mean SBP was above normal and in the pre-hypertensive classification with mean waist circumference above the cut off of 88 cm. The AI and AA women of child bearing age in

this study were already at high risk for cardiovascular disease. Interventions to reduce BMI and waist circumference in these women are imperative especially since the onset menopause further increases the risk for heart disease.

Table 1: Mean values for AI and AA women 22-45 years of age

Variable	AA Women N=33 mean±std.deviation		AI Women N=48 mean±std.deviation	
Age	34.4±5.7	(26-45)	34±5.9	(22-45)
Height	64.7±2.4	(61-71)	64.8±1.6	(61-67)
Weight	206.9±54.7	(111-346)	193±38.7	(107-300)
Body Mass Index	34.6±8.2	(18-50)	32.3±6.5	(18-52)
*MAMC	37.1±7.1	(27-59)	33.1±4.4	(24-46)
*Tricep Skin Fold	36.5±9.7	(17-54)	32.2±7.8	(13-48)
Hip	119.7±18.4	(83-150)	117.1±13.1	(92-150)
Waist	108.7±21.6	(69-150)	106.2±13.9	(77-145)
FFM	111.8±14.7	(88-145)	109.4±12.7	(83-138)
Percent body fat	43.9±8.5	(20-58)	42±6.6	(22-54)
Systolic	128.5±13.8	(100-160)	124.2±12	(97-145)
Diastolic	83.3±8.3	(60-100)	80.7±9.4	(58-100)
*Serum glucose	98.6±17.4	(60-161)	112.5±33.1	(63-262)
Serum total cholesterol	194.7±35.5	(105-272)	196.7±44.4	(70-274)
Serum HDL	52.9±16.5	(18-87)	47.9±16.1	(17-87)
*Serum triglycerides	95.5±56.6	(1-283)	145.6±91.7	(12-476)
*Leptin	35.6±17.3	(5-79)	28±12.3	(4-60)
Insulin	55.3±58.8	(4-269)	41.6±34	(5-171)
HOMA IR	4.9±1	(2-7)	5±0.79	(3-6)
Waist hip	0.8±0.08	(0.6-1.4)	0.9±0.06	(0.75-1.08)

*$p<0.05$, Significant difference between AI and AA women

Table: 2 Descriptive statistics for all women

Variables	Mean N=81	Std.deviation N=81	Min	Max	Mode
Age	34	5.79	22	45	30
BMI	32	7.3	18	52	29.75
Waist Circum(cm)	107.2	17.3	69	150	106
Total Chol(mg/dL)	196	41	70	274	175
HDL Chol(mg/dL)	50	16	17	88	45.7
SBP(mm/Hg)	126	13	97	160	120
HOMA IR	5	0.92	3	7	-
Percent body fat	43	7	20	58	40.9

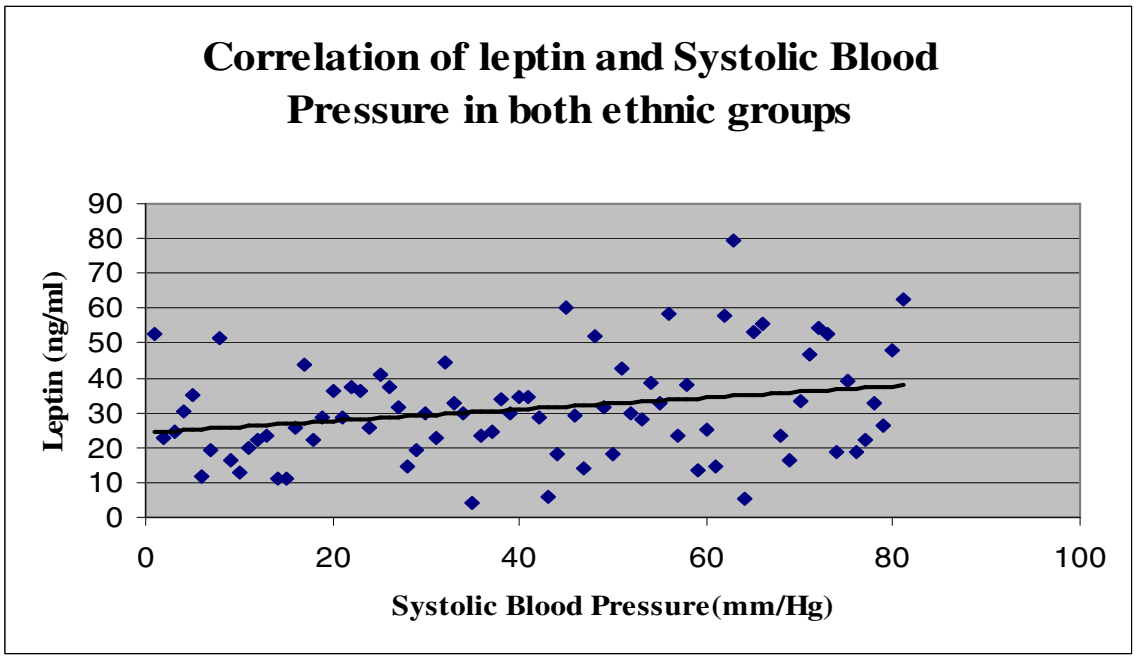
Table: 3 Correlation between leptin and cardiovascular risk factors in AI and AA women

Variables	r value	P value
Age	-0.13931	0.40490
Systolic blood pressure	0.33492	0.0043
HDL	0.01886	0.8682
Total Cholesterol	0.02460	0.8286

Table: 4 Correlation between waist circumference and insulin resistance in AI and AA women

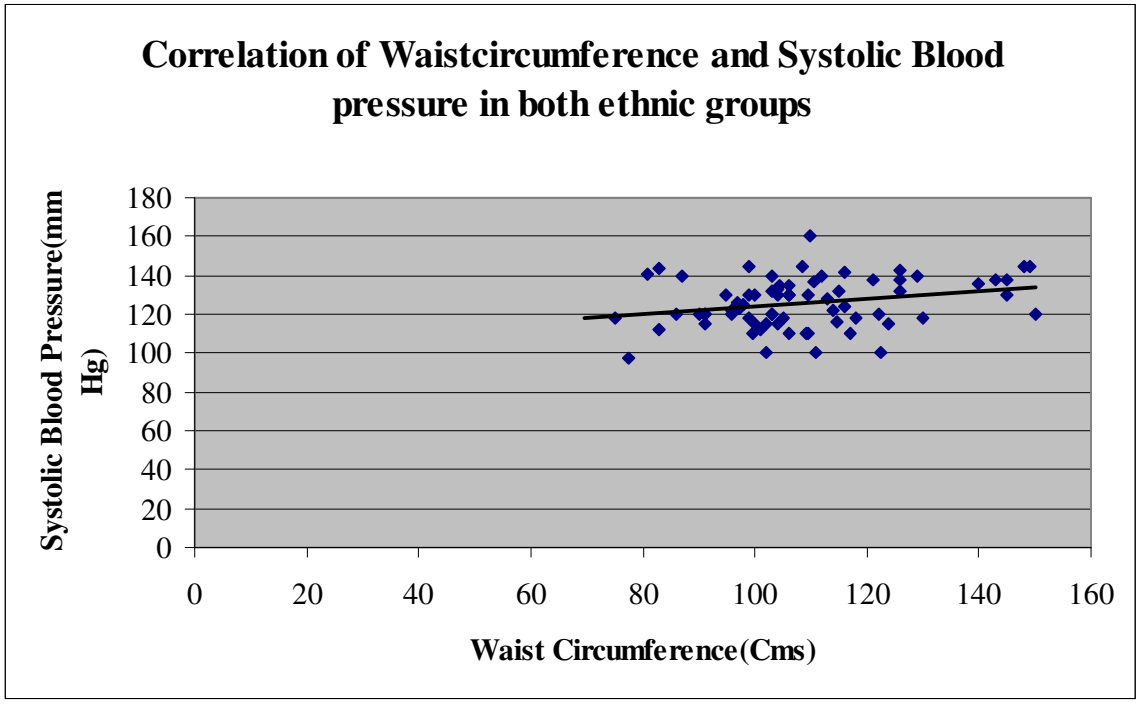
	Waist Circumference		HOMA IR	
Variables	**r value**	**p value**	**r value**	**p value**
Age	0.12741	0.2570	-0.01052	0.9267
Systolic blood pressure	0.26887	0.0224	0.12076	0.3194
HDL	-0.14742	0.1896	-0.07365	0.5189
Total cholesterol	-0.06140	0.5861	0.06008	0.5989

Figure 1: Correlation of Leptin and Systolic Pressure in both ethnic groups



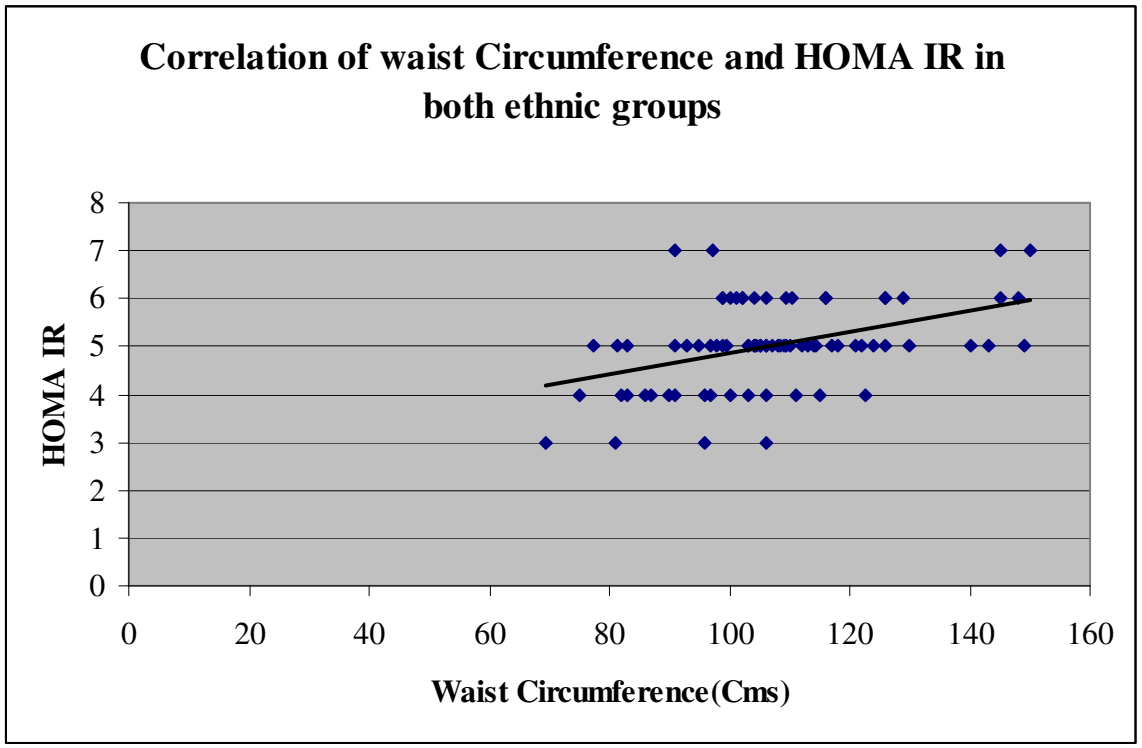
r=0.32266
p=0.0327

Figure 2: Correlation of Waist Circumference and Systolic Blood Pressure in both ethnic groups



r=0.26887
p=0.0224

Figure 3: Correlation of waist Circumference and HOMA IR in both ethnic groups



r=0.41629
p=0.0004

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CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMEDNATIONS

Summary

This study was designed to determine if ethnic differences exist between AI and AA women in leptin levels, insulin resistance and waist circumference in ethnic groups. The correlation of leptin levels, insulin resistance and waist circumference with cardiovascular risk factors such as SBP, age, HDL and total cholesterol was also determined. Finally, the correlation of waist circumference and insulin resistance was determined for this population.

Similarities were observed between the two ethnic groups for age, body mass index, waist circumference, percent body fat, HDL cholesterol, total cholesterol and SBP. Leptin levels were significantly higher in AA women when compared to AI women. There were no significant differences between the groups for insulin resistance and waist circumference. Leptin levels positively correlated with systolic blood pressure in both groups, although leptin levels were higher in AA women. Since no significant differences were observed for insulin resistance and waist circumference, both groups were combined and correlated with cardiovascular risk factors. Waist circumference positively correlated with SBP. Waist circumference increased with increased in SBP.

The relationship between waist circumference and insulin resistance was statistically significant. Insulin resistance increased with increased in waist circumference.

Conclusions

The objectives of this study to determine ethnic differences between AI and AA women and the relationship of leptin, insulin resistance and waist circumference to cardiovascular risk factors. The following hypotheses were tested with related with outcome.

Hypothesis I:

Thereis an ethnic difference between leptin levels, insulin resistance and waist circumference in American Indian and African American women.

Outcome I:

In this study, there was a significant difference ($p < .05$) between American Indian and African American women for leptin levels when compared to insulin resistance and waist circumference.

Hypothesis II:

Blood leptin concentration is positively correlated with age, systolic blood pressure and total cholesterol in American Indian and African American women.

Outcome II:

Although African American women had significantly greater serum leptin concentrations when compared to American Indian women, a significant relationship (p=0.03) between SBP and American Indian and African American women was exhibited.

Hypothesis III:

Insulin resistance (Homa-IR) is positively correlated with age, systolic blood pressure and total cholesterol in American Indian and African American women.

Outcome III:

Insulin resistance did not correlate with any of the cardiovascular risk factors in American Indian and African American women.

Hypothesis IV:

Waist circumference is positively correlated with age, systolic blood pressure and total cholesterol in American Indian and African American Women.

Outcome IV:

There was a significant relationship between waist circumference and SBP in both groups

Hypothesis V:

Waist circumference will be positively correlated with Insulin resistance (Homa-IR) in both ethnic groups.

Outcome V:

The relationship between insulin resistance and waist circumference was statistically significant for both ethnic groups. An increase in insulin resistance marked an increase in waist circumference.

It may be suggested that leptin concentrations in American Indian and African American women induce an increase in SBP due its effect on activation of sympathetic nervous system causing vasoconstriction and increasing renal tubular sodium reabsorption and inducing insulin resistance in obese subjects with increased body fat distribution through its effect on insulin. It may also be hypothesized that waist circumference is a better predictor of insulin resistance which is a major risk factor for metabolic syndrome and related diseases of the heart rather than HOMA IR. In these voluntary groups of participants greater BMI and SBP placed them at higher risk for cardiovascular disease. Lack of physical activity among the ethnic groups is evident in this cohort. This study gives an insight about the low socio-economic status strata in rural Oklahoma. It is evident that these cohorts of women are high risk for heart disease even before menopause.

Recommendations

Further studies should be conducted to determine the relationship of adipocytokines products of adipose tissue such as adinopectin, resistin, tumor necrosis factor (TNF-α) with cardiovascular risk factors in minority women who are more likely to be overweight or obese.

In this study, although leptin concentrations were higher in AA women when compared to AI women, body fat content was similar. Difference in subcutaneous and visceral fat between both groups should be studied. Further studies to determine the subcutaneous and visceral fat and its association with risk factors for heart disease in

minority women are recommended. Culturally appropriate interventions are highly recommended to decrease smoking, promote exercise and healthy eating to control body weight in AI and AA women especially Oklahoma. Waist circumference is a non invasive, economical, and practical tool for the identification of women with risk for cardiovascular risk. Further studies to investigate the relationship of leptin with cardiovascular disease in various ethnic groups are recommended.

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APPENDICES

APPENDIX A

Oklahoma State University
Institutional Review Board

Protocol Expires: 11/30/2001

Date : Friday, December 01, 2000 IRB Application No HE0116

Proposal Title: PREVALENCE OF AND FACTORS INFLUENCING CHILDHOOD OBESITY IN AFRICAN AMERICANS AND NOTIVE AMERICANS OF OKLAHOMA

Principal Investigator(s) :

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Maria Spicer
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Reviewed and Processed as: Full Board

Approval Status Recommended by Reviewer(s) : Approved

Signature :

Carol Olson, Director of University Research Compliance

Friday, December 01, 2000
Date

Approvals are valid for one calendar year, after which time a request for continuation must be submitted. Any modifications to the research project approved by the IRB must be submitted for approval with the advisor's signature. The IRB office MUST be notified in writing when a project is complete. Approved projects are subject to monitoring by the IRB. Expedited and exempt projects may be reviewed by the full Institutional Review Board.

APPENDIX B

Studies showing the normal values for insulin resistance calculation by HOMA IR[180, 184, 186, 187]

Authors (Year)	Normal Values Mean ± SD (Range)	Diabetes or IGT
Matthews (1985)	1.45	2.61
Kanauchi (2002)	1.69 ± 0.84	3.19 ± 1.43 1.89 ± 1.50
Bonora (2000)	2.06 ± 0.14 (0.7 - 6.0)	5.98 ± 0.48 (1.1-13.9)
Ascaso (2001)	≤3.8	-
Yeni-Komshan (2000)	2.7 ± 0.1 0.2-14.6	-

APPENDIX C

Obs	Participant ID	Ethnicity	Age	Height	Weight	MAMC	Tricep SF	Hip	Waist	FFM	percbod FAT	systolic
1	GP108	1	41	66.0	218.5	40.0	40.3	124.0	121.0	109.0	50.0	138
2	P108	1	26	65.5	165.5	30.5	29.6	103.0	87.0	103.0	37.9	140
3	P11	1	37	61.5	263.0	45.0	49.3	150.0	149.0	125.0	52.4	145
4	P18	1	32	63.5	217.0	38.5	42.0	117.0	109.5	117.0	46.2	110
5	P19	1	30	67.0	190.0	.	.	150.0	93.0	112.5	40.9	.
6	P193	1	30	63.5	166.5	33.5	41.3	112.0	105.0	96.5	42.1	118
7	P23	1	33	63.0	172.5	34.0	46.0	110.0	99.5	103.0	40.4	110
8	P24	1	30	63.5	227.0	38.0	38.0	130.0	114.0	112.0	50.7	122
9	P26	1	34	69.0	218.0	.	29.0	117.0	97.0	122.0	44.0	.
10	P27	1	43	62.0	195.0	30.5	42.0	124.0	122.5	104.5	46.3	100
11	P29	1	42	64.0	164.0	31.0	24.7	103.0	83.0	106.5	35.2	144
12	P31	1	44	65.5	215.5	39.0	38.3	126.0	112.0	121.0	43.9	140
13	P34	1	34	65.0	168.0	31.0	27.0	105.0	106.0	95.0	43.6	110
14	P39	1	32	63.0	253.0	37.0	43.0	135.0	104.0	122.5	51.6	133
15	P42	1	30	65.0	292.5	59.0	54.3	150.0	148.0	131.0	55.2	145
16	P43	1	44	63.0	111.0	32.0	17.5	83.5	69.5	88.5	20.3	.
17	P45	1	28	65.5	219.5	37.0	41.0	125.0	116.0	111.5	49.1	124
18	P60	1	38	66.5	284.5	45.0	46.7	142.0	143.0	133.0	53.3	138
19	P62	1	32	64.0	129.5	27.0	18.7	92.0	81.0	92.0	28.9	141
20	P63	1	45	64.5	167.5	31.0	32.0	110.0	103.0	97.5	41.7	120
21	P66	1	42	65.0	232.5	37.0	33.0	130.0	118.0	115.0	50.5	118
22	P67	1	31	67.5	183.0	30.5	33.3	104.0	100.0	107.5	41.3	130
23	P68	1	27	62.0	191.5	38.0	30.3	119.0	104.0	107.0	44.0	130

Obs	diastolic	serglucose	stotchol	ser HDL	sertriglyc	leptin	insulin	waisthip	bmi	homair
1	90	115	175	47.9	77	31.85	29.12	0.97581	35.3382	5.00284
2	84	101	122	35.3	69	17.85	7.80	0.84466	27.1767	3.55573
3	100	112	204	47.1	87	42.60	23.52	0.99333	48.9876	4.76283
4	80	108	175	63.1	71	29.61	36.43	0.93590	37.9134	5.16401
5	.	95	215	87.9	79	27.76	24.65	0.62000	29.8184	4.64514
6	84	102	179	50.3	94	38.60	24.58	0.93750	29.0903	4.71339
7	80	73	197	35.7	95	32.44	52.84	0.90455	30.6189	5.14421
8	92	84	161	45.9	178	58.56	45.37	0.87692	39.6606	5.13215
9	.	161	255	54.9	119	23.42	23.96	0.82906	32.2581	5.14427
10	60	94	235	69.7	119	37.76	17.17	0.98790	35.7382	4.27294
11	88	109	211	79.1	72	13.73	29.82	0.80583	28.2075	4.97301
12	90	60	105	18.8	187	25.17	47.27	0.88889	35.3872	4.83671
13	80	93	192	25.3	226	14.75	4.42	1.00952	28.0133	2.90522
14	82	92	248	71.6	46	58.10	43.18	0.77037	44.9077	5.17365
15	85	111	203	58.8	56	79.65	88.16	0.98667	48.7731	6.07517
16	.	79	178	60.2	1	5.29	6.86	0.83234	19.7026	3.18164
17	78	91	187	56.5	30	52.97	81.97	0.92800	36.0440	5.80370
18	92	119	194	39.6	76	55.49	30.78	1.00704	45.3231	5.09247
19	88	77	195	61.7	73	.	4.06	0.88043	22.2736	2.63147
20	90	99	244	76.1	48	23.12	9.75	0.93636	28.3646	3.75887
21	82	84	240	80.7	52	16.51	.	0.90769	38.7683	.
22	78	105	170	45.7	126	33.22	118.15	0.96154	28.2960	6.31240
23	84	94	142	41.6	149	46.73	160.89	0.87395	35.0967	6.51050

Obs	Participant ID	Ethnicity	Age	Height	Weight	MAMC	Tricep SF	Hip	Waist	FFM	percbod FAT	systolic
24	P69	1	34	62.0	264.5	43.0	42.00	146.0	129.0	122.0	53.9	140
25	P74	1	34	68.0	304.5	40.0	42.70	150.0	150.0	140.0	54.1	120
26	P76	1	33	62.5	152.5	34.0	42.30	101.0	91.0	97.0	36.4	120
27	P80	1	29	68.0	237.5	48.0	33.00	131.0	110.0	129.5	45.4	160
28	P81	1	32	61.0	141.0	29.5	27.00	104.5	82.0	93.0	33.9	.
29	P82	1	37	66.0	160.0	29.0	20.00	104.0	91.0	100.0	37.4	120
30	P83	1	33	66.0	231.0	37.0	.	122.0	118.0	126.5	45.3	.
31	P92	1	27	61.5	127.5	32.0	28.70	91.0	75.0	93.0	27.1	118
32	P94	1	44	64.0	216.0	43.0	50.00	122.5	113.0	112.0	48.2	128
33	P97	1	30	71.5	346.5	50.0	50.70	.	145.0	145.5	58.0	138
34	GP38	3	44	65.5	282.5	43.0	46.70	136.0	126.0	129.0	54.4	143
35	P1	3	31	64.5	206.0	33.0	32.00	121.0	105.0	138.5	32.7	.
36	P10	3	40	64.5	171.5	31.0	30.00	109.0	95.0	99.5	42.0	130
37	P100	3	31	66.0	217.0	35.0	35.30	126.0	115.0	118.5	45.4	132
38	P101	3	26	63.5	171.0	37.0	27.00	113.5	108.5	99.5	41.9	145
39	P103	3	33	65.0	149.0	28.0	27.30	102.0	90.0	95.0	36.4	120
40	P105	3	38	65.0	215.5	34.0	41.70	130.5	126.0	115.0	46.7	138
41	P106	3	23	64.5	236.0	37.5	33.00	136.0	116.0	115.5	50.8	142
42	P113	3	39	65.5	172.5	33.0	34.70	106.0	100.0	102.0	40.9	115

43	P114	3	30	65.0	157.0	32.0	34.00	95.0	86.0	103.0	34.3	120
44	P115	3	35	64.0	163.0	30.0	30.30	109.0	96.0	97.0	39.6	120
45	P12	3	31	64.0	235.0	35.6	48.00	134.6	110.5	135.0	41.2	137
46	P13	3	40	67.0	188.0	31.5	27.30	106.0	98.0	110.0	41.6	125

Obs	diastolic	serglucose	stotchol	ser HDL	sertriglyc	leptin	insulin	waisthip	bmi	homair
24	90	103	176	42.0	127	54.32	78.89	0.88356	48.4756	5.88927
25	80	94	178	35.6	92	52.44	174.89	1.00000	46.3928	6.59394
26	82	115	191	31.0	87	18.60	45.06	0.90099	27.5037	5.43941
27	70	95	171	39.2	88	39.30	32.10	0.83969	36.1849	4.90922
28	.	91	211	63.7	49	18.77	16.46	0.78469	26.6956	4.19828
29	70	100	272	57.6	283	22.29	269.58	0.87500	25.8770	7.08852
30	.	91	205	60.9	65	32.88	53.08	0.96721	37.3598	5.36914
31	76	85	210	69.2	61	26.02	8.91	0.82418	23.7488	3.51631
32	84	110	190	42.5	76	47.99	49.16	0.92245	37.1514	5.48205
33	95	114	197	51.5	96	62.26	130.60	.	47.7499	6.49482
34	95	106	239	56.4	132	52.57	81.02	0.92647	46.3892	5.94462
35	.	114	252	18.8	204	22.90	35.47	0.86777	34.8842	5.19137
36	78	98	208	32.9	224	24.42	28.07	0.87156	29.0419	4.80615
37	90	101	183	52.1	64	30.54	19.53	0.91270	35.0956	4.47356
38	98	103	155	40.3	144	35.24	20.22	0.95595	29.8765	4.52789
39	76	104	189	36.3	93	11.43	11.59	0.88235	24.8451	3.98102
40	88	135	216	33.4	182	19.45	19.61	0.96552	35.9337	4.76780
41	90	101	186	59.1	101	51.44	61.95	0.85294	39.9644	5.62793
42	80	95	236	56.9	65	16.43	18.41	0.94340	28.3261	4.35326
43	80	135	158	25.3	68	13.01	11.55	0.90526	26.1791	4.23844
44	78	78	185	50.2	84	20.03	24.62	0.88073	28.0355	4.44675
45	88	159	107	29.3	177	22.43	42.60	0.82095	40.4193	5.70724
46	82	123	274	67.4	12	23.56	30.21	0.92453	29.5046	5.10684

Obs	Participant ID	Ethnicity	Age	Height	Weight	MAMC	Tricep SF	Hip	Waist	FFM	percbod FAT	systolic
47	P14	3	35	62.0	149.0	.	32.00	108.0	81.3	111.0	25.4	.
48	P16	3	30	63.0	162.0	30.0	30.30	97.0	96.0	97.0	40.1	122
49	P165	3	31	62.0	166.5	32.0	27.70	108.0	102.0	100.0	39.8	100
50	P22	3	30	63.5	269.5	35.0	47.70	145.0	122.0	129.5	51.9	120
51	P28	3	40	66.0	196.5	31.8	30.00	119.4	104.3	105.5	46.3	135
52	P3	3	38	65.0	189.5	33.0	41.00	115.6	104.0	105.0	44.5	115
53	P38	3	22	63.5	205.0	38.5	41.30	109.0	106.0	112.5	45.0	130
54	P44	3	24	67.0	174.5	28.0	23.00	114.0	107.0	104.0	40.3	.
55	P48	3	38	66.5	192.0	33.5	34.00	117.2	97.2	109.5	42.9	123
56	P5	3	41	66.5	200.5	34.5	38.00	112.0	109.0	111.5	44.3	110
57	P51	3	44	67.0	264.0	39.0	30.70	129.0	140.0	134.5	49.0	136
58	P52	3	40	67.5	218.0	33.0	29.30	129.0	124.0	113.5	48.0	115
59	P55	3	39	65.5	210.0	34.5	38.50	123.0	114.5	.	.	116
60	P59	3	38	64.5	177.5	28.0	28.70	112.0	99.0	102.0	42.6	145
61	P6	3	29	64.0	145.0	26.0	16.00	100.0	91.0	95.5	34.3	115
62	P61	3	37	63.5	176.5	32.0	29.70	116.0	106.0	101.5	42.4	135
63	P65	3	31	62.0	166.5	35.0	27.75	108.0	102.0	100.0	39.8	115
64	P7	3	33	67.0	190.0	30.0	22.00	115.0	99.0	111.5	41.2	118
65	P70	3	39	66.0	226.5	39.0	44.70	131.0	126.0	115.0	49.3	132
66	P71	3	23	65.0	168.5	33.0	34.00	115.0	108.0	102.0	39.5	.
67	P73	3	28	65.5	236.5	41.0	45.30	127.0	106.0	118.0	50.1	130
68	P75	3	36	64.5	107.5	25.0	13.70	92.0	83.0	83.5	22.5	112
69	P77	3	31	64.0	221.0	33.0	40.00	135.0	130.0	110.5	49.9	118

Obs	diastolic	serglucose	stotchol	ser HDL	sertriglyc	leptin	insulin	waisthip	bmi	homair
47	.	106	196	75.6	118	11.33	25.79	0.75278	27.3076	4.79991
48	92	95	225	67.7	224	11.36	5.47	0.98969	28.7551	3.13964
49	58	91	179	54.0	152	25.51	67.90	0.94444	30.5149	5.61538
50	86	116	235	32.2	108	43.64	37.59	0.84138	47.0861	5.26681
51	80	94	181	40.1	193	21.96	28.21	0.87353	31.7801	4.76946
52	80	89	165	37.8	99	28.38	37.03	0.89965	31.5983	4.98685
53	83	98	152	46.1	15	36.22	59.27	0.97248	35.8169	5.55356
54	.	107	153	56.0	84	28.65	30.67	0.93860	27.3859	4.98260
55	72	176	267	59.5	203	37.63	89.34	0.82935	30.5871	6.54942
56	80	111	179	42.5	177	36.49	32.20	0.97321	31.9413	5.06798
57	82	99	160	39.9	105	25.96	30.18	1.08527	41.4319	4.88878
58	72	90	267	54.2	201	40.64	28.36	0.96124	33.7078	4.73127
59	70	105	230	47.7	77	37.13	27.90	0.93089	34.4840	4.86907
60	98	110	262	71.1	117	31.27	63.47	0.88393	30.0580	5.73753
61	70	71	192	53.4	94	14.57	25.51	0.91000	24.9396	4.38824
62	100	111	175	57.7	53	19.01	22.93	0.91379	30.8374	4.72846
63	72	178	212	45.7	286	29.73	79.14	0.94444	30.5149	6.43949

64	72	97	151	27.1	45	22.97	48.81	0.86087	29.8184	5.34913
65	83	123	198	71.4	146	44.45	115.69	0.96183	36.6321	6.44958
66	.	152	273	87.9	151	32.97	27.31	0.93913	28.0966	5.21762
67	94	93	202	47.9	148	29.52	15.19	0.83465	38.8356	4.13972
68	72	97	224	38.0	147	4.20	8.18	0.90217	18.2041	3.56289
69	68	262	150	35.0	154	23.09	16.69	0.96296	38.0114	5.26964

Obs	Participant ID	Ethnicity	Age	Height	Weight	MAMC	Tricep SF	Hip	Waist	FFM	percbod FAT	systolic
70	P79	3	32	62.0	185.5	31.5	31.70	121.0	111.0	102.5	44.8	100
71	P8	3	41	62.5	187.0	33.0	24.00	116.0	99.0	128.0	31.6	130
72	P84	3	45	67.5	203.0	24.5	25.00	119.5	103.0	119.0	41.4	120
73	P85	3	29	66.0	143.0	30.0	28.00	96.0	77.5	94.5	34.0	97
74	P87	3	34	67.5	181.5	30.0	24.70	115.0	103.0	107.5	40.9	140
75	P9	3	43	61.5	195.5	36.5	27.00	119.0	106.0	106.0	45.9	130
76	P90	3	30	67.5	180.0	30.0	27.70	113.0	101.0	104.0	42.3	112
77	P91	3	37	65.5	156.0	29.0	24.30	113.0	117.0	94.5	39.5	110
78	P95	3	38	63.5	300.5	46.0	43.00	150.0	145.0	136.0	54.7	130
79	P96	3	29	65.0	187.0	38.0	38.30	115.0	103.0	110.0	41.3	132
80	P98	3	29	64.5	136.0	28.0	27.00	103.0	97.0	89.0	34.4	126
81	P99	3	30	64.0	234.5	34.0	35.00	139.5	109.5	121.0	48.5	130

Obs	diastolic	serglucose	stotchol	ser HDL	sertriglyc	leptin	insulin	waisthip	bmi	homair
70	78	105	139	34.5	202	24.47	15.57	0.91736	33.9971	4.28579
71	82	108	243	35.7	476	34.15	73.19	0.85345	33.7258	5.86167
72	70	178	212	45.7	286	29.73	.	0.86192	31.3884	.
73	82	104	177	60.0	47	34.72	23.31	0.80729	23.1275	4.67976
74	84	85	176	48.6	55	34.21	26.99	0.89565	28.0640	4.62460
75	78	63	70	17.2	25	28.48	46.40	0.89076	36.4148	4.86692
76	74	103	182	40.1	237	6.02	142.21	0.89381	27.8321	6.47852
77	60	93	239	80.7	360	17.97	41.21	1.03540	25.6167	5.13777
78	84	123	160	48.5	154	60.32	171.18	0.96667	52.5023	6.84138
79	82	103	158	54.6	118	29.38	26.77	0.89565	31.1814	4.80850
80	84	98	209	69.0	80	14.23	8.89	0.94175	23.0303	3.65638
81	88	114	263	20.5	304	52.30	52.80	0.78495	40.3333	5.58919

Differences between ethnic groups

The GLM Procedure

Class Level Information

Class	Levels	Values
Ethnicity	2	1 3
Smoke	2	1 2
bp	2	1 2

Data for Analysis of Height Weight bmi Waist serglucose totchol HDL triglyc

Number of Observations Read 81
Number of Observations Used 80

Data for Analysis of MAMC

Number of Observations Read 81
Number of Observations Used 77

Data for Analysis of TricepSF

Number of Observations Read 81
Number of Observations Used 78

Data for Analysis of Wrist

Number of Observations Read 81
Number of Observations Used 77

Data for Analysis of Hip waisthip

Number of Observations Read 81
Number of Observations Used 79

Data for Analysis of FFM percbFAT

Number of Observations Read 81
Number of Observations Used 79

The GLM Procedure

Data for Analysis of diastolic systolic

Number of Observations Read 81
Number of Observations Used 72

Data for Analysis of leptin

Number of Observations Read 81
Number of Observations Used 79

Data for Analysis of insulin homair

Number of Observations Read 81
Number of Observations Used 78

NOTE: Variables in each group are consistent with respect to the presence or absence of missing values.

The GLM Procedure

Dependent Variable: Height

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	0.2684256	0.2684256	0.07	0.7960
Error	78	311.1534494	3.9891468		
Corrected Total	79	311.4218750			

R-Square	Coeff Var	Root MSE	Height Mean
0.000862	3.083122	1.997285	64.78125

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.26842561	0.26842561	0.07	0.7960

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.26842561	0.26842561	0.07	0.7960

The GLM Procedure

Dependent Variable: Weight

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	3421.5459	3421.5459	1.61	0.2083
Error	78	165796.0509	2125.5904		
Corrected Total	79	169217.5969			

R-Square	Coeff Var	Root MSE	Weight Mean
0.020220	23.16281	46.10413	199.0438

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3421.545940	3421.545940	1.61	0.2083

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3421.545940	3421.545940	1.61	0.2083

The GLM Procedure

Dependent Variable: bmi

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	90.963982	90.963982	1.70	0.1967
Error	78	4185.445240	53.659554		
Corrected Total	79	4276.409222			

R-Square	Coeff Var	Root MSE	bmi Mean
0.021271	21.95779	7.325268	33.36069

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	90.96398197	90.96398197	1.70	0.1967

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	90.96398197	90.96398197	1.70	0.1967

The GLM Procedure

Dependent Variable: Waist

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	122.84008	122.84008	0.40	0.5301
Error	78	24089.01380	308.83351		
Corrected Total	79	24211.85388			

R-Square	Coeff Var	Root MSE	Waist Mean
0.005074	16.38131	17.57366	107.2788

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	122.8400775	122.8400775	0.40	0.5301

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	122.8400775	122.8400775	0.40	0.5301

The GLM Procedure

Dependent Variable: serglucose

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	3272.91348	3272.91348	4.27	0.0421
Error	78	59807.88652	766.76778		
Corrected Total	79	63080.80000			

R-Square	Coeff Var	Root MSE	serglucose Mean
0.051884	26.04946	27.69057	106.3000

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3272.913475	3272.913475	4.27	0.0421

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3272.913475	3272.913475	4.27	0.0421

The GLM Procedure

Dependent Variable: totchol

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	2.2383	2.2383	0.00	0.9706
Error	78	127392.7492	1633.2404		
Corrected Total	79	127394.9875			

R-Square	Coeff Var	Root MSE	totchol Mean
0.000018	20.72613	40.41337	194.9875

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	2.23830593	2.23830593	0.00	0.9706

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	2.23830593	2.23830593	0.00	0.9706

The GLM Procedure

Dependent Variable: HDL

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	657.10571	657.10571	2.64	0.1086
Error	78	19450.80629	249.36931		
Corrected Total	79	20107.91200			

R-Square	Coeff Var	Root MSE	HDL Mean
0.032679	31.89544	15.79143	49.51000

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	657.1057073	657.1057073	2.64	0.1086

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	657.1057073	657.1057073	2.64	0.1086

The GLM Procedure

Dependent Variable: triglyc

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	48383.7873	48383.7873	7.56	0.0074
Error	78	498881.7627	6395.9200		
Corrected Total	79	547265.5500			

R-Square	Coeff Var	Root MSE	triglyc Mean
0.088410	64.01801	79.97450	124.9250

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	48383.78727	48383.78727	7.56	0.0074

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	48383.78727	48383.78727	7.56	0.0074

The GLM Procedure

Dependent Variable: MAMC

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	294.869292	294.869292	9.07	0.0035
Error	75	2437.137721	32.495170		
Corrected Total	76	2732.007013			

R-Square	Coeff Var	Root MSE	MAMC Mean
0.107931	16.42167	5.700453	34.71299

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	294.8692921	294.8692921	9.07	0.0035

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	294.8692921	294.8692921	9.07	0.0035

The GLM Procedure

Dependent Variable: TricepSF

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	353.585299	353.585299	4.68	0.0336
Error	76	5736.867169	75.485094		
Corrected Total	77	6090.452468			

R-Square	Coeff Var	Root MSE	TricepSF Mean
0.058056	25.59169	8.688216	33.94936

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	353.5852991	353.5852991	4.68	0.0336

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	353.5852991	353.5852991	4.68	0.0336

The GLM Procedure

Dependent Variable: Wrist

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	3.39228797	3.39228797	2.76	0.1009
Error	75	92.22946528	1.22972620		
Corrected Total	76	95.62175325			

R-Square	Coeff Var	Root MSE	Wrist Mean
0.035476	6.832923	1.108930	16.22922

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3.39228797	3.39228797	2.76	0.1009

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3.39228797	3.39228797	2.76	0.1009

The GLM Procedure

Dependent Variable: Hip

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	131.77567	131.77567	0.54	0.4634
Error	77	18685.65522	242.67085		
Corrected Total	78	18817.43089			

R-Square	Coeff Var	Root MSE	Hip Mean

0.007003	13.17574	15.57790	118.2316	

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	131.7756667	131.7756667	0.54	0.4634

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	131.7756667	131.7756667	0.54	0.4634

The GLM Procedure

Dependent Variable: waisthip

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	0.00199002	0.00199002	0.39	0.5361
Error	77	0.39660831	0.00515076		
Corrected Total	78	0.39859833			

R-Square	Coeff Var	Root MSE	waisthip Mean
0.004993	7.950381	0.071769	0.902709

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.00199002	0.00199002	0.39	0.5361

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.00199002	0.00199002	0.39	0.5361

The GLM Procedure

Dependent Variable: FFM

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	99.22325	99.22325	0.53	0.4683
Error	77	14380.22612	186.75618		
Corrected Total	78	14479.44937			

R-Square	Coeff Var	Root MSE	FFM Mean
0.006853	12.36448	13.66588	110.5253

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	99.22324719	99.22324719	0.53	0.4683

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	99.22324719	99.22324719	0.53	0.4683

The GLM Procedure

Dependent Variable: percbFAT

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	63.006800	63.006800	1.10	0.2970
Error	77	4399.601555	57.137683		
Corrected Total	78	4462.608354			

R-Square	Coeff Var	Root MSE	percbFAT Mean
0.014119	17.63606	7.558947	42.86076

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	63.00679975	63.00679975	1.10	0.2970

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	63.00679975	63.00679975	1.10	0.2970

The GLM Procedure

Dependent Variable: diastolic

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	322.91558	322.91558	1.99	0.1627
Error	70	11354.58442	162.20835		
Corrected Total	71	11677.50000			

R-Square	Coeff Var	Root MSE	diastolic Mean
0.027653	10.11471	12.73610	125.9167

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	322.9155844	322.9155844	1.99	0.1627

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	322.9155844	322.9155844	1.99	0.1627

The GLM Procedure

Dependent Variable: systolic

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	116.307540	116.307540	1.44	0.2348
Error	70	5668.678571	80.981122		
Corrected Total	71	5784.986111			

R-Square	Coeff Var	Root MSE	systolic Mean
0.020105	11.00602	8.998951	81.76389

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	116.3075397	116.3075397	1.44	0.2348

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	116.3075397	116.3075397	1.44	0.2348

The GLM Procedure

Dependent Variable: leptin

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	1125.07438	1125.07438	5.29	0.0241
Error	77	16373.99149	212.64924		
Corrected Total	78	17499.06587			

R-Square	Coeff Var	Root MSE	leptin Mean
0.064293	46.95997	14.58250	31.05304

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	1125.074383	1125.074383	5.29	0.0241

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	1125.074383	1125.074383	5.29	0.0241

The GLM Procedure

Dependent Variable: insulin

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	3370.3652	3370.3652	1.59	0.2106
Error	76	160684.1380	2114.2650		
Corrected Total	77	164054.5031			

R-Square	Coeff Var	Root MSE	insulin Mean
0.020544	96.97593	45.98114	47.41500

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3370.365163	3370.365163	1.59	0.2106

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3370.365163	3370.365163	1.59	0.2106

The GLM Procedure

Dependent Variable: homair

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	1	0.02325180	0.02325180	0.03	0.8705
Error	76	66.03676713	0.86890483		
Corrected Total	77	66.06001893			

R-Square	Coeff Var	Root MSE	homair Mean
0.000352	228.2188	0.932151	0.408446

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.02325180	0.02325180	0.03	0.8705

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.02325180	0.02325180	0.03	0.8705

The GLM Procedure

Class Level Information

Class	Levels	Values
Ethnicity	2	1 3
Smoke	2	1 2
bp	2	1 2

Data for Analysis of Height Weight bmi Waist serglucose totchol HDL triglyc

Number of Observations Read 81
Number of Observations Used 80

Data for Analysis of MAMC

Number of Observations Read 81
Number of Observations Used 77

Data for Analysis of TricepSF

Number of Observations Read 81
Number of Observations Used 78

Data for Analysis of Wrist

Number of Observations Read 81
Number of Observations Used 77

Data for Analysis of Hip waisthip

Number of Observations Read 81
Number of Observations Used 79

Data for Analysis of FFM percbFAT

Number of Observations Read 81
Number of Observations Used 79

The GLM Procedure

Data for Analysis of diastolic systolic

Number of Observations Read 81
Number of Observations Used 72

Data for Analysis of leptin

Number of Observations Read 81
Number of Observations Used 79

Data for Analysis of insulin homair

Number of Observations Read	81
Number of Observations Used	78

NOTE: Variables in each group are consistent with respect to the presence or absence of missing values.

The GLM Procedure

Dependent Variable: Height

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	3.4864130	1.1621377	0.29	0.8348
Error	76	307.9354620	4.0517824		
Corrected Total	79	311.4218750			

R-Square	Coeff Var	Root MSE	Height Mean
0.011195	3.107232	2.012904	64.78125

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.26842561	0.26842561	0.07	0.7976
bp	1	0.44402197	0.44402197	0.11	0.7415
Ethnicity*bp	1	2.77396546	2.77396546	0.68	0.4106

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.31938482	0.31938482	0.08	0.7797
bp	1	0.89848159	0.89848159	0.22	0.6391
Ethnicity*bp	1	2.77396546	2.77396546	0.68	0.4106

The GLM Procedure

Dependent Variable: Weight

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	27960.2856	9320.0952	5.01	0.0032
Error	76	141257.3113	1858.6488		
Corrected Total	79	169217.5969			

R-Square	Coeff Var	Root MSE	Weight Mean
0.165233	21.65958	43.11205	199.0438

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3421.54594	3421.54594	1.84	0.1789
bp	1	23445.12977	23445.12977	12.61	0.0007
Ethnicity*bp	1	1093.60985	1093.60985	0.59	0.4454

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3239.13031	3239.13031	1.74	0.1908
bp	1	24505.93082	24505.93082	13.18	0.0005
Ethnicity*bp	1	1093.60985	1093.60985	0.59	0.4454

The GLM Procedure

Dependent Variable: bmi

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	751.716821	250.572274	5.40	0.0020
Error	76	3524.692401	46.377532		
Corrected Total	79	4276.409222			

R-Square	Coeff Var	Root MSE	bmi Mean
0.175782	20.41356	6.810105	33.36069

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	90.9639820	90.9639820	1.96	0.1654
bp	1	641.8896467	641.8896467	13.84	0.0004
Ethnicity*bp	1	18.8631920	18.8631920	0.41	0.5256

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	86.6020038	86.6020038	1.87	0.1758
bp	1	660.7261198	660.7261198	14.25	0.0003
Ethnicity*bp	1	18.8631920	18.8631920	0.41	0.5256

The GLM Procedure

Dependent Variable: Waist

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	2256.90215	752.30072	2.60	0.0579
Error	76	21954.95172	288.88094		
Corrected Total	79	24211.85388			

R-Square	Coeff Var	Root MSE	Waist Mean
0.093215	15.84330	16.99650	107.2788

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	122.840077	122.840077	0.43	0.5163
bp	1	1968.425080	1968.425080	6.81	0.0109
Ethnicity*bp	1	165.636995	165.636995	0.57	0.4513

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	111.092995	111.092995	0.38	0.5370
bp	1	2110.084892	2110.084892	7.30	0.0085
Ethnicity*bp	1	165.636995	165.636995	0.57	0.4513

The GLM Procedure

Dependent Variable: serglucose

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	3614.07014	1204.69005	1.54	0.2111
Error	76	59466.72986	782.45697		
Corrected Total	79	63080.80000			

R-Square	Coeff Var	Root MSE	serglucose Mean
0.057293	26.31461	27.97243	106.3000

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3272.913475	3272.913475	4.18	0.0443
bp	1	106.620428	106.620428	0.14	0.7131
Ethnicity*bp	1	234.536237	234.536237	0.30	0.5856

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3311.978818	3311.978818	4.23	0.0431
bp	1	55.987076	55.987076	0.07	0.7898
Ethnicity*bp	1	234.536237	234.536237	0.30	0.5856

The GLM Procedure

Dependent Variable: totchol

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	8245.4216	2748.4739	1.75	0.1633
Error	76	119149.5659	1567.7574		
Corrected Total	79	127394.9875			

R-Square	Coeff Var	Root MSE	totchol Mean
0.064723	20.30639	39.59492	194.9875

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	2.238306	2.238306	0.00	0.9700
bp	1	3768.783259	3768.783259	2.40	0.1252
Ethnicity*bp	1	4474.400025	4474.400025	2.85	0.0952

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.308283	0.308283	0.00	0.9888
bp	1	5207.299767	5207.299767	3.32	0.0723
Ethnicity*bp	1	4474.400025	4474.400025	2.85	0.0952

The GLM Procedure

Dependent Variable: HDL

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	1448.92553	482.97518	1.97	0.1260
Error	76	18658.98647	245.51298		
Corrected Total	79	20107.91200			

R-Square	Coeff Var	Root MSE	HDL Mean
0.072057	31.64786	15.66885	49.51000

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	657.1057073	657.1057073	2.68	0.1060
bp	1	76.9577117	76.9577117	0.31	0.5772
Ethnicity*bp	1	714.8621088	714.8621088	2.91	0.0920

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	695.6103927	695.6103927	2.83	0.0964
bp	1	177.4709862	177.4709862	0.72	0.3979

Ethnicity*bp	1	714.8621088	714.8621088	2.91	0.0920

The GLM Procedure

Dependent Variable: triglyc

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	48542.1449	16180.7150	2.47	0.0686
Error	76	498723.4051	6562.1501		
Corrected Total	79	547265.5500			

R-Square	Coeff Var	Root MSE	triglyc Mean
0.088699	64.84459	81.00710	124.9250

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	48383.78727	48383.78727	7.37	0.0082
bp	1	138.18438	138.18438	0.02	0.8850
Ethnicity*bp	1	20.17325	20.17325	0.00	0.9559

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	48378.18357	48378.18357	7.37	0.0082
bp	1	116.34409	116.34409	0.02	0.8944
Ethnicity*bp	1	20.17325	20.17325	0.00	0.9559

The GLM Procedure

Dependent Variable: MAMC

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	828.748071	276.249357	10.60	<.0001
Error	73	1903.258942	26.072040		
Corrected Total	76	2732.007013			

R-Square	Coeff Var	Root MSE	MAMC Mean
0.303348	14.70942	5.106079	34.71299

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	294.8692921	294.8692921	11.31	0.0012
bp	1	508.5362492	508.5362492	19.51	<.0001
Ethnicity*bp	1	25.3425300	25.3425300	0.97	0.3274

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	260.7694740	260.7694740	10.00	0.0023
bp	1	533.6675850	533.6675850	20.47	<.0001
Ethnicity*bp	1	25.3425300	25.3425300	0.97	0.3274

The GLM Procedure

Dependent Variable: TricepSF

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	785.751821	261.917274	3.65	0.0163

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Error	74	5304.700647	71.685144		
Corrected Total	77	6090.452468			

R-Square	Coeff Var	Root MSE	TricepSF Mean
0.129014	24.93923	8.466708	33.94936

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	353.5852991	353.5852991	4.93	0.0294
bp	1	425.5124183	425.5124183	5.94	0.0172
Ethnicity*bp	1	6.6541040	6.6541040	0.09	0.7615

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	317.3667423	317.3667423	4.43	0.0388
bp	1	428.9357847	428.9357847	5.98	0.0168
Ethnicity*bp	1	6.6541040	6.6541040	0.09	0.7615

The GLM Procedure

Dependent Variable: Wrist

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	7.26330787	2.42110262	2.00	0.1214
Error	73	88.35844538	1.21038966		
Corrected Total	76	95.62175325			

R-Square	Coeff Var	Root MSE	Wrist Mean
0.075959	6.778989	1.100177	16.22922

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3.39228797	3.39228797	2.80	0.0984
bp	1	3.78561733	3.78561733	3.13	0.0812
Ethnicity*bp	1	0.08540257	0.08540257	0.07	0.7913

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	3.32418097	3.32418097	2.75	0.1018
bp	1	3.86942605	3.86942605	3.20	0.0779
Ethnicity*bp	1	0.08540257	0.08540257	0.07	0.7913

The GLM Procedure

Dependent Variable: Hip

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	1814.77037	604.92346	2.67	0.0537
Error	75	17002.66052	226.70214		
Corrected Total	78	18817.43089			

R-Square	Coeff Var	Root MSE	Hip Mean
0.096441	12.73486	15.05663	118.2316

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	131.775667	131.775667	0.58	0.4482

bp	1	1649.474409	1649.474409	7.28	0.0086
Ethnicity*bp	1	33.520295	33.520295	0.15	0.7017

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	140.484454	140.484454	0.62	0.4336
bp	1	1678.922342	1678.922342	7.41	0.0081
Ethnicity*bp	1	33.520295	33.520295	0.15	0.7017

The GLM Procedure

Dependent Variable: waisthip

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	0.00320769	0.00106923	0.20	0.8941
Error	75	0.39539064	0.00527188		
Corrected Total	78	0.39859833			

R-Square	Coeff Var	Root MSE	waisthip Mean
0.008047	8.043313	0.072608	0.902709

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.00199002	0.00199002	0.38	0.5408
bp	1	0.00089233	0.00089233	0.17	0.6819
Ethnicity*bp	1	0.00032534	0.00032534	0.06	0.8045

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.00197571	0.00197571	0.37	0.5423
bp	1	0.00107260	0.00107260	0.20	0.6532
Ethnicity*bp	1	0.00032534	0.00032534	0.06	0.8045

The GLM Procedure

Dependent Variable: FFM

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	1683.11615	561.03872	3.29	0.0252
Error	75	12796.33322	170.61778		
Corrected Total	78	14479.44937			

R-Square	Coeff Var	Root MSE	FFM Mean
0.116242	11.81817	13.06207	110.5253

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	99.223247	99.223247	0.58	0.4481
bp	1	1530.826534	1530.826534	8.97	0.0037
Ethnicity*bp	1	53.066364	53.066364	0.31	0.5787

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	98.960271	98.960271	0.58	0.4487
bp	1	1583.306075	1583.306075	9.28	0.0032
Ethnicity*bp	1	53.066364	53.066364	0.31	0.5787

The GLM Procedure

Dependent Variable: percbFAT

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	694.777976	231.592659	4.61	0.0051
Error	75	3767.830379	50.237738		
Corrected Total	78	4462.608354			

R-Square	Coeff Var	Root MSE	percbFAT Mean
0.155689	16.53694	7.087859	42.86076

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	63.0067998	63.0067998	1.25	0.2663
bp	1	624.0785616	624.0785616	12.42	0.0007
Ethnicity*bp	1	7.6926143	7.6926143	0.15	0.6967

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	63.9270878	63.9270878	1.27	0.2629
bp	1	629.9131487	629.9131487	12.54	0.0007
Ethnicity*bp	1	7.6926143	7.6926143	0.15	0.6967

Dependent Variable: diastolic

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	8003.17326	2667.72442	49.37	<.0001
Error	68	3674.32674	54.03422		
Corrected Total	71	11677.50000			

R-Square	Coeff Var	Root MSE	diastolic Mean
0.685350	5.837827	7.350797	125.9167

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	322.915584	322.915584	5.98	0.0171
bp	1	7651.108137	7651.108137	141.60	<.0001
Ethnicity*bp	1	29.149540	29.149540	0.54	0.4652

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	133.217475	133.217475	2.47	0.1210
bp	1	7430.630803	7430.630803	137.52	<.0001
Ethnicity*bp	1	29.149540	29.149540	0.54	0.4652

Dependent Variable: systolic

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	2256.265211	752.088404	14.49	<.0001
Error	68	3528.720900	51.892954		
Corrected Total	71	5784.986111			

R-Square	Coeff Var	Root MSE	systolic Mean
0.390021	8.810340	7.203676	81.76389

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	116.307540	116.307540	2.24	0.1390
bp	1	2051.032227	2051.032227	39.52	<.0001
Ethnicity*bp	1	88.925445	88.925445	1.71	0.1949

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	89.868771	89.868771	1.73	0.1926
bp	1	1738.084758	1738.084758	33.49	<.0001
Ethnicity*bp	1	88.925445	88.925445	1.71	0.1949

The GLM Procedure

Dependent Variable: leptin

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	4772.74901	1590.91634	9.38	<.0001
Error	75	12726.31686	169.68422		
Corrected Total	78	17499.06587			

R-Square	Coeff Var	Root MSE	leptin Mean
0.272743	41.94852	13.02629	31.05304

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	1125.074383	1125.074383	6.63	0.0120
bp	1	3148.350094	3148.350094	18.55	<.0001
Ethnicity*bp	1	499.324529	499.324529	2.94	0.0904

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	1151.414624	1151.414624	6.79	0.0111
bp	1	3520.020025	3520.020025	20.74	<.0001
Ethnicity*bp	1	499.324529	499.324529	2.94	0.0904

The GLM Procedure

Dependent Variable: insulin

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	5370.8306	1790.2769	0.83	0.4790
Error	74	158683.6726	2144.3740		
Corrected Total	77	164054.5031			

R-Square	Coeff Var	Root MSE	insulin Mean
0.032738	97.66400	46.30739	47.41500

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	3370.365163	3370.365163	1.57	0.2139
bp	1	1927.464451	1927.464451	0.90	0.3462
Ethnicity*bp	1	73.000975	73.000975	0.03	0.8541

Source	DF	Type III SS	Mean Square	F Value	Pr > F

Ethnicity	1	3366.561187	3366.561187	1.57	0.2142
bp	1	1733.903346	1733.903346	0.81	0.3715
Ethnicity*bp	1	73.000975	73.000975	0.03	0.8541

The GLM Procedure

Dependent Variable: homair

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	2.33798494	0.77932831	0.91	0.4429
Error	74	63.72203399	0.86110857		
Corrected Total	77	66.06001893			

R-Square	Coeff Var	Root MSE	homair Mean
0.035392	227.1927	0.927959	0.408446

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.02325180	0.02325180	0.03	0.8699
bp	1	2.01050390	2.01050390	2.33	0.1308
Ethnicity*bp	1	0.30422924	0.30422924	0.35	0.5541

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Ethnicity	1	0.03829401	0.03829401	0.04	0.8336
bp	1	2.23257755	2.23257755	2.59	0.1116
Ethnicity*bp	1	0.30422924	0.30422924	0.35	0.5541

The MEANS Procedure

Variable	N	Mean	Std Dev	Std Error	Minimum	Maximum
Ethnicity	81	2.1851852	0.9888265	0.1098696	1.0000000	3.0000000
Age	81	34.2345679	5.7971364	0.6441263	22.0000000	45.0000000
Height	81	64.7839506	1.9731610	0.2192401	61.0000000	71.5000000
Weight	81	198.6666667	46.1165643	5.1240627	107.5000000	346.5000000
MAMC	78	34.6910256	5.9597173	0.6748051	24.5000000	59.0000000
TricepSF	79	33.9500000	8.8364448	0.9941777	13.7000000	54.3000000
Hip	80	118.1912500	15.4378066	1.7259992	83.5000000	150.0000000
Waist	81	107.2876543	17.3969709	1.9329968	69.5000000	150.0000000
FFM	80	110.4187500	13.5717605	1.5173689	83.5000000	145.5000000
percbodFAT	80	42.8187500	7.5252839	0.8413523	20.3000000	58.0000000
systolic	72	125.9166667	12.8246571	1.5114003	97.0000000	160.0000000
diastolic	72	81.7638889	9.0265540	1.0637896	58.0000000	100.0000000
serglucose	81	106.8641975	28.5358341	3.1706482	60.0000000	262.0000000
stotchol	81	195.9506173	40.8359221	4.5373247	70.0000000	274.0000000
serHDL	81	49.9839506	16.4177911	1.8241990	17.2000000	87.9000000
sertriglyc	81	125.2469136	82.7599738	9.1955526	1.0000000	476.0000000
leptin	80	31.0770000	14.8846598	1.6641556	4.2000000	79.6500000
insulin	79	47.1605063	45.9170933	5.1660766	4.0600000	269.5800000
waisthip	80	0.9031639	0.0711486	0.0079547	0.6200000	1.0852713
bmi	81	33.2956970	7.3346587	0.8149621	18.2041344	52.5022630
homair	79	5.0161985	0.9205705	0.1035723	2.6314731	7.0885201

The TTEST Procedure

Statistics

Variable	Ethnicity	N	Lower CL Mean	Mean	Upper CL Mean	Lower CL Std Dev	Std Dev	Upper CL Std Dev	Std Err
Age	1	33	32.467	34.485	36.503	4.5763	5.6906	7.5269	0.9906
Age	3	48	32.343	34.063	35.782	4.9307	5.9229	7.4189	0.8549
Age	Diff (1-2)		-2.202	0.4223	3.0464	5.0455	5.8299	6.9054	1.3183
Height	1	33	63.858	64.712	65.566	1.9363	2.4078	3.1847	0.4191
Height	3	48	64.359	64.833	65.308	1.3612	1.6352	2.0482	0.236

Height	Diff (1-2)			-1.015	-0.121	0.7721	1.7177	1.9847	2.3508	0.4488
Weight		1	33	187.44	206.85	226.26	44.017	54.735	72.398	9.5282
Weight		3	48	181.79	193.04	204.29	32.251	38.741	48.526	5.5918
Weight	Diff (1-2)			-6.851	13.807	34.465	39.721	45.896	54.363	10.379
bmi		1	33	31.705	34.633	37.561	6.6408	8.2577	10.922	1.4375
bmi		3	48	30.472	32.376	34.281	5.4603	6.5592	8.2159	0.9467
bmi	Diff (1-2)			-1.026	2.2571	5.5407	6.3135	7.295	8.6407	1.6496
MAMC		1	31	34.473	37.097	39.721	5.7165	7.1536	9.562	1.2848
MAMC		3	47	31.804	33.104	34.404	3.6796	4.428	5.5615	0.6459
MAMC	Diff (1-2)			1.3829	3.9925	6.6021	4.8881	5.6628	6.7317	1.3102
TricepSF		1	31	32.993	36.571	40.149	7.7956	9.7553	13.04	1.7521
TricepSF		3	48	29.982	32.257	34.532	6.5222	7.8347	9.8136	1.1308
TricepSF	Diff (1-2)			0.3523	4.3137	8.2751	7.4594	8.6339	10.251	1.9894
Hip		1	32	113.14	119.8	126.45	14.8	18.46	24.543	3.2634
Hip		3	48	113.3	117.12	120.94	10.946	13.149	16.47	1.8979
Hip	Diff (1-2)			-4.357	2.676	9.7092	13.385	15.48	18.357	3.5327
Waist		1	33	101.09	108.76	116.42	17.388	21.622	28.599	3.7639
Waist		3	48	102.23	106.28	110.32	11.604	13.939	17.46	2.0119
Waist	Diff (1-2)			-5.38	2.4805	10.341	15.114	17.463	20.685	3.949
FFM		1	33	106.64	111.85	117.06	11.812	14.689	19.429	2.557
FFM		3	47	105.66	109.41	113.17	10.633	12.796	16.072	1.8665
FFM	Diff (1-2)			-3.718	2.4336	8.5848	11.764	13.604	16.133	3.0897
percbodFAT		1	33	40.877	43.915	46.953	6.8898	8.5674	11.332	1.4914
percbodFAT		3	47	40.085	42.049	44.013	5.5574	6.6878	8.3998	0.9755
percbodFAT	Diff (1-2)			-1.532	1.8662	5.2645	6.4992	7.516	8.913	1.707
systolic		1	28	123.22	128.57	133.93	10.916	13.807	18.793	2.6092
systolic		3	44	120.57	124.23	127.88	9.9273	12.015	15.224	1.8114
systolic	Diff (1-2)			-1.797	4.3442	10.485	10.931	12.736	15.26	3.0789
diastolic		1	28	80.147	83.357	86.567	6.5452	8.2786	11.268	1.5645
diastolic		3	44	77.885	80.75	83.615	7.7857	9.4232	11.939	1.4206
diastolic	Diff (1-2)			-1.732	2.6071	6.946	7.7237	8.999	10.783	2.1755
serglucose		1	33	92.471	98.667	104.86	14.051	17.473	23.111	3.0416
serglucose		3	48	102.88	112.5	122.12	27.592	33.144	41.516	4.784
serglucose	Diff (1-2)			-26.38	-13.83	-1.285	24.128	27.879	33.022	6.3043
stotchol		1	33	182.18	194.79	207.4	28.595	35.557	47.031	6.1897
stotchol		3	48	183.84	196.75	209.66	37.005	44.452	55.68	6.4162
stotchol	Diff (1-2)			-20.45	-1.962	16.529	35.555	41.082	48.66	9.29
serHDL		1	33	47.048	52.93	58.812	13.34	16.589	21.942	2.8877
serHDL		3	48	43.266	47.958	52.651	13.453	16.16	20.242	2.3325
serHDL	Diff (1-2)			-2.381	4.972	12.325	14.137	16.335	19.349	3.6939
sertriglyc		1	33	75.472	95.576	115.68	45.596	56.698	74.994	9.8698
sertriglyc		3	48	118.99	145.65	172.3	76.417	91.796	114.98	13.25

The TTEST Procedure

Statistics

Variable	Ethnicity	N	Lower CL Mean	Mean	Upper CL Mean	Lower CL Std Dev	Std Dev	Upper CL Std Dev	Std Err
sertriglyc	Diff (1-2)		-85.84	-50.07	-14.3	68.777	79.469	94.129	17.971
leptin		1	29.392	35.627	41.861	13.863	17.292	22.989	3.0568
leptin		3	24.468	28.044	31.62	10.253	12.316	15.427	1.7777
leptin	Diff (1-2)		0.9947	7.5826	14.17	12.538	14.5	17.195	3.3091
insulin		1	34.064	55.296	76.528	47.213	58.89	78.293	10.41
insulin		3	31.619	41.621	51.624	28.308	34.066	42.787	4.9691
insulin	Diff (1-2)		-7.186	13.675	34.536	39.493	45.711	54.273	10.476
homair		1	4.5989	4.9929	5.3869	0.8761	1.0928	1.4529	0.1932
homair		3	4.7987	5.0321	5.2654	0.6604	0.7947	0.9981	0.1159
homair	Diff (1-2)		-0.462	-0.039	0.3836	0.8003	0.9263	1.0998	0.2123
waisthip		1	0.8664	0.8966	0.9269	0.0673	0.084	0.1116	0.0148
waisthip		3	0.8896	0.9075	0.9254	0.0514	0.0617	0.0773	0.0089
waisthip	Diff (1-2)		-0.043	-0.011	0.0215	0.0617	0.0714	0.0847	0.0163

T-Tests

Variable	Method	Variances	DF	t Value	Pr > |t|
Age	Pooled	Equal	79	0.32	0.7495
Age	Satterthwaite	Unequal	70.7	0.32	0.7478
Height	Pooled	Equal	79	-0.27	0.7878
Height	Satterthwaite	Unequal	52	-0.25	0.8020
Weight	Pooled	Equal	79	1.33	0.1872
Weight	Satterthwaite	Unequal	53.5	1.25	0.2168
bmi	Pooled	Equal	79	1.37	0.1751
bmi	Satterthwaite	Unequal	58.3	1.31	0.1949
MAMC	Pooled	Equal	76	3.05	0.0032
MAMC	Satterthwaite	Unequal	45.2	2.78	0.0080

TricepSF	Pooled	Equal	77	2.17	0.0332
TricepSF	Satterthwaite	Unequal	54.2	2.07	0.0434
Hip	Pooled	Equal	78	0.76	0.4510
Hip	Satterthwaite	Unequal	51.6	0.71	0.4816
Waist	Pooled	Equal	79	0.63	0.5317
Waist	Satterthwaite	Unequal	50.1	0.58	0.5637
FFM	Pooled	Equal	78	0.79	0.4333
FFM	Satterthwaite	Unequal	62.8	0.77	0.4449
percbodFAT	Pooled	Equal	78	1.09	0.2776
percbodFAT	Satterthwaite	Unequal	57.9	1.05	0.2994
systolic	Pooled	Equal	70	1.41	0.1627
systolic	Satterthwaite	Unequal	51.7	1.37	0.1773
diastolic	Pooled	Equal	70	1.20	0.2348
diastolic	Satterthwaite	Unequal	63	1.23	0.2219
serglucose	Pooled	Equal	79	-2.19	0.0312
serglucose	Satterthwaite	Unequal	74.7	-2.44	0.0171
stotchol	Pooled	Equal	79	-0.21	0.8333
stotchol	Satterthwaite	Unequal	77.1	-0.22	0.8264

The TTEST Procedure

T-Tests

Variable	Method	Variances	DF	t Value	Pr > |t|
serHDL	Pooled	Equal	79	1.35	0.1822
serHDL	Satterthwaite	Unequal	67.7	1.34	0.1849
sertriglyc	Pooled	Equal	79	-2.79	0.0067
sertriglyc	Satterthwaite	Unequal	78.2	-3.03	0.0033
leptin	Pooled	Equal	78	2.29	0.0246
leptin	Satterthwaite	Unequal	51.6	2.14	0.0367
insulin	Pooled	Equal	77	1.31	0.1957
insulin	Satterthwaite	Unequal	45.2	1.19	0.2420
homair	Pooled	Equal	77	-0.18	0.8542
homair	Satterthwaite	Unequal	52.7	-0.17	0.8628
waisthip	Pooled	Equal	78	-0.67	0.5056
waisthip	Satterthwaite	Unequal	52.8	-0.63	0.5318

Equality of Variances

Variable	Method	Num DF	Den DF	F Value	Pr > F
Age	Folded F	47	32	1.08	0.8227
Height	Folded F	32	47	2.17	0.0154
Weight	Folded F	32	47	2.00	0.0304
bmi	Folded F	32	47	1.58	0.1479
MAMC	Folded F	30	46	2.61	0.0033
TricepSF	Folded F	30	47	1.55	0.1742
Hip	Folded F	31	47	1.97	0.0347
Waist	Folded F	32	47	2.41	0.0061
FFM	Folded F	32	46	1.32	0.3859
percbodFAT	Folded F	32	46	1.64	0.1220
systolic	Folded F	27	43	1.32	0.4074
diastolic	Folded F	43	27	1.30	0.4804
serglucose	Folded F	47	32	3.60	0.0003
stotchol	Folded F	47	32	1.56	0.1854
serHDL	Folded F	32	47	1.05	0.8562
sertriglyc	Folded F	47	32	2.62	0.0051
leptin	Folded F	31	47	1.97	0.0347
insulin	Folded F	31	46	2.99	0.0008
homair	Folded F	31	46	1.89	0.0487
waisthip	Folded F	31	47	1.85	0.0555

---------- Ethnicity=1 ----------

The CORR Procedure

20 Variables: Age Height Weight bmi MAMC TricepSF Hip Waist FFM percbodFAT systolic diastolic serglucose stotchol serHDL sertriglyc leptin insulin homair waisthip

Simple Statistics

Variable	N	Mean	Std Dev	Sum	Minimum	Maximum

Age	33	34.48485	5.69057	1138	26.00000	45.00000
Height	33	64.71212	2.40777	2136	61.00000	71.50000
Weight	33	206.84848	54.73508	6826	111.00000	346.50000
bmi	33	34.63326	8.25771	1143	19.70257	48.98764
MAMC	31	37.09677	7.15358	1150	27.00000	59.00000
TricepSF	31	36.57097	9.75528	1134	17.50000	54.30000
Hip	32	119.79688	18.46045	3834	83.50000	150.00000
Waist	33	108.75758	21.62179	3589	69.50000	150.00000
FFM	33	111.84848	14.68869	3691	88.50000	145.50000
percbodFAT	33	43.91515	8.56742	1449	20.30000	58.00000
systolic	28	128.57143	13.80668	3600	100.00000	160.00000
diastolic	28	83.35714	8.27855	2334	60.00000	100.00000
serglucose	33	98.66667	17.47260	3256	60.00000	161.00000
stotchol	33	194.78788	35.55696	6428	105.00000	272.00000
serHDL	33	52.93030	16.58867	1747	18.80000	87.90000
sertriglyc	33	95.57576	56.69768	3154	1.00000	283.00000
leptin	32	35.62656	17.29197	1140	5.29000	79.65000
insulin	32	55.29625	58.89027	1769	4.06000	269.58000
homair	32	4.99292	1.09282	159.77330	2.63147	7.08852
waisthip	32	0.89663	0.08396	28.69203	0.62000	1.00952

---------- Ethnicity=1 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Age	Height	Weight	bmi	MAMC	TricepSF	Hip
Age	1.00000	-0.09327	-0.07184	-0.03980	-0.11129	-0.05245	-0.02960
		0.6057	0.6911	0.8259	0.5511	0.7793	0.8723
	33	33	33	33	31	31	32
Height	-0.09327	1.00000	0.48427	0.19187	0.34751	0.08950	0.23086
	0.6057		0.0043	0.2848	0.0554	0.6321	0.2036
	33	33	33	33	31	31	32
Weight	-0.07184	0.48427	1.00000	0.94824	0.82814	0.73537	0.90862
	0.6911	0.0043		<.0001	<.0001	<.0001	<.0001
	33	33	33	33	31	31	32
bmi	-0.03980	0.19187	0.94824	1.00000	0.81391	0.79026	0.89852
	0.8259	0.2848	<.0001		<.0001	<.0001	<.0001
	33	33	33	33	31	31	32
MAMC	-0.11129	0.34751	0.82814	0.81391	1.00000	0.72844	0.79604
	0.5511	0.0554	<.0001	<.0001		<.0001	<.0001
	31	31	31	31	31	30	30
TricepSF	-0.05245	0.08950	0.73537	0.79026	0.72844	1.00000	0.74395
	0.7793	0.6321	<.0001	<.0001	<.0001		<.0001
	31	31	31	31	30	31	30
Hip	-0.02960	0.23086	0.90862	0.89852	0.79604	0.74395	1.00000
	0.8723	0.2036	<.0001	<.0001	<.0001	<.0001	
	32	32	32	32	30	30	32
Waist	0.05356	0.30520	0.90930	0.91361	0.77253	0.78921	0.84493
	0.7672	0.0841	<.0001	<.0001	<.0001	<.0001	<.0001
	33	33	33	33	31	31	32
FFM	-0.09275	0.57240	0.95608	0.87122	0.80821	0.64481	0.84530
	0.6077	0.0005	<.0001	<.0001	<.0001	<.0001	<.0001
	33	33	33	33	31	31	32
percbodFAT	-0.03996	0.34562	0.91825	0.92166	0.71875	0.77942	0.87064
	0.8253	0.0488	<.0001	<.0001	<.0001	<.0001	<.0001
	33	33	33	33	31	31	32
systolic	-0.11159	0.30797	0.34062	0.29205	0.50670	0.03598	0.29989
	0.5718	0.1109	0.0761	0.1316	0.0059	0.8558	0.1286
	28	28	28	28	28	28	27

---------- Ethnicity=1 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Age	Height	Weight	bmi	MAMC	TricepSF	Hip
diastolic	0.09547	0.05000	0.34266	0.36337	0.34114	0.26995	0.27236
	0.6289	0.8005	0.0743	0.0573	0.0756	0.1648	0.1693
	28	28	28	28	28	28	27
serglucose	-0.04044	0.34454	0.30543	0.23648	0.38411	0.22508	0.17197
	0.8232	0.0496	0.0839	0.1852	0.0329	0.2235	0.3466
	33	33	33	33	31	31	32
stotchol	0.22416	0.01350	-0.03118	-0.03563	-0.16101	-0.10857	-0.00110
	0.2098	0.9406	0.8632	0.8439	0.3869	0.5610	0.9952
	33	33	33	33	31	31	32
serHDL	0.12024	-0.11095	-0.16635	-0.14959	-0.21502	-0.24718	-0.03595
	0.5051	0.5388	0.3548	0.4060	0.2454	0.1801	0.8451
	33	33	33	33	31	31	32
sertriglyc	-0.00990	0.12429	-0.01255	-0.03308	-0.13303	-0.16229	-0.00439
	0.9564	0.4907	0.9447	0.8550	0.4756	0.3831	0.9810
	33	33	33	33	31	31	32
leptin	-0.32733	0.15270	0.77995	0.81641	0.75711	0.75984	0.72116
	0.0674	0.4041	<.0001	<.0001	<.0001	<.0001	<.0001
	32	32	32	32	30	30	31
insulin	-0.15265	0.31044	0.31515	0.24279	0.15836	0.05477	0.17457
	0.4042	0.0838	0.0789	0.1806	0.4033	0.7738	0.3476
	32	32	32	32	30	30	31
homair	-0.15701	0.33961	0.58533	0.54246	0.46242	0.45353	0.47185
	0.3908	0.0572	0.0004	0.0013	0.0101	0.0118	0.0074
	32	32	32	32	30	30	31
waisthip	0.22637	0.05079	0.37361	0.37273	0.32481	0.51888	0.14120
	0.2128	0.7825	0.0352	0.0356	0.0799	0.0033	0.4408
	32	32	32	32	30	30	32

---------- Ethnicity=1 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Waist	FFM	percbod FAT	systolic	diastolic	serglucose	stotchol
Age	0.05356	-0.09275	-0.03996	-0.11159	0.09547	-0.04044	0.22416
	0.7672	0.6077	0.8253	0.5718	0.6289	0.8232	0.2098
	33	33	33	28	28	33	33
Height	0.30520	0.57240	0.34562	0.30797	0.05000	0.34454	0.01350
	0.0841	0.0005	0.0488	0.1109	0.8005	0.0496	0.9406
	33	33	33	28	28	33	33
Weight	0.90930	0.95608	0.91825	0.34062	0.34266	0.30543	-0.03118
	<.0001	<.0001	<.0001	0.0761	0.0743	0.0839	0.8632
	33	33	33	28	28	33	33
bmi	0.91361	0.87122	0.92166	0.29205	0.36337	0.23648	-0.03563
	<.0001	<.0001	<.0001	0.1316	0.0573	0.1852	0.8439
	33	33	33	28	28	33	33
MAMC	0.77253	0.80821	0.71875	0.50670	0.34114	0.38411	-0.16101
	<.0001	<.0001	<.0001	0.0059	0.0756	0.0329	0.3869
	31	31	31	28	28	31	31
TricepSF	0.78921	0.64481	0.77942	0.03598	0.26995	0.22508	-0.10857

	<.0001	<.0001	<.0001	0.8558	0.1648	0.2235	0.5610
	31	31	31	28	28	31	31
Hip	0.84493	0.84530	0.87064	0.29989	0.27236	0.17197	-0.00110
	<.0001	<.0001	<.0001	0.1286	0.1693	0.3466	0.9952
	32	32	32	27	27	32	32
Waist	1.00000	0.81041	0.90817	0.17929	0.31965	0.23668	-0.06395
		<.0001	<.0001	0.3613	0.0973	0.1848	0.7237
	33	33	33	28	28	33	33
FFM	0.81041	1.00000	0.81045	0.44967	0.28961	0.30554	-0.06032
	<.0001		<.0001	0.0164	0.1350	0.0838	0.7388
	33	33	33	28	28	33	33
percbodFAT	0.90817	0.81045	1.00000	0.16328	0.30251	0.30392	-0.01259
	<.0001	<.0001		0.4064	0.1177	0.0855	0.9446
	33	33	33	28	28	33	33
systolic	0.17929	0.44967	0.16328	1.00000	0.45860	0.21177	-0.31220
	0.3613	0.0164	0.4064		0.0141	0.2793	0.1058
	28	28	28	28	28	28	28

---------- Ethnicity=1 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Waist	FFM	percbod FAT	systolic	diastolic	serglucose	stotchol
diastolic	0.31965	0.28961	0.30251	0.45860	1.00000	0.20723	-0.27689
	0.0973	0.1350	0.1177	0.0141		0.2900	0.1537
	28	28	28	28	28	28	28
serglucose	0.23668	0.30554	0.30392	0.21177	0.20723	1.00000	0.31340
	0.1848	0.0838	0.0855	0.2793	0.2900		0.0757
	33	33	33	28	28	33	33
stotchol	-0.06395	-0.06032	-0.01259	-0.31220	-0.27689	0.31340	1.00000
	0.7237	0.7388	0.9446	0.1058	0.1537	0.0757	
	33	33	33	28	28	33	33
serHDL	-0.25341	-0.15610	-0.19238	-0.14843	-0.15145	0.02622	0.65316
	0.1547	0.3857	0.2835	0.4510	0.4417	0.8848	<.0001
	33	33	33	28	28	33	33
sertriglyc	0.04784	-0.03272	0.11287	-0.19319	-0.17169	-0.03194	-0.04765
	0.7915	0.8565	0.5317	0.3246	0.3823	0.8599	0.7923
	33	33	33	28	28	33	33
leptin	0.74213	0.67253	0.77223	0.29269	0.23077	0.13328	-0.08609
	<.0001	<.0001	<.0001	0.1385	0.2468	0.4671	0.6394
	32	32	32	27	27	32	32
insulin	0.27219	0.30263	0.27909	-0.04163	-0.18658	0.03614	0.08829
	0.1318	0.0923	0.1219	0.8367	0.3514	0.8443	0.6309
	32	32	32	27	27	32	32
homair	0.49609	0.57875	0.57969	0.08624	-0.03101	0.29365	0.04024
	0.0039	0.0005	0.0005	0.6689	0.8780	0.1028	0.8269
	32	32	32	27	27	32	32
waisthip	0.64313	0.23388	0.44275	-0.26418	0.07979	0.11834	-0.12812
	<.0001	0.1976	0.0112	0.1830	0.6924	0.5189	0.4847
	32	32	32	27	27	32	32

---------- Ethnicity=1 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0

Number of Observations

	serHDL	sertriglyc	leptin	insulin	homair	waisthip
Age	0.12024	-0.00990	-0.32733	-0.15265	-0.15701	0.22637
	0.5051	0.9564	0.0674	0.4042	0.3908	0.2128
	33	33	32	32	32	32
Height	-0.11095	0.12429	0.15270	0.31044	0.33961	0.05079
	0.5388	0.4907	0.4041	0.0838	0.0572	0.7825
	33	33	32	32	32	32
Weight	-0.16635	-0.01255	0.77995	0.31515	0.58533	0.37361
	0.3548	0.9447	<.0001	0.0789	0.0004	0.0352
	33	33	32	32	32	32
bmi	-0.14959	-0.03308	0.81641	0.24279	0.54246	0.37273
	0.4060	0.8550	<.0001	0.1806	0.0013	0.0356
	33	33	32	32	32	32
MAMC	-0.21502	-0.13303	0.75711	0.15836	0.46242	0.32481
	0.2454	0.4756	<.0001	0.4033	0.0101	0.0799
	31	31	30	30	30	30
TricepSF	-0.24718	-0.16229	0.75984	0.05477	0.45353	0.51888
	0.1801	0.3831	<.0001	0.7738	0.0118	0.0033
	31	31	30	30	30	30
Hip	-0.03595	-0.00439	0.72116	0.17457	0.47185	0.14120
	0.8451	0.9810	<.0001	0.3476	0.0074	0.4408
	32	32	31	31	31	32
Waist	-0.25341	0.04784	0.74213	0.27219	0.49609	0.64313
	0.1547	0.7915	<.0001	0.1318	0.0039	<.0001
	33	33	32	32	32	32
FFM	-0.15610	-0.03272	0.67253	0.30263	0.57875	0.23388
	0.3857	0.8565	<.0001	0.0923	0.0005	0.1976
	33	33	32	32	32	32
percbodFAT	-0.19238	0.11287	0.77223	0.27909	0.57969	0.44275
	0.2835	0.5317	<.0001	0.1219	0.0005	0.0112
	33	33	32	32	32	32
systolic	-0.14843	-0.19319	0.29269	-0.04163	0.08624	-0.26418
	0.4510	0.3246	0.1385	0.8367	0.6689	0.1830
	28	28	27	27	27	27

---------- Ethnicity=1 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	serHDL	sertriglyc	leptin	insulin	homair	waisthip
diastolic	-0.15145	-0.17169	0.23077	-0.18658	-0.03101	0.07979
	0.4417	0.3823	0.2468	0.3514	0.8780	0.6924
	28	28	27	27	27	27
serglucose	0.02622	-0.03194	0.13328	0.03614	0.29365	0.11834
	0.8848	0.8599	0.4671	0.8443	0.1028	0.5189
	33	33	32	32	32	32
stotchol	0.65316	-0.04765	-0.08609	0.08829	0.04024	-0.12812
	<.0001	0.7923	0.6394	0.6309	0.8269	0.4847
	33	33	32	32	32	32
serHDL	1.00000	-0.43385	-0.13593	-0.16167	-0.15988	-0.43767
		0.0117	0.4582	0.3767	0.3821	0.0122
	33	33	32	32	32	32
sertriglyc	-0.43385	1.00000	-0.03893	0.49626	0.27484	0.14440
	0.0117		0.8325	0.0039	0.1279	0.4304
	33	33	32	32	32	32
leptin	-0.13593	-0.03893	1.00000	0.31670	0.56995	0.29037

	0.4582	0.8325		0.0826	0.0008	0.1130
	32	32	32	31	31	31
insulin	-0.16167	0.49626	0.31670	1.00000	0.82634	0.14597
	0.3767	0.0039	0.0826		<.0001	0.4333
	32	32	31	32	32	31
homair	-0.15988	0.27484	0.56995	0.82634	1.00000	0.16430
	0.3821	0.1279	0.0008	<.0001		0.3771
	32	32	31	32	32	31
waisthip	-0.43767	0.14440	0.29037	0.14597	0.16430	1.00000
	0.0122	0.4304	0.1130	0.4333	0.3771	
	32	32	31	31	31	32

---------- Ethnicity=3 ----------

The CORR Procedure

20 Variables: Age Height Weight bmi MAMC TricepSF Hip Waist FFM percbodFAT systolic diastolic serglucose stotchol serHDL sertriglyc leptin insulin homair waisthip

Simple Statistics

Variable	N	Mean	Std Dev	Sum	Minimum	Maximum
Age	48	34.06250	5.92293	1635	22.00000	45.00000
Height	48	64.83333	1.63516	3112	61.50000	67.50000
Weight	48	193.04167	38.74121	9266	107.50000	300.50000
bmi	48	32.37613	6.55918	1554	18.20413	52.50226
MAMC	47	33.10426	4.42802	1556	24.50000	46.00000
TricepSF	48	32.25729	7.83474	1548	13.70000	48.00000
Hip	48	117.12083	13.14876	5622	92.00000	150.00000
Waist	48	106.27708	13.93907	5101	77.50000	145.00000
FFM	47	109.41489	12.79619	5143	83.50000	138.50000
percbodFAT	47	42.04894	6.68778	1976	22.50000	54.70000
systolic	44	124.22727	12.01523	5466	97.00000	145.00000
diastolic	44	80.75000	9.42319	3553	58.00000	100.00000
serglucose	48	112.50000	33.14443	5400	63.00000	262.00000
stotchol	48	196.75000	44.45246	9444	70.00000	274.00000
serHDL	48	47.95833	16.16023	2302	17.20000	87.90000
sertriglyc	48	145.64583	91.79568	6991	12.00000	476.00000
leptin	48	28.04396	12.31622	1346	4.20000	60.32000
insulin	47	41.62128	34.06625	1956	5.47000	171.18000
homair	47	5.03205	0.79468	236.50639	3.13964	6.84138
waisthip	48	0.90752	0.06173	43.56108	0.75278	1.08527

---------- Ethnicity=3 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Age	Height	Weight	bmi	MAMC	TricepSF	Hip
Age	1.00000	0.20651	0.20862	0.14871	0.02977	-0.00780	0.13276
		0.1591	0.1547	0.3131	0.8426	0.9580	0.3684
	48	48	48	48	47	48	48
Height	0.20651	1.00000	0.11968	-0.12848	-0.16599	-0.05903	0.03421
	0.1591		0.4178	0.3842	0.2648	0.6902	0.8175
	48	48	48	48	47	48	48
Weight	0.20862	0.11968	1.00000	0.96856	0.77323	0.70978	0.91700
	0.1547	0.4178		<.0001	<.0001	<.0001	<.0001
	48	48	48	48	47	48	48
bmi	0.14871	-0.12848	0.96856	1.00000	0.80177	0.72275	0.90985
	0.3131	0.3842	<.0001		<.0001	<.0001	<.0001
	48	48	48	48	47	48	48

MAMC	0.02977	-0.16599	0.77323	0.80177	1.00000	0.73208	0.64711
	0.8426	0.2648	<.0001	<.0001		<.0001	<.0001
	47	47	47	47	47	47	47
TricepSF	-0.00780	-0.05903	0.70978	0.72275	0.73208	1.00000	0.64330
	0.9580	0.6902	<.0001	<.0001	<.0001		<.0001
	48	48	48	48	47	48	48
Hip	0.13276	0.03421	0.91700	0.90985	0.64711	0.64330	1.00000
	0.3684	0.8175	<.0001	<.0001	<.0001	<.0001	
	48	48	48	48	47	48	48
Waist	0.20528	0.10681	0.82956	0.79933	0.65955	0.53359	0.84382
	0.1616	0.4700	<.0001	<.0001	<.0001	<.0001	<.0001
	48	48	48	48	47	48	48
FFM	0.20410	0.08215	0.86204	0.83784	0.62627	0.58052	0.77715
	0.1688	0.5830	<.0001	<.0001	<.0001	<.0001	<.0001
	47	47	47	47	46	47	47
percbodFAT	0.13321	0.16208	0.83730	0.79685	0.69509	0.63473	0.78406
	0.3720	0.2764	<.0001	<.0001	<.0001	<.0001	<.0001
	47	47	47	47	46	47	47
systolic	0.06306	0.03463	0.42638	0.40922	0.42402	0.27246	0.40422
	0.6842	0.8234	0.0039	0.0058	0.0041	0.0736	0.0065
	44	44	44	44	44	44	44

---------- Ethnicity=3 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Age	Height	Weight	bmi	MAMC	TricepSF	Hip
diastolic	-0.14368	-0.08552	0.31719	0.33302	0.39160	0.37555	0.24561
	0.3521	0.5810	0.0359	0.0272	0.0086	0.0120	0.1081
	44	44	44	44	44	44	44
serglucose	-0.03593	0.01983	0.18441	0.18152	0.03836	0.27663	0.25802
	0.8084	0.8936	0.2096	0.2169	0.7979	0.0570	0.0766
	48	48	48	48	47	48	48
stotchol	0.15457	0.22246	-0.04217	-0.10314	-0.18024	-0.06201	-0.02734
	0.2942	0.1286	0.7760	0.4854	0.2254	0.6755	0.8536
	48	48	48	48	47	48	48
serHDL	-0.09236	0.13935	-0.23254	-0.27387	-0.07873	-0.07151	-0.21541
	0.5324	0.3449	0.1117	0.0596	0.5988	0.6291	0.1415
	48	48	48	48	47	48	48
sertriglyc	0.19199	-0.13386	0.03974	0.07110	-0.07912	-0.08994	0.15501
	0.1911	0.3644	0.7886	0.6311	0.5970	0.5432	0.2928
	48	48	48	48	47	48	48
leptin	0.04870	0.04606	0.71515	0.70786	0.65763	0.47562	0.68908
	0.7424	0.7559	<.0001	<.0001	<.0001	0.0006	<.0001
	48	48	48	48	47	48	48
insulin	0.14755	0.00718	0.43984	0.44646	0.45054	0.21789	0.40467
	0.3223	0.9618	0.0020	0.0017	0.0017	0.1412	0.0048
	47	47	47	47	46	47	47
homair	0.12302	0.02795	0.50096	0.50031	0.45940	0.32135	0.50853
	0.4100	0.8521	0.0003	0.0003	0.0013	0.0276	0.0003
	47	47	47	47	46	47	47
waisthip	0.13475	0.12691	0.08631	0.04615	0.16064	-0.02143	-0.01599
	0.3612	0.3900	0.5597	0.7554	0.2807	0.8850	0.9141
	48	48	48	48	47	48	48

---------- Ethnicity=3 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Waist	FFM	percbod FAT	systolic	diastolic	serglucose	stotchol
Age	0.20528	0.20410	0.13321	0.06306	-0.14368	-0.03593	0.15457
	0.1616	0.1688	0.3720	0.6842	0.3521	0.8084	0.2942
	48	47	47	44	44	48	48
Height	0.10681	0.08215	0.16208	0.03463	-0.08552	0.01983	0.22246
	0.4700	0.5830	0.2764	0.8234	0.5810	0.8936	0.1286
	48	47	47	44	44	48	48
Weight	0.82956	0.86204	0.83730	0.42638	0.31719	0.18441	-0.04217
	<.0001	<.0001	<.0001	0.0039	0.0359	0.2096	0.7760
	48	47	47	44	44	48	48
bmi	0.79933	0.83784	0.79685	0.40922	0.33302	0.18152	-0.10314
	<.0001	<.0001	<.0001	0.0058	0.0272	0.2169	0.4854
	48	47	47	44	44	48	48
MAMC	0.65955	0.62627	0.69509	0.42402	0.39160	0.03836	-0.18024
	<.0001	<.0001	<.0001	0.0041	0.0086	0.7979	0.2254
	47	46	46	44	44	47	47
TricepSF	0.53359	0.58052	0.63473	0.27246	0.37555	0.27663	-0.06201
	<.0001	<.0001	<.0001	0.0736	0.0120	0.0570	0.6755
	48	47	47	44	44	48	48
Hip	0.84382	0.77715	0.78406	0.40422	0.24561	0.25802	-0.02734
	<.0001	<.0001	<.0001	0.0065	0.1081	0.0766	0.8536
	48	47	47	44	44	48	48
Waist	1.00000	0.61578	0.80004	0.33381	0.12723	0.22131	-0.06254
		<.0001	<.0001	0.0268	0.4105	0.1306	0.6728
	48	47	47	44	44	48	48
FFM	0.61578	1.00000	0.48219	0.43050	0.29431	0.20524	0.00841
	<.0001		0.0006	0.0040	0.0554	0.1664	0.9552
	47	47	47	43	43	47	47
percbodFAT	0.80004	0.48219	1.00000	0.38035	0.31096	0.13279	-0.10614
	<.0001	0.0006		0.0119	0.0424	0.3736	0.4777
	47	47	47	43	43	47	47
systolic	0.33381	0.43050	0.38035	1.00000	0.71017	-0.01223	-0.02238
	0.0268	0.0040	0.0119		<.0001	0.9372	0.8853
	44	43	43	44	44	44	44

---------- Ethnicity=3 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Waist	FFM	percbod FAT	systolic	diastolic	serglucose	stotchol
diastolic	0.12723	0.29431	0.31096	0.71017	1.00000	-0.14737	-0.00817
	0.4105	0.0554	0.0424	<.0001		0.3398	0.9580
	44	43	43	44	44	44	44
serglucose	0.22131	0.20524	0.13279	-0.01223	-0.14737	1.00000	0.08890
	0.1306	0.1664	0.3736	0.9372	0.3398		0.5479
	48	47	47	44	44	48	48
stotchol	-0.06254	0.00841	-0.10614	-0.02238	-0.00817	0.08890	1.00000
	0.6728	0.9552	0.4777	0.8853	0.9580	0.5479	
	48	47	47	44	44	48	48

serHDL	-0.06582	-0.32150	-0.09936	-0.00489	0.05165	-0.03529	0.40375
	0.6567	0.0276	0.5064	0.9749	0.7392	0.8118	0.0044
	48	47	47	44	44	48	48
sertriglyc	0.10458	0.17555	-0.06337	-0.09579	-0.21959	0.24902	0.35936
	0.4793	0.2379	0.6722	0.5362	0.1521	0.0879	0.0121
	48	47	47	44	44	48	48
leptin	0.56432	0.54761	0.66557	0.32266	0.27794	0.08427	0.12212
	<.0001	<.0001	<.0001	0.0327	0.0677	0.5691	0.4083
	48	47	47	44	44	48	48
insulin	0.36265	0.37431	0.35968	0.09966	-0.06512	0.11948	0.01932
	0.0122	0.0104	0.0141	0.5249	0.6782	0.4238	0.8974
	47	46	46	43	43	47	47
homair	0.40403	0.49196	0.39892	0.14893	-0.09357	0.37557	0.07723
	0.0049	0.0005	0.0060	0.3405	0.5507	0.0093	0.6059
	47	46	46	43	43	47	47
waisthip	0.51952	-0.09809	0.25549	-0.03453	-0.15958	-0.00683	-0.05292
	0.0002	0.5118	0.0830	0.8239	0.3008	0.9632	0.7209
	48	47	47	44	44	48	48

---------- Ethnicity=3 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	serHDL	sertriglyc	leptin	insulin	homair	waisthip
Age	-0.09236	0.19199	0.04870	0.14755	0.12302	0.13475
	0.5324	0.1911	0.7424	0.3223	0.4100	0.3612
	48	48	48	47	47	48
Height	0.13935	-0.13386	0.04606	0.00718	0.02795	0.12691
	0.3449	0.3644	0.7559	0.9618	0.8521	0.3900
	48	48	48	47	47	48
Weight	-0.23254	0.03974	0.71515	0.43984	0.50096	0.08631
	0.1117	0.7886	<.0001	0.0020	0.0003	0.5597
	48	48	48	47	47	48
bmi	-0.27387	0.07110	0.70786	0.44646	0.50031	0.04615
	0.0596	0.6311	<.0001	0.0017	0.0003	0.7554
	48	48	48	47	47	48
MAMC	-0.07873	-0.07912	0.65763	0.45054	0.45940	0.16064
	0.5988	0.5970	<.0001	0.0017	0.0013	0.2807
	47	47	47	46	46	47
TricepSF	-0.07151	-0.08994	0.47562	0.21789	0.32135	-0.02143
	0.6291	0.5432	0.0006	0.1412	0.0276	0.8850
	48	48	48	47	47	48
Hip	-0.21541	0.15501	0.68908	0.40467	0.50853	-0.01599
	0.1415	0.2928	<.0001	0.0048	0.0003	0.9141
	48	48	48	47	47	48
Waist	-0.06582	0.10458	0.56432	0.36265	0.40403	0.51952
	0.6567	0.4793	<.0001	0.0122	0.0049	0.0002
	48	48	48	47	47	48
FFM	-0.32150	0.17555	0.54761	0.37431	0.49196	-0.09809
	0.0276	0.2379	<.0001	0.0104	0.0005	0.5118
	47	47	47	46	46	47
percbodFAT	-0.09936	-0.06337	0.66557	0.35968	0.39892	0.25549
	0.5064	0.6722	<.0001	0.0141	0.0060	0.0830
	47	47	47	46	46	47
systolic	-0.00489	-0.09579	0.32266	0.09966	0.14893	-0.03453
	0.9749	0.5362	0.0327	0.5249	0.3405	0.8239
	44	44	44	43	43	44

---------- Ethnicity=3 ----------

The CORR Procedure

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	serHDL	sertriglyc	leptin	insulin	homair	waisthip
diastolic	0.05165	-0.21959	0.27794	-0.06512	-0.09357	-0.15958
	0.7392	0.1521	0.0677	0.6782	0.5507	0.3008
	44	44	44	43	43	44
serglucose	-0.03529	0.24902	0.08427	0.11948	0.37557	-0.00683
	0.8118	0.0879	0.5691	0.4238	0.0093	0.9632
	48	48	48	47	47	48
stotchol	0.40375	0.35936	0.12212	0.01932	0.07723	-0.05292
	0.0044	0.0121	0.4083	0.8974	0.6059	0.7209
	48	48	48	47	47	48
serHDL	1.00000	-0.08327	0.01854	0.04613	0.00696	0.21101
		0.5736	0.9005	0.7582	0.9630	0.1500
	48	48	48	47	47	48
sertriglyc	-0.08327	1.00000	0.06137	0.26609	0.29016	-0.02871
	0.5736		0.6786	0.0706	0.0479	0.8464
	48	48	48	47	47	48
leptin	0.01854	0.06137	1.00000	0.47323	0.56351	-0.04455
	0.9005	0.6786		0.0008	<.0001	0.7637
	48	48	48	47	47	48
insulin	0.04613	0.26609	0.47323	1.00000	0.87311	0.01296
	0.7582	0.0706	0.0008		<.0001	0.9311
	47	47	47	47	47	47
homair	0.00696	0.29016	0.56351	0.87311	1.00000	-0.07175
	0.9630	0.0479	<.0001	<.0001		0.6318
	47	47	47	47	47	47
waisthip	0.21101	-0.02871	-0.04455	0.01296	-0.07175	1.00000
	0.1500	0.8464	0.7637	0.9311	0.6318	
	48	48	48	47	47	48

The CORR Procedure

7 Variables: Waist systolic Age serHDL stotchol sertriglyc homair

Simple Statistics

Variable	N	Mean	Std Dev	Sum	Minimum	Maximum
Waist	81	107.28765	17.39697	8690	69.50000	150.00000
systolic	72	125.91667	12.82466	9066	97.00000	160.00000
Age	81	34.23457	5.79714	2773	22.00000	45.00000
serHDL	81	49.98395	16.41779	4049	17.20000	87.90000
stotchol	81	195.95062	40.83592	15872	70.00000	274.00000
sertriglyc	81	125.24691	82.75997	10145	1.00000	476.00000
homair	79	5.01620	0.92057	396.27968	2.63147	7.08852

Pearson Correlation Coefficients
Prob > |r| under H0: Rho=0
Number of Observations

	Waist	systolic	Age	serHDL	stotchol	sertriglyc	homair
Waist	1.00000	0.26887	0.12741	-0.14724	-0.06140	0.04981	0.45538
		0.0224	0.2570	0.1896	0.5861	0.6588	<.0001
	81	72	81	81	81	81	79
systolic	0.26887	1.00000	-0.01263	-0.04765	-0.13391	-0.15742	0.12076

	0.0224		0.9161	0.6910	0.2621	0.1866	0.3194
	72	72	72	72	72	72	70
Age	0.12741	-0.01263	1.00000	-0.00147	0.17678	0.11439	-0.01052
	0.2570	0.9161		0.9896	0.1144	0.3092	0.9267
	81	72	81	81	81	81	79
serHDL	-0.14724	-0.04765	-0.00147	1.00000	0.48046	-0.21833	-0.07365
	0.1896	0.6910	0.9896		<.0001	0.0502	0.5189
	81	72	81	81	81	81	79
stotchol	-0.06140	-0.13391	0.17678	0.48046	1.00000	0.25065	0.06008
	0.5861	0.2621	0.1144	<.0001		0.0240	0.5989
	81	72	81	81	81	81	79
sertriglyc	0.04981	-0.15742	0.11439	-0.21833	0.25065	1.00000	0.26082
	0.6588	0.1866	0.3092	0.0502	0.0240		0.0203
	81	72	81	81	81	81	79
homair	0.45538	0.12076	-0.01052	-0.07365	0.06008	0.26082	1.00000
	<.0001	0.3194	0.9267	0.5189	0.5989	0.0203	
	79	70	79	79	79	79	79

Smoking levels among ethnic groups

chi analysis for smoking

The FREQ Procedure

Table of ethnicity by smoke

ethnicity smoke

Frequency, Expected , Percent , Row Pct , Col Pct	1	2	Total
1	6 12.375 7.50 18.18 20.00	27 20.625 33.75 81.82 54.00	33 41.25
3	24 17.625 30.00 51.06 80.00	23 29.375 28.75 48.94 46.00	47 58.75
Total	30 37.50	50 62.50	80 100.00

Frequency Missing = 1

chi analysis for smoking

The FREQ Procedure

Statistics for Table of ethnicity by smoke

Statistic	DF	Value	Prob
Chi-Square	1	8.9439	0.0028
Likelihood Ratio Chi-Square	1	9.4224	0.0021
Continuity Adj. Chi-Square	1	7.5960	0.0058
Mantel-Haenszel Chi-Square	1	8.8321	0.0030
Phi Coefficient		-0.3344	
Contingency Coefficient		0.3171	
Cramer's V		-0.3344	

Fisher's Exact Test

Cell (1,1) Frequency (F)	6
Left-sided Pr <= F	0.0025
Right-sided Pr >= F	0.9995
Table Probability (P)	0.0020
Two-sided Pr <= P	0.0045

Effective Sample Size = 80
Frequency Missing = 1

chi analysis for smoking

The FREQ Procedure

Summary Statistics for ethnicity by smoke

Cochran-Mantel-Haenszel Statistics (Based on Table Scores)

Statistic	Alternative Hypothesis	DF	Value	Prob
1	Nonzero Correlation	1	8.8321	0.0030
2	Row Mean Scores Differ	1	8.8321	0.0030
3	General Association	1	8.8321	0.0030

Estimates of the Common Relative Risk (Row1/Row2)

Type of Study	Method	Value	95% Confidence Limits	
Case-Control (Odds Ratio)	Mantel-Haenszel	0.2130	0.0743	0.6106
	Logit	0.2130	0.0743	0.6106
Cohort (Col1 Risk)	Mantel-Haenszel	0.3561	0.1639	0.7736
	Logit	0.3561	0.1639	0.7736
Cohort (Col2 Risk)	Mantel-Haenszel	1.6719	1.1979	2.3335
	Logit	1.6719	1.1979	2.3335

Effective Sample Size = 80
Frequency Missing = 1

VITA

Archana Ellath

Candidate for the Degree of

Master of Science

Thesis: CARDIOVASCULAR RISK FACTORS IN AMERICAN INDIAN AND AFRICAN AMERICAN WOMEN OF CHILD BEARING AGE AND THEIR RELATIONSHIP TO BLOOD LEPTIN CONCENTRATION, INSULIN RESISTANCE, AND WAIST CIRCUMFERENCE

Major Field: Nutritional Sciences

Education:
Graduate from Christhu Jyothi Convent, Erode, India in April 1996; received Bachelor of Science degree in Clinical Nutrition and Dietetics, Avinashilingam Deemed University, Coimbatore, India in May 1999; received Masters in International Business from Bharathiyar University, Coimbatore, India in May 2001.

Experience:
Worked as a Trainee Dietitian in a hospital; employed by Oklahoma State University department of Nutritional Sciences as a teaching assistant and research assistant.

Awards and Honors
Scholarship recipient, Study Abroad Travel Grant, PFIZER Inc., New York, U.S.A
Fellowship award, International Food and Nutrition Conference Global Fellow Student, 2006

Professional Memberships:
Institute of Food Technologists (IFT), Kappa Omicron Nu

Name: Archana Ellath Date of Degree: December, 2006

Institution: Oklahoma State University Location: Stillwater, Oklahoma

Title of Study: CARDIOVASCULAR RISK FACTORS IN AMERICAN INDIAN AND AFRICAN AMERICAN WOMEN OF CHILD BEARING AGE AND THEIR RELATIONSHIP TO BLOOD LEPTIN CONCENTRATION, INSULIN RESISTANCE AND WAIST CIRCUMFERENCE

Pages in Study: 126 Candidate for the Degree of Master of Science
Major Field: Nutritional Sciences

Scope and Method of Study: Health disparities and cardiovascular diseases are major concerns in the nation. Cardiovascular disease is the leading cause of death among American Indian (AI) and African American (AA) women. Leptin concentrations, insulin resistance, and waist circumference have been found to be predictors of cardiovascular disease in American Indians and African Americans but little is known about AI and AA women of child bearing age. This study was designed to compare leptin levels, insulin resistance and waist circumference in AI and AA women and to determine the relationship of these variables with systolic blood pressure, HDL cholesterol and total cholesterol.

This is a prospective epidemiological study of 81 women (48 AI, 33 AA women) of child bearing age from rural Oklahoma. Anthropometric, blood pressure and biochemical data were collected from this cohort and analyzed using SAS (Version 9.1.3).

Findings and Conclusions: Ninety percent of the women were overweight to obese with a mean BMI of 33.2±7.3 kg/m^2. Fifty eight percent of the AI women and 61% of the AA women were obese. Leptin concentrations were significantly higher for AA women when compared to AI women. Waist circumference (107±17 cm) and insulin resistance (5±0.9) were not significantly different between the groups but are alarmingly morbid. There was a positive but weak correlation between leptin and systolic blood pressure (SBP, $r=0.335$, $p<0.005$) and between waist circumference and SBP ($r=0.269$, $p<0.05$) but not insulin resistance with SBP. The mean SBP in this cohort was 126±12 mm Hg, within the borderline high range. Although leptin concentrations were significantly different between both groups, insulin resistance and waist circumference were not. This suggests that there is a phenotypic difference in the etiology of CVD and the involvement of leptin in the development of heart disease.

ADVISOR'S APPROVAL: Dr. Maria Spicer