CARDIOVASCULAR RISK FACTORS AMONG PRIMARY SCHOOL CHILDREN AGED 6 – 15 YEARS IN URBAN DAR ES SALAAM AND RURAL MOROGORO.

Prevalence, Awareness and Knowledge.

By

Dr. Pilly Chillo, MD.

A dissertation submitted in partial fulfilment of the requirements for the degree of Masters of Medicine (Internal Medicine) of the University of Dar es Salaam.

University of Dar es Salaam
September 2002

CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by the University of Dar es Salaam a dissertation entitled: ***Cardiovascular Risk Factors among Primary school children aged 6 – 15 years in Urban Dar es Salaam and Rural Morogoro,*** in partial fulfilment of the requirements for the degree of Master of Medicine (Internal Medicine).

....
Dr. J.M. Lwakatare
(SUPERVISOR)

....
Dr. Gottfried Greve
(SECOND SUPERVISOR)

Date 4/10 - 02

DECLARATION AND COPYRIGHT

I, Pilly Chillo, declare that this dissertation is my own original work and that it has not been presented and will not be presented to any other University for a similar or any other degree award.

Signature.........................

ACKOWLEDGEMENT

I would like to thank Prof W. Matuja, Head of the Department of Internal Medicine and all other members of the department for the support they have given me during the course of this work. Special thanks goes to my supervisor, Dr J. Lwakatare for all the tireless efforts he put from the beginning to the completion of this work, without him this dissertation would have been a total failure.

I appreciate and give a big thank the Coordinator of the Bergen-MUCHS sandwich program, Prof Nina Langeland for the financial support, as well as for giving me the chance to use their laboratory for lipids analyses while in Bergen. Dr. Gottfried Greve, my co-supervisor, has been of great help while in Bergen with regard to data handling, reading and correcting this work at the very beginning of my write up.

I thank Mr. Makwaya and Mr Method Kazaura for helping me with the statistics, Mr. Abel Mweta for typesetting and printing this work. I also thank Prof George Lwihula for the assistance he gave me on the social and behavioral part of my work, Mr. Msuya for helping me with blood samples collection and initial preparation while in Dar es Salaam, Ms Tone Minde, laboratory technician in Bergen, for helping with lipid analyses.

I appreciate the valuable comments given by all academic and non-academic members of staff in the Department of Internal Medicine during the formulation and write up of this work, your comments were all constructive. I cannot mention everyone here, but Dr M. Janabi deserves special thanks for all the kinds of help and support he gave me during the course of this work.

Lastly, my sincere thanks go to my parents, brothers, sisters and all my well wishers for their love, support and encouragement.

DEDICATION

To my parents, for making me who I am.

LIST OF ABBREVIATIONS

BMI	Body Mass Index
CVD	Cardiovascular Disease
DBP	Diastolic Blood Pressure
HDL	High Density Lipoprotein
LDL	Low Density Lipoprotein
SBP	Systolic Blood Pressure
UHD	Urban High Density
ULD	Urban Low Density
WHO	World Health Organization

ABSTRACT

Background: Cardiovascular diseases, once known as diseases of the Western world have recently been emerged as diseases of public health importance in many developing countries. Change in life style and socio-economic status associated with urbanization is thought to be the major contributing factor for this trend. In its message to the developing countries, the World Health Organization has urged for a prompt control of cardiovascular diseases at an early stage before the burden becomes more severe. The need for Primordial Prevention has highly been recommended.

Objective: To determine the prevalence, awareness and knowledge of the conventional cardiovascular risk factors among primary school children aged 6 – 15 years in Urban Dar es Salaam and Rural Morogoro.

Methods: Cross sectional survey of children aged 6 – 15 years from four primary schools in Urban Dar es Salaam and two primary schools in Rural Morogoro. Anthropometric and Blood Pressure measurements were done using the standard techniques, blood samples taken and analysed for sugar, total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides levels. A standard questionnaire was used to obtain sociodemographic characteristics and to assess the level of awareness and knowledge of the main cardiovascular diseases among the children.

Results: The prevalence of systolic, diastolic and both (systolic and diastolic) hypertension was found to be 11.4%, 8.1% and 3.9% respectively among the 508 children studied. Twenty one (4.1%) subjects had high total cholesterol levels, while high LDL-C levels were found in 10 subjects (2%). The prevalence of obesity was found to be 5.3%. Only six (1.2%) subjects were having active cigarette smoking. Passive smoking was found in 35% of the total study population. Majority of the study population were generally active. None of the subjects had blood sugar levels that were equivalent to diabetic levels.

Subjects from the Urban Low Density schools were more likely to be hypertensive, obese and with high levels of total cholesterol and LDL-C, furthermore they were more likely to have less physical activities as compared to those from the Urban High Density and Rural schools. In addition, subjects from ULD schools were more likely to be aware and to have more knowledge of cardiovascular diseases as compared to those from the UHD and Rural schools.

Conclusion: Cardiovascular risk factors are generally low among children in this society although certain groups of children have

increased risk. There is low level of awareness and knowledge of cardiovascular diseases and their precursors among children.

'ABLE OF CONTENTS

1. 0 INTRODUCTION AND LITERATURE REVIEW

1. 1 Overview of cardiovascular diseases

Cardiovascular diseases are diseases of the heart and arteries. The main pathological changes that occur in these arteries is atherosclerosis which in simple terms means hardening and plugging of arterial vessels walls.[1]

The process of atherosclerosis most commonly occur in coronary and cerebral blood vessels, typically causing angina and heart attacks as a result of coronary artery affection, also strokes as a manifestation of the cerebral blood vessels involvement.

The distribution of lesions among these sites may be determined by genetic as well as environmental conditions. In Europe and North America, dyslipidaemia and coronary artery atherosclerosis are very prevalent, while persons in Africa and Asia are more likely to exhibit hypertension and cerebrovascular arterial involvement.[1]

The main mechanism as to how atherosclerosis is brought about is still not fully understood, although a number of risk factors for atherosclerosis and hence cardiovascular diseases are well recognized.[2] The risk factors for Cardiovascular diseases are traditionally divided into fixed and modifiable risk factors. The former include male gender and family history of cardiovascular diseases while the modifiable factors include Hypertension, Diabetes Mellitus, Cigarrette smoking, Inactivity, Obesity, Low levels of High Density Lipoproteins and intake of high levels of saturated fatty acids.[2]

Given the fact that most of the cardiovascular risk factors are modifiable, these diseases are sometimes known as diseases of lifestyles, meaning that behavioral lifestyles play a major role in the occurance of cardiovascular diseases. It is also known that modification of risk-related behaviours can, in a great way reduce the likelihood of having cardiovascular diseases.[2]

Atherogenesis develops as a decade-long process in which the lumen of a blood vessel becomes narrowed by cellular and extracellular substances to the point of being obstructed.[3] The earliest lesion of atherosclerosis is known to be the fatty streak[4], which is a grossly visible area of yellowish discolouration on the surface of the intimal layer of the vessel wall.

In the third decade of life, some atheromatous lesions evolve into complicated fibrous plaques, consisting of a central acellular area of lipid, covered by a cap of smooth muscle cells and collagen.[3] Caps tend to form slowly at first, which appear to be the result of endothelial injury; then the caps thicken quickly, possibly as a result of thrombosis-dependent fibrotic organization.[5]

The progression of early atherosclerotic lesions to clinically relevant advanced atherosclerotic lesions occurs with increased frequency in persons with one or more of the risk factors for atherosclerosis.[6]

Over the past 20 years the world has witnessed an enormous improvement in the understanding of cardiovascular diseases.[7] The pathophysiology of risk factors such as hypertension, hyperlipidemia, diabetes and hyperhomocystinaemia has largerly been unravelled. For instance, the biochemical pathways that regulate cholesterol synthesis in the liver cell and the role of low-density lipoprotein (LDL) receptors in clearing LDL from the circulation have been elucidated.[8] We have also become familiar with blood-pressure regulation systems such as the renin-angiotensin system and natriuretic peptides.[9] This knowledge has led directly to the introduction of new drugs such as statins and angiotensin-converting enzyme (ACE) inhibitors, which specifically interfere with these regulatory pathways. More recently we have seen the success of these drugs in clinical medicine. Large trials investigating the effects of lipid-lowering treatment have demonstrated that secondary prevention [as in the Cholesterol and Recurrent Events (CARE)[10], Scandinavian Simvastatin Survival Study (4S)[11], Regression Growth Evaluation Study (REGRESS)[12] and Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID)[13] trials] as well as primary prevention [as in the West of Scotland Coronary Prevention Study (WOSCOPS)[14] trial with statins cause remarkable reductions in cardiovascular mortality]. Similarly, in the Hypertension Optimal Treatment (HOT)[15] study, blood-pressure reduction in hypertensive patients to 85 mmHg was associated with a reduction in cardiovascular mortality.

Although the success of the drugs in reducing cardiovascular risk is impressive, it is not sufficient to control the pandemic of cardiovascular diseases. Over 100 million people a year worldwide die from cardiovascular diseases, and the numbers are still increasing, mainly in third world countries.[16] Therefore, to improve our understanding and treatment options further, we have to take a different perspective. Two issues are crucial in this respect. First, most patients have multiple risk factors, some of them still unknown. These risk factors potentiate each other with regard to cardiovascular risk.[17] It is necessary therefore to understand how risk factors interact with each other and to identify any final common pathways in their pathogenesis. Second, patients with vascular diseases usually present themselves late in the disease process when clinical symptoms have developed and the process of vascular damage has been ongoing for 15 to 20 years i.e normally starts in childhood.[18] The issue of drug costs need also to be addressed. In a country like Tanzania, drug therapy for patients with cardiovascular diseases will bring more burden to the country as these drugs are costly and people are still suffering from infectious diseases which account for most of the Ministry of Health's drug budget.

Therefore, recognising the signs and understanding the pathobiology of much ealier stages will allow new preventive strategies. Interestingly, all major cardiovascular risk factors can impair endothelial function at a very early stage, before any clinical disease can be detected.[18]

2 The problem of Cardiovascular diseases.

Cardiovascular diseases are among the most feared diseases all over the world.[19] These diseases of the heart and arteries account for a quarter of the total number of deaths each year, taking an estimated 12 million lives worldwide annually.[19] They kill more people than any other single disease, and disable millions.[19] Even worse, many victims are under the age of 65 years and given the today's life expectance, these deaths are premature, causing anguish in families and loss of the talents that many countries need for economic development.[19]

The epidemiology of cardiovascular diseases has been vigorously investigated during the last 50 years. During this time, the international cooperation of cardiovascular epidemiologists and other scientists first documented the rapid rise in mortality attributed to cardiovascular diseases and its sequelae in developed countries.[20,21] However, during the past 30 years, large declines in cardiovascular diseases death rates have been experienced in several western countries, whereas substantial increases have been experienced in developing countries.[22] Unfortunately these contrasting trends are expected to continue. Over the next three decades, premature morbidity and mortality attributable to cardiovascular diseases will almost double globally from 85 million Disability Adjusted Life Years (DALY) in 1990, to 140-160 million DALY in 2020, with about 80% of this burden occuring in developing countries.[23]

Estimates of the World Health Organization show that in absolute figures there are already as many people dying each year from cardiovascular diseases in the developing world- 6 millions as in the developed world.[19] People in developing countries are at the same time suffering from forms of heart diseases that are not prevalent elsewhere, such as rheumatic heart diseases, which is linked to poverty, and a heart condition triggered by Chaga's disease, a parasitic malady that afflicts about 17 million Latin Americans.[19] To these countries, the diseases of lifestyle are an added burden on the scarce resources available.

The increases in cardiovascular diseases in developing countries are probably a result of at least three contributing factors.[23] First, decreasing mortality from infectious diseases and increases in life expectancy results in a higher proportion of individuals reaching middle and old age. Second, lifestyle and socioeconomic changes associated with urbanization in developing countries lead to higher levels of risk factors for cardiovascular diseases. Third, special susceptibility of certain populations (e.g. due to specific genes) may lead to a greater impact on clinical events compared to western populations.

The problem of cardiovascular diseases is generally low in Tanzania[31,32] as is in most Sub-Saharan Africa.[24,25,26] This situation is however changing and there is evidence from some developing countries in Asia, South America and the Carribean and within specific societies in African countries.[27,28,29,30]

Studies done in Tanzania to determine the prevalence of risk factors for cardiovascular diseases have shown a generally low prevalence of these risk factors namely hypertension, overweight and obesity, hypercholesterolemia, impared glucose tolerence and diabetes. Kitange et al[31] found the prevalence of Hypertension, Diabetes, Hypercholesterolemia and cigarrette smoking in Tanzanian Urban and Rural adolescents aged 15-19 years to be 0.4%, 0.28%, 7% and 7.3% respectively. Their conclusion was, within populations certain risk factors do exsist at individual adolescents and these are related to dietary pattern.

A similar study done by the same group of investigators among Tanzanian adults showed a generally increased levels of risk factors for cardiovascular diseases as compared to adolescents but lower than those reported from developed countries.[32] There was also a tendency of the risk factors to be more prevalent in the city of Dar es Salaam and Kilimanjaro region which are the most prosperous regions. In Dar es Salaam adults, the prevalence of hypertension, diabetes mellitus, hypercholesterolemia and cigarrette smoking was found to be 13%, 1.1%, 15% and 21% respectively, while the prevalences in Morogoro were, 4%, 1%, 7% and 28% respectively.[32]

1. 3. 0 Cardiovascular risk factors in children

It has become quite clear that the process of atherosclerosis begins early in life.[33] Autopsy studies of men killed in the Korean War[35], and the Vietnem war[36] documented significant atheromatous lesions. In the earlier study, more than three quarters of male soldiers (mean age 22) had fibrous plaques in their coronary arteries; in the latter investigation of men somewhat younger, nearly half had coronary lesions. Autopsies in Louisiana indicated that, even in the absence of the stress of combat, atherosclerotic lesions are found in adolescents and young adults.[37] Fatty streaks and intimal thickenings have been found in most Finnish and American children[38], leading to some people to question their importance. However, the extent of arterial fatty streaking in children has been directly correlated with systolic blood pressure and levels of blood total and LDL cholesterol determined earlier in childhood.[39] Further, progression of these lesions to fibrous plaques have been demonstrated. Although white adults have more extensive aortic surface involvement with fibrous plaques than do blacks, adolescent blacks have been found to have more fatty streaks than their white contemporaries.[40]

Several longitudinal studies of children were designed to assess characteristics that are indicative of coronary artery disease in adults. Within North America, large-scale investigations have been carried out in Bogalusa, Louisiana[41], Cincinnati, Ohio[42] and Muscatine, Iowa.[43] The Bogalusa study, which began in 1973, has followed cohorts of black and white children from birth to 17 years of age. It described ``tracking´´ of height, weight, blood pressure and lipid and lipoprotein values, indicating that children tend to remain in the same relative ranking through their young adult years.[44,45,46] Similar findings have been reported by Muscatine and Cincinnati investigators.[47,48]

1. 3. 1 Hypertension:

Raised blood pressure is a risk factor for both coronary heart disease and stroke.[49] According to epidemiological studies, and after adjustment for confounding factors, high blood pressure alone is an important risk factor for cardiovascular diseases.[50] Furthermore, the treatment of hypertension reduces the risk of cardiovascular diseases in clinical drug intervention trials.[15] More so, lifestyle interventions for mildly eleveted blood pressure have also been effective in risk reduction.[51]

Although less is known about the distribution of blood pressure in children than in adults, ample evidence now supports the idea that the roots of essential hypertension extend back to early adulthood. There is evidence that the hypertensive process begins in childhood[52], with numerous studies having found correlations between blood pressure levels from early into late childhood[53] and from childhood into adulthood.[54] These reports suggest the importance of tracking blood pressure in children to detect the early stages of hypertension.

In children, the blood pressure is known to have a stronger relationship with age, height and weight than in adults.[55,56,57] This is reflected in the most recent guidelines on high blood pressure in children which take account of both specific age-year and height percentile.[58]

1. 3. 2 Type 2 Diabetes

The incidence of type 2 diabetes mellitus is increasing worldwide.[19] Type 2 diabetes results from the interaction between a genetic predisposition and behavioral and environmental risk factors.[59] Although the genetic basis of type 2 diabetes has yet to be identified, there is strong evidence that such modifiable risk factors as obesity and physical inactivity are the main nongenetic determinants of the disease.[60]

Until recently, most children with diabetes mellitus had type 1 diabetes, one of the most common[61] and increasingly prevalent[62] chronic disease in children. Increasingly, however, type 2 diabetes is being reported in children from the United States, Canada, Japan, Hong Kong, Australia, New Zealand, Libya and Bangladesh.[63]

The prevalence of type 2 diabetes in children ranges from 4.1 per 100 12-19 year olds in the US to 50.9 per 100 15-19 year olds Pima Indians of Arizona[64]. Between 8% and 45% of recently diagnosed cases of diabetes among children and adolescents in the United States is Type 2, and the magnitude of this disease may be underestimated. The prevalence of the disease is on the rise in North America, and its incidence almost doubled in Japan between 1976 - 80 and 1991 - 95 from 7.3 to 13.9 per 100 000 junior high school children[64]. These trends coincide with the rising prevalence of overweight and physical inactivity worldwide.[65]

Among US children the mean age at diagnosis of type 2 diabetes is between 12 and 14 years, corresponding with puberty.[66] The disease affects girls more than boys, predominantly people of non-European origin, and is associated with obesity, physical inactivity, a family history of type 2 diabetes, exposure to diabetes in utero, and signs of insulin resistance. In Tanzania, the problem seem to be low as diabetes was found in only 0.28% of adolescents aged 15 – 19 years.[31]

1. 3. 3 Hypercholesterolemia

Levels of serum cholesterol have been correlated with a person's subsequent risk of heart disease. Clinical studies have shown that serum cholesterol screening may reduce the risk of cardiovascular diseases in adults, and for high risk children and adolescents.[67]

Evidence that atherosclerosis begins early in life comes from both clinical and epidemiological data. The clinical data is provided by children with homozygous familial hypercholesterolemia. These children develop significant heart disease in the first decade of life and frequently die from myocardial infarction before the age of 20.[68] Furthermore, blood cholesterol levels measured at 22 years of age predict the risk for cardiovascular disease over the next 30 to 40 years[69] and data from the Framingham study show that cholesterol levels measured in young adult males and females predict cardiovascular disease mortality 30 years later.[70] In familial hypercholesterolemia there is a direct association between the duration and severity of the hypercholesterolemia and extravascular lipid deposition in tissues of these patients.[71]

Epidemilogical data support the relationship between childhood cholesterol levels and adult levels[72] and the relationship between hyperlipidemia in childhood and parental premature cardiovascular disease.[73]

Different policies have been put forward regarding cholesterol screening, defining who should be screened, when and how often.[74] In the United States, selective screening of cholesterol in children and adolescents is recommended by the National Cholesterol Education Program Expert Panel on Blood Cholesterol Levels in Children and Adolescents, the American Academy of Pediatrics, the National Center for Education in Maternal and Child Health's Bright Future guidelines, the American Medical Association Guidelines for Preventive Services (GAPS), and the American Academy of Family Physician. Screening with nonfasting cholesterol in all children and adolescents who have a parental history of hypercholesterolemia, and with fasting lipid profile in those with a family history of premature cardiovascular disease, is recommended by these organizations.

The US National Cholesterol Education Program, Expert Panel on Blood Cholesterol Levels in Children and

Adolescents[75] defines high total cholesterol levels in children as being ≥ 5.15 mmol/l regardless of family history of hypercholesterolemia or premature cardiovascular disease. The Panel also defines high LDL cholesterol as levels ≥ 3.35 mmol/l.

1. 3. 4 Obesity

Obesity is defined as the presence of excess adipose tissue. In normal individuals, the percentage of body tissue that is adipose varies by gender (greater in postpubertal females than males) and age (about 12% at birth, increasing to 25% at 5 months, then decreasing to 15% - 18% during puberty).[76] Clinical standards for defining obesity in children are not well established. In general, any child with weight for height above the 95th percentile for age and sex or who has significantly increased his weight for height percentile should be considered obese.[77]

Obesity is strongly linked to cardiovascular diseases and type 2 Diabetes Mellitus through the promotion of insulin resistance and other associated physiological abnormalities including dyslipidaemia, eleveted blood pressure, and increased left ventricular mass.[78,79,80,81] Overweight and insulin resistance have been linked to the early development of atheromata in young adults independent of other cardiovascular risk factors.[82]

Obesity is a health problem with both genetic and environmental causes.[83,84] Longitudinal studies of children followed into young adulthood suggest that overweight children may become overweight adults, particularly if obesity is present in adolescence.[85,86,87] There is also substantial evidence that obesity in childhood lays the metabolic groundwork for adult cardiovascular disease.[88] Studies of families and twins have clearly demonstrated a strong genetic component in the aetiology of obesity.[89,90,91] Just as persuasively, a recent study of secular trends in anthropometry has shown an increase in the prevalence of overweight and obesity in children and young adults over the last 10-20 years, indicating a potent interaction between environment and genetics.[92] This secular trend has been associated with worsening of cardiovascular risk.

1. 3. 5 Physical Inactivity

Recent studies have clearly shown that early signs of chronic disease and risk factors for chronic disease such as eleveted cholesterol and hypertension which would be considered normal in a middle-aged population, can be found in children.[93,94,95] Several studies have documented that the presence of chronic disease risk factors in children is associated with low aerobic fitness and low levels of physical activity.[96,97,98]

Physical inactivity has been shown to be significant predictor and cause of obesity in children, independent of nutritional habits.[99] In the US, children's obesity appears to be increasing, with sedentary activities such as television viewing having replaced recreational pursuits that involve more physical activity.[100] This becomes problematic since children with lowest physical/fitness levels, and highest percentage of body fatness are more likely to develop other risk factors for cardiovascular disease, including eleveated blood pressure and serum cholesterol levels.[101,102] It is encouraging to note that weight and blood pressure can be lowered in children when physical activity is an integral part of the treatment regimen.[103]

Unfortunately, many cardiovascular disease risk factors tend to ``track´´ over time.[104] In other words, if you have them as a child, you will likely keep them as an adult. For example, a follow-up of the Havard growth study of 1922-1935 showed that being overweight during adolescence was a greater predictor of chronic disease development (ie, cardiovascular disease, arthritis, etc) than being overweight as an adult.[105] Likewise, aerobic fitness and physical activity behaviors tend to track into adulthood. Dennison et al[92,106] found that very inactive young adults had the lowest aerobic fitness scores (as measured by the 600 yard run) when they were youngsters. In Finland, a longitudinal study found that children who were most sedentary showed the least favorable cardiovascular disease risk profile when they became young adults.[93,107]

In adults, the relationship between physical activity and fitness, and their combined influence on cardiovascular disease risk is clear.[108,109,110] In children, it is not known whether fitness or activity is the most important predictor for developing cardiovascular disease in adulthood.[111,112,113] Also, there is no concensus on whether regular physical activity will result in significant gains in aerobic fitness in children, particularly those who are prepubescent.[114] Significant associations between children's level of physical activity and their fitness have been reported[83,101,115], however the associations reported have been tenuous at best.[101,102,116] It is possible that large variability in growth curves make it impossible to correlate the fitness and activity variables. Lack of a strong association between fitness and activity in children may also be attributed in part, to methodological problems. Although a number of valid and objective tests of aerobic fitness have been developed, it is more difficult to quantitatively evaluate varying degrees of physical activity in youngsters.[98,103,104,117,118] In any event, in a review of cross-sectional studies designed to measure children's activity levels, Sallis reported that boys were approximately 23% more active than girls; moreover, boys' activity levels declined 2.7% per year, while girls' declined 7.4% per year thoughout adolescence.[98]

1. 3. 6 Smoking

In children, smoking adoption is closely related to smoking by parents, older siblings and close friends. Children perceive smoking as a way to appear mature, relax, and release anger, as well as to enhance their appeal to the opposite gender.[119]

At least 10 major prospective studies in Canada, Europe, Japan, The United Kingdom and the United States have found that all-cause mortality in smokers is approximately 1.7 times that of non-smokers. Most studies show a strong dose-response relation of cigarettes smoked to deaths from CVD. In addition, smoking increases the risk of peripheral arterial occlusive disease, and various forms of cancer (lung, larynx, oral, oesophagus, bladder, pancrease and others).[120,121] Studies in Swedish and Finnish twins have confirmed the risks of developing these diseases as a consequence of smoking.[122,123]

More than 90% of regular smokers begin their habit before they reach the age of 20.[119] A majority of Canadian children have tried smoking or are current smokers by age 13.[124] These facts raise the possibilitiy that young persons are ill-informed about the hazards of adopting cigarrette smoking. One study found that those children at greatest risk for smoking were the misformed, they also held different beliefs and attitudes than no-smokers. They greatly overestimated the prevalence of adult and peer smoking, underestimated the negative attitudes of their peer towards smoking, and felt they were less likely than others to contract a smoking-related illness.[125]

A British study found that those who eventually smoked reported that they believed teachers and parents would not mind if they took up smoking, but parental smoking attitudes, strictiness and beliefs about the effects of smoking on health did not predict subsequent smoking behaviour.[91]

In Bogalusa, half of children starting to smoke before age 12 years smoked their first cigarettes wih a family member or older friend. Black children showed a greater sibling and peer effect, while whites were more influenced by parents' smoking habits. Smoking habits were well established by age 14.[134,135]

There are several possibilities for the effects of smoking on CVD. These include direct atherogenic effect, disturbances of coagulation function, exacerbation of a tendency toward coronary artery spasm, chronic effect of nicotine, and acute effects of carbon monoxide ingestion.[126] Direct smoking increases sympathetic discharge and leads to increases in the adrenomedullary hormone epinephrine and the sympathetic neurotransmitter norepinephrine.[127] This neurohumoral response appears to result in the down regulation of β-adrenergic receptors in long term smokers.[128]

Exposure to smoke within confined spaces such as commercial Airplane cabins can represent a significant health hazard[129,130], leading to surgeon general of the USA to issue a report on the health consequence of such involuntary smoking.[131] Approximately 85% of involuntary smoking exposure is from sidestream smoke, that which emanates from the end of the cigarrette between puffs. The concentration of carbon monoxide is 2.5 times higher in sidestream smoke than in mainstream smoke inhaled and exhaled by the smoker. Sidestream smoke particles are smaller and more likely to penetrate deeper in distal alveoli.[132] The mechanisms of increased risk are probably similar to those who receive the smoke directly.[133]

1. 4 Predicting healthy behaviors in children

Health behaviors have been defined as ``any activity undertaken by a person believing himself to be healthy for the purpose of preventing disease or detecting it at an asymptomatic stage´´. The health behaviors in question include a variety of behaviours such as medical service usage (eg physician visits, vaccination, screening for different diseases, etc), compliance with medical regimens (eg dietary, diabetic, antihypertensive regimens, etc) and self-directed health behaviours (eg diet, exercise, breast or testicular examintaion, brushing and flossing teeth, smoking, alcohol consumption, contraceptive use, etc).

Demographic variables show reliable associations with the perfomance of health behaviours. For example, age appears to show a curvilinear relationship with many health behaviours, with high incidences of many health-risking behaviours such as smoking in young adults and much lower incidences in young children and older adults[122]. Health behaviours also varies by gender, with females being generally less likely to smoke, consume large amounts of alcohol and engage in regular excercise, but more likely to monitor their diet, take vitamins and engage in dental care.

Differences in socioeconomic status and ethnic status are also apparent for behaviours such as diet, excercise, alcohol consumption and smoking.[122] Generally, younger, wealthier, better educated individuals under low levels of stress with high levels of social support are more likely to practice health-enhancing behaviours. On the other hand, higher levels of stress and/or fewer resources are associated with health-compromising behaviours such as smoking and alcohol abuse.[125]

In children, social factors such as parental and teachers' models, seem to be important in instilling health behaviours early in life. Peer influences are also important, for example in the initiation of smoking.[122] Values of a culture in a given society also appear to be influential, for instance in determining the number of girls engaging in exercise behaviours in paticular culture. Emotional factors also play an important role in the practice of some health habits in children. For example, over eating is linked to stress in some obese children. Self-esteem also appears to be an important influence in the practice of health behaviours by some children.

Finally, in children, cognitive factors also determine whether or not the child will practice health behaviours. For example, knowledge about behaviour-health links (or risk awareness) is an essential factor in an informed choice concerning a healthy lifestyle. A good example is the reduction of smoking over the past 20 years in the Western world which can largely be attributed to a growing awareness of the serious health risks posed by tobacco use brought about by widespread publicity in the mass media.

A variety of other cognitive variables have been studied in children. These factors include perception of health risk, potential efficacy of behaviours in influencing this risk, perceived social pressures to perform the behaviour and control over performance of the behaviour. For instance, the belief that a particular health behaviour is beneficial and can help stave off a particular illness may contribute to the practice of the behaviour.

2.0 RATIONALE

The proposed mode of preventing cardiovascular diseases in a developing country like Tanzania is Primordial prevention.[19] There has been no study in Tanzania which looked on the prevalence of the cardiovascular risk factors in children below 15 years. This group of the population is thought to be crucial when the aim is to target Primordial prevention since we have enough evidence to suggest that the process of atherogenesis begins early in childhood[33] and that children with cardiovascular risk factors tend to ``track´´ them into adulthood.[33]

The fact that behavioral lifestyles are forming in childhood increases the importance of this group of the population as a target for primordial prevention, as it is easier to instill healthy lifestyles at this age.[19]

Since cardiovascular diseases were once considered as diseases of the developed world, it is thought that there is generally lack of awareness and knowledge of cardiovascular diseases and their risk factors among adults and children in Tanzania.

It is against the above reasons that this study was conducted.

3. 0 OBJECTIVES

3. 1 Broad objective

To determine the prevalence of the conventional cardiovascular risk factors and to assess the level of awareness and knowledge of cardiovascular diseases among primary school children aged 6 - 15 years in Urban Dar es Salaam and Rural Morogoro.

3. 2 Specific objectives

1. To determine the prevalence of hypertension among primary school children aged 6-15 years in Urban Dar es Salaam and Rural Morogoro.
2. To determine the prevalence of obesity among primary school children aged 6-15 years in Urban Dar es Salaam and Rural Morogoro.
3. To determine the prevalence of cigarette smoking among primary school children aged 6-15 years in Urban Dar es Salaam and Rural Morogoro.
4. To determine the prevalence of physical inactivity among primary school children aged 6 – 15 years in Urban Dar es Salaam and Rural Morogoro.

5. To determine the prevalence of dyslipidaemia among primary school children aged 6 – 15 years in Urban Dar es Salaam and Rural Morogoro.
6. To determine the prevalence of type 2 diabetes mellitus among primary school children aged 6 – 15 years in Urban Dar es Salaam and Rural Morogoro.
7. To assess the level of awareness and knowledge of cardiovascular diseases among primary school children aged 6 – 15 years in Urban Dar es Salaam and Rural Morogoro.

4. 0 METHODOLOGY

4. 1 Study design: Cross sectional survey.

4. 2 Setting and Subjects:

The study was conducted among healthy primary school going children aged 6 to 15 years in Urban Dar es Salaam and Rural Morogoro from Dec 2000 to January 2001. Dar es Salaam, the capital city of Tanzania, is inhabitated by about 3million people most of whom from different regions within the country. The city has a total of 199 primary schools distributed in down town and some in the outskirts of the city. Bunge and Academic Primary schools are schools with similarities as both of them are situated in less crowded areas (Low Density Areas) within the city of Dar es Salaam and the children that go to these schools are mainly from affluent families with a better socio-economic background. Academic is a private school and Bunge was once a private school used mostly by children of top governmental officials including members of parliament.

Mbagala Kuu and Miburani primary schools are situated in heavily crowded areas (High Density Areas) in the outskirts of Dar es Salaam. The children that go to these schools are from families with a low income background, mostly self employed petty business traders or peasants. Few are employed in Industries or government offices.

Lungo and Manyenga are typical Rural schools from Morogoro Rural District. The inhabitants of this district depend on agriculture as their main source of income. The main food crops grown include maize, cassava, sweet potatoes, etc, while sugar cane is the main cash crop.

4. 3 Sample size:

Using a sample size formula for prevalence studies, 464 subjects were estimated after substituting 5.1% as the population prevalence (for hypertension) and taking the margin of error to be 5%. The number was rounded up to be 500 subjects to account for drop outs.

4. 4 Sampling method:

A multi-staged sampling technique was employed. The list of all primary schools in Urban Dar es Salaam and Rural Morogoro was obtained from the office of the Director of Primary Education in the Ministry of Education and Culture.

Using tables of random numbers, two schools were selected from the following school settings;

1. Urban Low Density (Academic and Bunge)
2. Urban High Density (Miburani and Mbagala Kuu)
3. Rural (Lungo and Manyenga)

The study population was then divided into strata according to age and sex.

A samplig fraction (one out of ten) was then used to select the 508 subjects for the study. The children were all asymptomatic at screening.

4. 5 Data Collection:

At least a minimum of four days were used to collect data from each of the six schools.

Day 1:

Explanation of the study aims and distribution of consent forms was done. Forms were sent to parents/guardians through their children for consenting.

Day 2:

Children whose parents/guardians consented were assisted to fill a questionnaire in which basic background information was obtained and questions regarding awareness and knowledge of cardiovascular diseases were asked.

Day 3:

Blood Pressure and anthropometric measurements were taken from morning to mid-day.

Day 4:

Blood samples were collected.

Blood Pressure measurements:

BP was recorded with a standard mercury sphygmomanometer, using a 20 cm x 9.5 cm cuff for children 10 years or less, and a 22.5 cm x 15 cm for children older than 10 years.The first and fifth Korotkoff's sounds were taken for systolic and diastolic pressures respectively.

It was recorded in a familiar, spacious room with subjects sitting comfortably and the right arm rested at the heart level.

Measurements were taken in the morning and during class hours. Children were comming in turns from class sessions in a calm situation. A 5 minute rest was then taken before the measurements were done. An average of two readings was taken as the blood pressure of the individual.

Body weight:

Body weight was measured using a weighing scale (Momert, Hungary) with subjects without shoes or heavy clothing like jackets. It was recorded to the nearest 0.5 kg.

Height:

Height was measured using a scale made over the wall. It was taken with subjects wearing no shoes or caps and recorded to the nearest decimal point in centimetres.

Body Mass Index (BMI):

After obtaining weight in kilograms and height in metres, the BMI was calculated from the following formula: weight in kg/height in $metre^2$. It was recorded in $kg/metre^2$.

Blood Samples:

Blood samples were collected from the antecubital vein, using a 5cc syringe. This was done before the children had their school snacks/meals but without an overnight fast. Whole blood glucose level was determined on the spot by a Glucometer-4 (Amron-UK).

The rest of the samples were kept in empty sterile bottles, serum separated and stored at –20°C, thereafter sent to Bergen, Norway for lipid analyses.

4. 6. 0 Definition of terms

4. 6. 1 Systolic hypertension:

Systolic blood pressure greater than or equal to the 95^{th} percentile for age, sex and height of the child.

4. 6. 2 Diastolic hypertension:

Diastolic blood pressure greater than or equal to the 95^{th} percentile for age, sex and height of the child.

4. 6. 3 Obesity:

Weight for height (BMI) measurements that are equal or above the 95^{th} percentile for age and sex of the child.

4. 6. 4 *Awareness of cardiovascular diseases* (Hypertension, Diabetes Mellitus, Stroke or Heart Attack):

defined as ``yes´´ if the respondent has ever heard of such a disease

``no´´ if the respondent has never heard of such a disease

4. 6. 5 *Knowledge of cardiovascular diseases* (Hypertension, Diabetes Mellitus, Stroke, Heart Attack)

Defined as ``yes´´ if the respondent was able to mention two or more of the following;

a) One or more symptom of the disease

b) One or more behaviour that predispose one to the disease

c) One or more preventive behaviour for the disease

4. 7 Data Analysis

Data was entered and analysed using the *Statistical Package for Social Sciences* (SPSS/PC+ Version 10) for the IBM/AT microcomputer. *Chi square* test was used for categorical variables and the differences between two group means was analysed using the *student's t-test.* For groups more than two, one way *Analysis of Variance* (ANOVA) was used to compare group means.

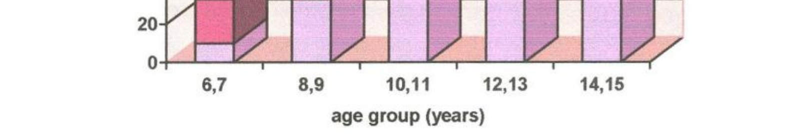
Differences were considered significant if the *p*-value was less than 0.05.

5. 0 RESULTS

5. 1 Characteristics of the study population

To determine the prevalence of cardiovascular risk factors among children, a total of 508 primary school children from six schools were involved. (Four schools in Urban Dar es Salaam and two schools in Rural Morogoro). Males were 228 (44.9%) and females were 280 (55.1%), giving a ratio of males to females of 0.8:1. Figure 1 shows the age and sex distribution of the study population.

Fig. 1. Distribution of the study population by age and gender



Sixty percent of the study population was from Urban Dar es Salaam, representing two different school settings: Urban Low

Density (ULD) schools (Academic and Bunge) and Urban High Density (UHD) schools (Miburani and Mbagala Kuu). Fourty percent came from two schools in Rural Morogoro (Lungo and Manyenga). Females outnumbered males in all school settings, as shown in table 1.

Table 1: Distribution of the study population by school setting and gender.

	Males	*Females*	*Total*
School setting	*n (%)*	*n (%)*	*n (%)*
ULD	61 (43.0)	81 (57.0)	142 (27.9)
UHD	69 (41.6)	97 (58.4)	166 (32.7)
Rural	98 (49.0)	102 (51.8)	200 (39.4)
Total	228 (44.9)	280 (55.1)	508 (100)

ULD = Urban Low Density, UHD = Urban High Density

The mean (SD) age of the total study population was 11.5 (2.4) years. The mean (SD) age for males was 11.89 (2.46) years while that for females was 11.28 (2.33) years. Two hundred and seventy five (54.13%) subjects were between 12 and 15 years. Table 2 shows demographic characteristics of the study population by gender.

Table 2. Demographic characteristics of the study population by gender.

Characteristic	*Males* *n (%)*	*Females* *n (%)*
Total series	228 (44.9)	280 (55.1)
Age group (years)		
6 – 11	93 (40.8)	140 (50.0)
12 - 15	135 (59.2)	140 (50.0)
Class		
1 – 4	110 (48.2)	140 (50.0)
5 – 7	118 (51.8)	140 (50.0)
	Mean (SD)	*mean (SD)*
Age (years)	11.89 (2.46)	11.28 (2.33)

Subjects from Dar es Salaam Urban Low Density schools had statistically significant higher mean (SD) levels for both systolic [112.94 (9.65) mmHg] and diastolic blood pressure [69.20 (8.70) mmHg], $p < 0.001$ compared to those from Dar es Salaam Urban High Density schools and Rural Morogoro schools. In addition subjects from Rural Morogoro schools were taller (142.91 ± 11.43 cm) as compared to those from Urban Dar es Salaam, ($p = 0.01$).

Subjects from ULD schools had higher mean (SD) levels of body weight [36.48 (12.5) kg] and BMI [18.38 (4.93) kg/m^2] compared to those from UHD [33.84 (9.23) kg and 16.64 (4.92) kg/m^2 respectively] and Rural schools [33.81 (8.29) kg and 16.50 (2.04) kg/m² respectively). However these differences were statistically non significant. (Table 3).

Table 3. Comparison of anthropometric and blood pressure measurements between the school settings

	ULD *mean (SD)*	*UHD* *mean (SD)*	*Rural* *mean (SD)*	*p value*
SBP (mmHg)	112.94 (9.65)	109.81 (9.34)	103.60 (12.95)	< 0.001
DBP (mmHg)	69.20 (8.70)	65.99 (6.97)	64.69 (8.92)	< 0.001
Height (cm)	139.77 (12.05)	141.02 (10.79)	142.91 (11.43)	0.01
Weight (kg)	36.48 (12.75)	33.84 (9.23)	33.81 (8.29)	0.023
BMI (kg/m²)	18.38 (4.93)	16.64 (4.92)	16.50 (2.04)	<0.001

ULD = Urban Low Density, UHD = Urban High Density, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, BMI = Body Mass Index
p, significance of difference between groups

5. 2 Cardiovascular risk factors:

5. 2. 1 Hypertension

The blood pressure (both systolic and diastolic) was found to rise with age. Females had slightly higher mean values for both systolic (108.47mmHg versus 107.96mmHg) and diastolic (66.83mmHg versus 65.81mmHg) blood pressure. The difference was more seen in the younger subjects and it normalized as the age increased. Figure 2 and Figure 3 show age and sex - blood pressure curves for systolic and diastolic respectively.

Figure 2. Systolic Blood Pressure curves for males and females by age.

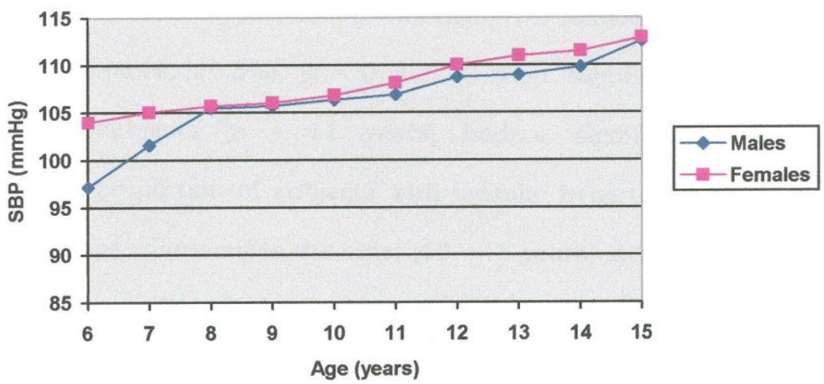
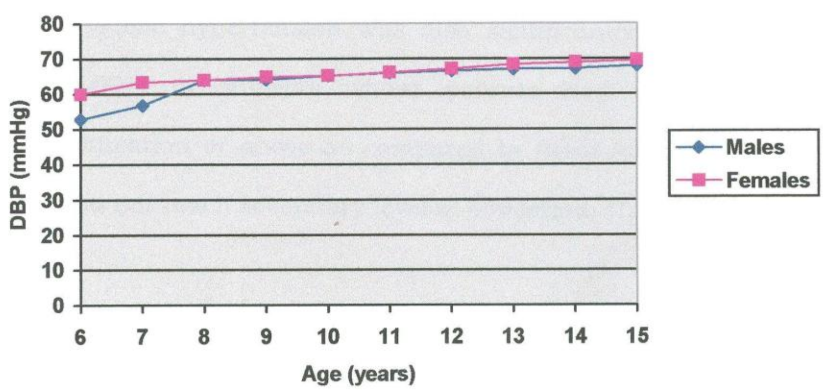


Figure 3. Diastolic Blood Pressure curves for males and females by age.



5. 2. 1. 1. Systolic Hypertension

A total of 58 (11.4%) subjects out of the total population had their systolic blood pressure greater than the 95th percentile for age, sex and height hence defined as having systolic hypertension.

Systolic hypertension was higher in females (12.1%) than males (10.5%), p = 0.568793 (non significant). Younger subjects (6 – 11 years) had a significantly higher proportion of subjects with systolic hypertension (16.7%) as compared to the older (12 – 15 years) subjects (6.9%), $p < 0.001$. Subjects from ULD schools had a significantly higher prevalence (29.6%) of systolic hypertension compared to students from UHD (4.8%) and Rural (4.0%) schools, who had almost similar proportions of subjects with systolic hypertension, $p < 0.001$.

Systolic hypertension was also significantly higher ($p < 0.001$) in subjects whose parents had a secondary education or above as compared to those whose parents did not reach secondary level of education, (Table 4).

Table 4. Prevalence of Systolic hypertension by demographic characteristics.

Characteristic	*Systolic Hypertension (N = 58) n (%)*
Gender	
Males	24 (10.5)[NS]
Females	34 (12.1)
Age	
6 – 11	39 (16.7)**
12 – 15	19 (6.9)
School category	
ULD	42 (29.6)**
UHD	8 (4.8)
Rural	8 (4.0)
¶Father's education	
Secondary+	40 (17.6)**
Below secondary	8 (5.3)
¶Mother's education	
Secondary+	34 (19.5)**
Below secondary	13 (6.7)

ULD = Urban Low Density, UHD = Urban High Density
¶ Not all subjects knew their parents' level of education
** $p < 0.001$, NS Not Significant

5. 2. 1. 2 Diastolic Hypertension

Fourty one subjects (8.1%) out of the total population were found to have diastolic hypertension. Females had an insignificant higher proportion of subjects with diastolic hypertension (8.9%) compared to males (7.0%), p = 0.431579. Those subjects aged 6 – 11 years had a higher proportion (9.4%) of diastolic hypertension compared to those aged 12 – 15 years (6.9%). The difference was not statistically significant, $p = 0.296313$.

Diastolic hypertension was significantly higher in subjects from ULD schools (20.4%) compared to those from UHD (3.0%) and Rural (3.5%) schools, $p < 0.001$. Diastolic hypertension was also significantly higher in subjects whose parents had a level of education of secondary or above as shown in table 5.

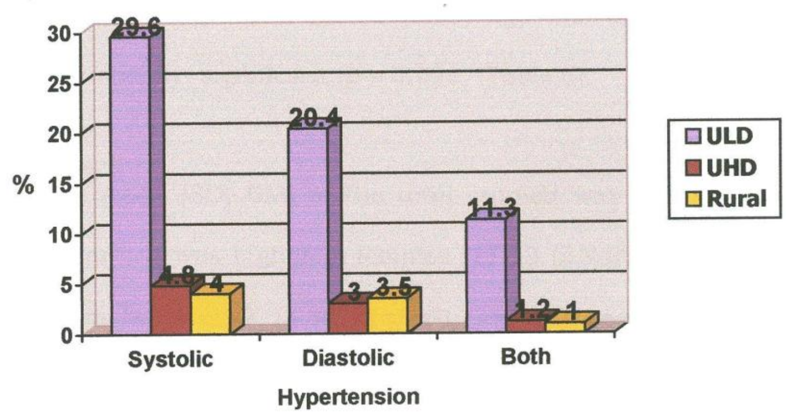
Twenty subjects (3.9%) out of the total study population were found to have both systolic and diastolic hypertension. Of the twenty subjects, 16 (11.3%) were from ULD schools and only 4 (two from each) subjects were from the UHD (1.2%) and Rural (1.0%) schools. This difference was statistically significant, $p < 0.001$. Figure 4 summarises the differences in the prevalence of Systolic, Diastolic and both (systolic and diastolic) hypertension in the three school settings.

Table 5. Prevalence of Diastolic hypertension by demographic characteristics.

Characteristic	*Diastolic Hypertension (N = 41) n (%)*
Gender	
Males	16 (7.0)[NS]
Females	25 (8.9)
Age	
6 – 11	22 (9.4)[NS]
12 – 15	19 (6.9)
School category	
ULD	29 (20.4)***
UHD	5 (3.0)
Rural	7 (3.5)
¶Father's education	
Secondary+	25 (11.0)*
Below secondary	4 (2.6)
¶Mother's education	
Secondary+	20 (11.5)*
Below secondary	6 (3.1)

ULD = Urban Low Density, UHD = Urban High Density
¶ Not all subjects knew their parents' level of education
* $p < 0.05$, ** $p < 0.001$, NS Not Significat

Figure 4. Prevalence of Systolic, Diastolic and both (Systolic and Diastolic) hypertension in the three school settings.



5. 2. 2. Obesity

The mean (SD) height of the total population was 141.41 (11.46) cm. Overall, males were taller [141.96 (11.37) cm] than females [140.97 (11.53) cm] though this difference was not statistically significant, p = 0.333. Furthermore, subjects from Rural schools were taller [142.91 (12.05) cm], followed by subjects from UHD [141.02 (10.79) cm] and last from ULD [139.77 (12.05) cm]. The differences in height was statistically significant, p = 0.010. (Table 6).

The mean (SD) weight of the total population was 34.67 (11.46) kg. There was almost similar mean (SD) values for males [34.43 (10.65) kg] and females [34.68 (9.58) kg] , p = 0.783. Subjects from ULD had a higher mean (SD) weight [36.48 (12.75) kg] compared to those from UHD [33.84 (9.23)kg] and Rural [33.81 (8.29) kg]. The difference was statistically significant, $p = 0.023$. (Table 6).

The mean (SD) BMI of the total studied was 17.07 (3.35) kg/m^2 . It was higher in females [17.13 (2.94) kg/m^2] than in males [16.99 (3.80) kg/m^2], the difference was not statistically significant, $p = 0.910$. Subjects from ULD had the highest mean BMI [18.38 (2.04) kg/m^2], followed by UHD [16.64 (4.93) kg/m^2] then Rural subjects [16.50 (2.04) kg/m^2], $p < 0.001$, (p, significance of difference between groups, Table 6.

Twenty seven subjects (5.3%) out of the studied population were found to have obesity. Females had a higher proportion of obese subjects (5.7%) compared to males (4.8%), $p = 0.6566$, non significant. Obesity was significantly more prevalent in the younger subjects ie 6 – 11 years olds (7.7%) compared to the older subjects (3.3%), $p = 0.019$.

Table 6. Means (SD) for Height, Weight and BMI by demographic characteristics

Characteristic	*Height (cm) mean (SD)*	*Weight (kg) mean (SD)*	*BMI (kg/m²) mean (SD)*
Total series	141.41 (11.46)	34.67 (11.46)	17.07 (3.35)
Gender			
Males	141.96 (11.37)	34.43 (10.65)	16.99 (3.80)
Females	140.97 (11.53)	34.68 (9.58)	17.13 (2.94)
Age group (years)			
6 – 11	133.15 (8.55)**	29.98 (9.71)**	16.68 (4.00)*
12 – 15	148.42 (8.58)	38.65 (8.39)	17.40 (2.64)
School category			
ULD	139.77 (12.05)*	36.48 (12.75)*	18.38 (4.93)**
UHD	141.02 (10.79)	33.84 (9.23)	16.64 (4.93)
Rural	142.91 (11.43)	33.81 (8.29)	16.50 (2.04)
Father's education			
Secondary+	141.46 (11.42)	36.32 (11.33)	17.88 (4.13)**
Below secondary	143.32 (11.31)	34.57 (8.41)	16.59 (2.21)
Mother's education			
Secondary+	140.03 (11.59)**	35.00 (10.12)	17.57 (3.35)
Below secondary	144.08 (10.84)	35.96 (10.09)	17.09 (3.53)

ULD = Urban Low Density, UHD = Urban High Density
* $p < 0.05$, ** $p < 0.001$

Investigation on obesity from the different school settings revealed that subjects from ULD schools were more likely to be obese (16.9%) compared to UHD (1.8%) and the Rural schools subjects were less likely (0.0%). This difference was statistically significant, $p < 0.001$.

Subjects whose parents had an education level of secondary or above had an increased chance of being obese than those whose parents had lower levels of education. These results are shown in table 7.

Table 7. Prevalence of obesity by demographic characteristics

Characteristic	*Obese (N = 27) n (%)*	*Not Obese (N = 481) n (%)*	*p value*
Total series	27 (5.3)	481 (94.7)	
Gender			
Males	11 (4.8)	217 (95.2)	0.6566
Females	16 (5.7)	264 (94.3)	
Age group			
6 – 11	18 (7.7)	215 (92.3)	0.0193
12 – 15	9 (3.3)	266 (96.7)	
School setting			
ULD	24 (16.9)	118 (83.1)	< 0.001
UHD	3 (1.8)	163 (98.2)	
Rural	0 (0.0)	200 (0.0)	
¶Father's education			
Secondary+	23 (10.1)	204 (89.9)	< 0.001
Below secondary	2 (1.3)	150 (98.7)	
¶Mother's education			
Secondary+	18 (10.3)	156 (89.7)	< 0.001
Below secondary	4 (2.1)	191 (97.9)	

ULD = Urban Low Density, UHD = Urban High Density

¶ Not all subjects knew their parents' level of education

5. 2. 3 Cigarrette smoking

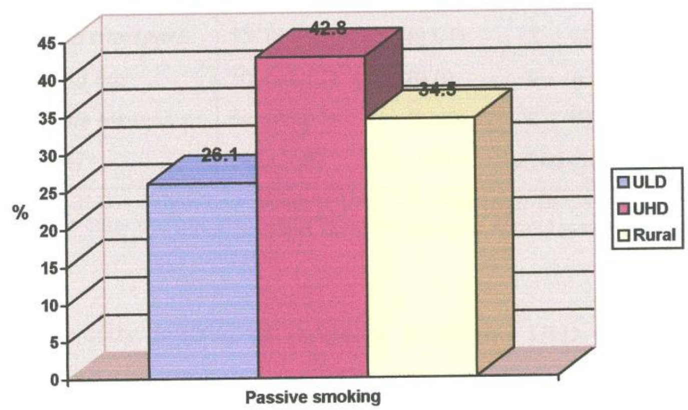
Twenty six (5.1%) subjects out of the total population admitted to have smoked at least once till the time of this study. Only six (1.2%) subjects out of the total study population were smoking cigarrette on a regular basis, (2 from UHD schools and 4 from Rural schools).

Thirty five percent of the total study population reported they were living in the same house with a smoking person ie having passive smoking. Among the reported people smoking, 99% were males (Fathers, Uncles, Grandfathers).

Subjects from the UHD schools had the highest proportion of passive smokers (42.8%) in comparison to the Rural schools subjects (34.5%). The ULD schools subjects had the lowest proportion of individuals having passive smoking (26.1%). This difference between the groups was statistically significant, $p = 0.009$.

On attitude regarding smoking 97.2% were aware that smoking was bad for health. Three (0.6%) subjects thought smoking has no harm on health on the contrary it freshenes and relaxes the mind and body. Eleven (2.2%) subjects were undecissive on whether smoking was bad or good for health. Figure 5 shows the proportion of subjects under passive smoking in the three school categories.

Figure 5. Prevalence of Passive Smoking by school setting



5. 2. 4 Physical activity

The level of physical activity was assessed using two criteria: (1) means of coming to school, (2) leisure time activities. In addition the study subjects' attitudes in sports activities was assessed.

About two thirds (68.9%) of the study subjects were going to school on foot, 29.1% were using motor vehicles and 2% were using bicycles. Table 8 shows the means of going to school by school category.

Table 8. Means of going to school by school setting

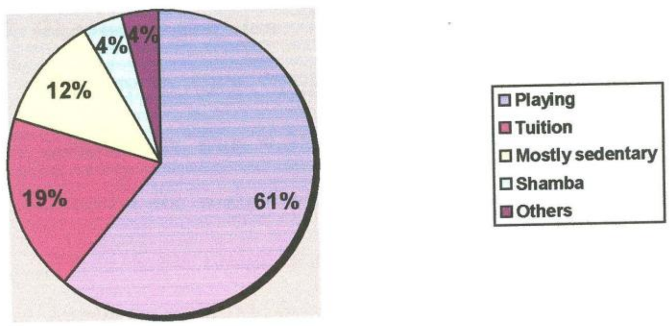
	ULD n (%)	*UHD* n (%)	*Rural* n (%)
On foot	13 (9.15)	150 (90.36)	187 (93.5)
By bicycle	0 (0.0)	0 (0.0)	10 (5.0)
Public transport	35 (24.65)	15 (9.04)	3 (1.5)
School bus	30 (21.13)	0 (0.0)	0 (0.0)
Private transport	64 (45.07)	1 (0.60)	0 (0.0)
****Total***	142 (100)	166 (100)	200 (100)

ULD = Urban Low Density, UHD = Urban High Density
* Only the usual/regular mean of going to school was considered

Majority (>90%) of subjects from the ULD schools were using motor vehicles as means of transport for going to school in either school bus or private owned motor vehicle. On the contrary, the UHD and Rural schools, majority of subjects were going to school on foot.

The following responses were given when asked about the subjects' leisure time physical activities. (Figure 6)

Figure 6. Leisure time physical activities mentioned.



More than half (61.0%) of the study subjects mentioned playing football or other sports as their main leisure time activity. Nineteen percent of the subjects mentioned going to tuition after their school time. Twelve percent of the study population were mostly sedentary (watching TV or just staying at home) during their leisure time. Four percent of the subjects engaged themselves either in farm or garden works post school.

On the response on their attitude towards sports, majority (87.6%) of the study population responded by saying that sports was good for health. Fifty three (10.6%) subjects said sports are bad for health and 10 (2.0%) subjects did not know whether sports were good or bad for health.

5. 2. 5 Glycaemic profiles

The mean (SD) blood glucose level of the total study population was 4.88 (0.74) mmol/l. It was 4.84 (0.63) mmol/l for males while that for females was 4.98 (0.64) mmol/l, an insignificant difference, $p = 0.1130$. Younger subjects (6 – 11 years) had a significantly higher mean blood glucose level 4.99 (0.55) mmol/l compared to the older subjects 4.78 (0.69) mmol/l, $p < 0.001$.

The mean (SD) blood glucose level was also significantly higher in subjects from ULD, 5.07 (0.47) mmol/l followed by the UHD school subjects 4.87 (0.54) mmol/l. The rural schools had the least mean glucose level, 4.73 (0.81) mmol/l. This difference between the groups was statistically significant, $p < 0.001$.

Equally, subjects whose fathers had secondary or above level of education had significantly higher mean blood glucose level, 4.93 (0.68) mmol/l as compared to those whose fathers had below secondary level of education 4.81

(0.52) mmol/l, p = 0.045. There was no difference with mothers' level of education. Table 9.

None of the subjects had blood glucose levels that met the WHO's definition for diabetes.

5. 2. 6 Lipid profiles

The mean (SD) total serum cholesterol, LDL-C, HDL-C and triglycerides for the total study population was found to be 3.56 (0.88) mmol/l, 1.99 (0.66) mmol/l, 0.88 (0.38) mmol/l and 0.98 (0.42) mmol/l respectively. There were no significant differences of mean lipid levels as far as gender is concerned although females had a slight higher mean values for total cholesterol (3.61 versus 3.52 mmol/l) and for LDL-C levels (2.03 versus 1.96 mmol/l), $p = 0.2517$.

Younger subjects (6 – 11 years) had significantly higher mean values for total cholesterol (3.79 mmol/ versus 3.39 mmol/l), $p < 0.001$, LDL-C (2.10 mmol/l versus 1.91 mmol/l), $p < 0.001$ and HDL-C (0.95 mmol/l versus 0.82 mmol/l), $p < 0.001$ as compared to the older subjects (12 –

15 years). This difference was not seen on the triglycerides levels.

Table 9. Means (SD) of blood glucose levels by demographic characteristics.

Characteristic	*Blood sugar level (mmol/l) (N = 508) mean (SD)*
Total series	4.88 (0.74)
Gender	
Males	4.84 (0.63)
Females	4.93 (0.64)
Age group (years)	
6 – 11	4.99 (0.55)**
12 – 15	4.78 (0.69)
School category	
ULD	5.07 (0.47)**
UHD	4.84 (0.54)
Rural	4.73 (0.81)
Father's education	
Secondary+	4.93 (0.68)*
Below secondary	4.81 (0.52)
Mother's education	
Secondary+	4.90 (0.69)
Below secondary	4.89 (0.56)

* $p < 0.05$, ** $p < 0.001$
ULD = Urban Low Density, UHD = Urban High Density

Subjects from ULD schools had significantly higher mean levels for total cholesterol (4.19 mmol/l), $p < 0.001$ and LDL-C (2.26 mmol/l), $p < 0.001$ compared to the subjets from UHD (3.74 mmol/l and 1.96 mmol/l respectively) and

Rural schools (2.98 mmol/l and 1.83 mmol/l respectively). Furthermore, subjects whose parents had level of education of secondary or above had significantly higher mean levels of total cholesterol, LDL-C, and HDL-C. This difference was not notted with triglycerides levels. Results are shown in table 10.

Twenty one (4.1%) out of the 483 subjects (subjects with blood samples) were found to have their total cholesterol levels above 5.15 mmol/l, ie elevated serum total cholesterol. Hypercholestrolemia was slightly higher in females (5.2%) than males (3.2%), $p = 0.2918$.

Younger subjects had significantly higher proportion of individuals with hypercholesterolemia (6.8%) compared to the older subjects (2.3%), $p = 0.014$. Hypercholesterolemia was also significantly higher in subjects from ULD sujects (12.1%), than the UHD subjects (3.1%). None (0.0%) of the subjects from Rural schools had total cholesterol levels greater than 5.15 mmol/l, $p < 0.001$.

Ten subjects (2%) out of the total study population were found to have elevated LDL-C (defined as levels >3.35mmol/l), the prevalence was 2.6% in females and 1.4% in males, an insignificat difference, p = 0.5244. With regard to age, the younger subjects had a slight higher prevalence (3.2%) compared to the older subjects (1.1%), equally this difference was not statistically significant, p = 0.2115.

Table 10. Lipid profiles by demographic characteristics.

Mean (SD) values

Characteristic	*Total-C (mmol/l) mean (SD)*	*LDL-C (mmol/l) mean (SD)*	*HDL-C (mmol/l) mean (SD)*	*Triglyc (mmol/l) Mean (SD)*
Total series	3.56 (0.88)	1.99 (0.66)	0.88 (0.38)	0.98 (0.42)
Gender				
Males	3.52 (0.84)	1.96 (0.65)	0.88 (0.39)	0.98 (0.41)
Females	3.61 (0.91)	2.03 (0.66)	0.88 (0.38)	0.98 (0.42)
Age group (years)				
6 – 11	3.79 (0.90)**	2.10 (0.69)*	0.95 (0.38)**	0.99 (0.47)
12 – 15	3.39 (0.82)	1.91 (0.62)	0.82 (0.37)	0.98 (0.36)
School category				
ULD	4.19 (0.82)**	2.26 (0.63)**	1.03 (0.36)**	1.02 (0.45)*
UHD	3.74 (0.73)	1.96 (0.59)	1.05 (0.30)	0.91 (0.42)
Rural	2.98 (0.64)	1.83 (0.68)	0.63 (0.33)	1.02 (0.38)
Father's education				
Secondary+	3.90 (0.85)**	2.15 (0.63)**	0.97 (0.38)**	0.99 (0.44)
Below secondary	3.21 (0.81)	1.81 (0.61)	0.75 (0.37)	0.97 (0.36)
Mother's education				
Secondary+	3.99 (0.80)**	2.19 (0.63)**	1.00 (0.35)**	1.00 (0.45)
Below secondary	3.27 (0.83)	1.84 (0.60)	0.78 (0.38)	0.99 (0.40)

* $p < 0.05$, ** $p < 0.001$, ULD = Urban Low Density, UHD = Urban High Density

Subjects from the ULD schools had a significantly higher proportion of individuals with high LDL-C levels (5.3%) as opposed to UHD subjects (0.6%) and the Rural subjects (1.1%), $p = 0.009$.

Table 11. Means (SD) and prevalences of cardiovascular risk Factors by school setting

CV risk factor	*ULD N = 142*	*UHD N = 166*	*Rural N = 200*	*p value*
Blood pressure (mmHg)				
Systolic	112.94 (9.65)	109.81 (9.34)	103.60 (12.95)	< 0.001
Diastolic	69.20 (8.70)	65.99 (6.97)	64.69 (8.92)	< 0.001
Syst HT (%)	29.6	4.8	4.0	< 0.001
Diast HT (%)	20.4	3.0	3.5	< 0.001
Both (Sys+Dias) (%)	11.3	1.2	1.0	< 0.001
Serum cholesterol (mmol/l)				
Total	4.19 (0.82)	3.74 (0.73)	2.98 (0.64)	< 0.001
LDL-C	2.26 (0.63)	1.96 (0.59)	1.83 (0.68)	< 0.001
HDL-C	1.03 (0.36)	1.05 (0.30)	0.63 (0.33)	< 0.001
Tryglycerides	1.02 (0.45)	0.91 (0.42)	1.02 (0.38)	0.0198
Total-C > 5.15 (%)	12.1	3.1	0.0	< 0.001
LDL-C > 3.15 (%)	5.3	0.6	1.1	0.0035
Blood glucose (mmol/l)	5.07 (0.47)	4.84 (0.54)	4.73 (0.81)	< 0.001
BMI (kg/m^2)	18.38 (4.93)	16.64 (4.92)	16.50 (2.04)	< 0.001
Obesity (%)	16.9	1.8	0.0	< 0.001
Smoking				
Active (%)	0.0	1.2	2.0	NS
Passive (%)	26.1	42.8	34.5	0.0089

ULD = Urban Low Density, UHD = Urban High Density, NS = Not Significant
p, significance of difference between groups

Table 11 summarises the means and prevalences of cardiovascular risk factors between the three school settings. Of note is the higher prevalences of the risk factors among the subjects from ULD schools as compared to those from the other schools. It can also be seen that subjects from UHD schools had a lot of similarities to those from the Rural schools as far as risk factors for cardiovascular diseases is concerned.

5. 3. 0 Awareness and Knowledge of Cardiovascular diseases.

5. 3. 1 Hypertension

A total of 499 subjects responded to the question as to whether they have ever heard of a disease called hypertension. 46.5% said they have heard and that they were aware of hypertension, females were more aware than males (50.9% versus 41.9%), $p = 0.028$.

There was a significant difference in awareness of hypertension as far as age was concerned, being 56.6% in the age group 12 –15 years, as compared to 34.9% in subjects aged 6 – 11 years, $p < 0.001$. There was no significant difference of awareness between schools in different settings.

There was a significant difference between those subjects attending lower classes (34.7%) and higher classes (58.2%) in regard to awareness of hypertension, $p < 0.001$.

Offsprings whose fathers had a level of education of secondary or above were more likely to be aware of hypertension (56.2%) as compared to those whose fathers' levels of education were below secondary (45.7%), $p = 0.045$. When considering mothers' level of education, there was no significant difference in the level of awareness between the two groups, 55.2% versus 49.2%, $p = 0.2546$.

Ninety six (41.4%) out of the 232 subjects who were aware of hypertension as a disease had knowledge of hypertension .
A higher proportion of females (42.9%) had knowledge of hypertension as compared to their male counterparts (39.1%). This difference was however not statistically significant, p = 0.5728.

There was no significant difference in the knowledge of hypertension as far as age was concerned, although the level of knowledge was slightly higher in the older subjects (43.0%) than the younger ones (38.3%), $p = 0.4814$.

Knowledge of hypertension was significantly higher in subjects from the ULD schools (58.9%), followed by the UHD schools (50.6%) and the rural schools subjects had the least proportion of subjects that had knowledge of hypertension (15.4%), $p < 0.001$.

There was almost same proportions of subjects that had knowledge of hypertension in the lower and the higher classes ie 41.9% versus 41.1%, $p = 0.9090$.

Knowledge of hypertension was higher in subjects whose fathers' level of education was secondary or above (48.8%) than those whose fathers' level of education was below secondary (34.8%). This difference was however not statistically significant, $p = 0.05858$. Equally, subjects whose mothers' level of education was secondary or above had a slightly higher proportion of subjects that had knowledge of hypertension (50.0%) than those whose mothers' had below secondary level of education (36.8%), this difference was also not statistically significant, $p = 0.666$.

5. 3. 2 Diabetes

About two thirds (67.0%) of the total study population were aware of diabetes. However, there was no significant difference in the level of awareness between males and females (68.6% vs 65.0%), $p = 0.3971$.

Awareness of diabetes was higher in the age group 12 –15 years (75.5%) compared to under 12 (56.8%), this difference was statistically significant, $p < 0.001$.

With respect to location, awareness of diabetes was significantly higher in the urban compared to the rural schools (UHD 76.1%, ULD schools 67.5%). The Rural schools had the least proportion of subjects that were aware of diabetes (60.3%), $p = 0.0064$. These results are shown in table 12.

One hundred and eighty six (55.9%) out of the 333 subjects that were aware of diabetes had knowledge about diabetes. Knowledge of diabetes was slightly higher in females (59.0%) as compared to males (51.7%), an insignificant difference, $p = 0.1823$.

No significant difference was found in the level of knowledge of diabetes with regard to age. The proportion of subjects that had knowledge of diabetes was almost similar in the younger (56.9%) and the older subjects (55.2%), $p = 0.7536$.

The ULD schools had the highest proportion of subjects that had knowledge of diabetes (73.9%), followed by the UHD schools (54.8%) and the rural schools had the least proportion (42.7%). This difference was statistically significant, $p < 0.001$.

Class of study seem to have no influence on the knowledge of diabetes. There was almost similar proportions of subjects that were aware of diabetes in the lower classes (55.4%) as compared to the higher classes (56.2%), $p = 0.8822$.

A significantly higher proportion of subjects whose fathers' education level was secondary and above had knowledge about diabetes (69.2%) as compared to those whose fathers' level of education was below secondary (49.5%), $p = 0.0015$. Equally subjects whose mothers' level of education was secondary and above had a higher proportion of individuals that had knowledge of diabetes (68.3%) as compared to those whose mothers' had below secondary level of education (54.6%), $p = 0.02616$. These results are shown in table 13.

5. 3. 3 Stroke

A total of 487 subjects responded to this question regarding awareness of stroke. Out of which 199 (24.4%) said that they have heard and were aware of stroke as a disease. Females were more informed than males (27.5% vs 20.6%, $p = 0.077$). Age was also an influencing factor as it was found that those aged 12 – 15 years were more aware of stroke (28.7%) as opposed to the younger subjects (19.5%), $p = 0.017$.

Awareness of stroke was significantly higher in subjects from ULD schools (38.1%) compared to UHD schools (24.4%), and the rural schools had the least proportion of subjects that were aware of the disease (14.6%), $p < 0.001$. Subjects in the higher classes were more aware of stroke (35.1%) as opposed to those in the lower classes (13.4%), $p < 0.001$.

Subjects whose fathers had secondary education or above had a significantly higher level of awareness of stroke (34.4%) compared to those whose fathers had lower education levels (16.4%), $p < 0.001$. Similar results were observed in offsprings of mothers who had secondary or higher education, table 12.

Fifty (42.0%) out of the 199 subjects that were aware of stroke had knowledge about the disease. Approximately similar proportions of males and females had knowledge about stroke, ie 40.0% versus 43.2%, $p = 0.7281$.

There was no significant difference as far as age was concerned in the knowledge of stroke. The proportion of subjects that had knowledge of stroke was (40.9%) in the younger subjects and (42.7%) in the older subjects, $p = 0.8512$.

Knowledge of stroke was significantly higher in the ULD schools (52.8%) as compared to the UHD schools (39.5%) and the Rural schools (25.0%), $p = 0.05$. With regard to class of study, there was no significant difference in the level of knowledge about stroke between the lower (37.5%) and the upper classes (43.7%), $p = 0.544899$.

Subjects whose fathers had secondary or above level of education had a slightly higher proportion of individuals that had knowledge of stroke (43.4%) as compared to those whose fathers had below secondary level of education (41.7%). This difference was however not statistically significant, p = 0.879704. Knowledge of stroke was also not associated to mothers' level of education, being 48.1% in those whose mothers' had secondary or above level of education and 34.1% in the subjects whose mothers had below secondary level of education, p = 0.1709. These results are shown in table 13.

5. 3. 4 Heart Attack

A total of 117 (24.5%) subjects admitted to have heard about heart attack and thus were aware of the disease. Males were more aware (25.2%) compared to females (23.9%). This difference was not statistically significant, p 0.729. The older subjects were more likely to be aware of heart attack (31.5%) as opposed to the younger subjects (16.5%), $p < 0.001$.

A significantly higher proportion of subjects in the ULD schools were aware of heart attack (34.3%) compared to the

UHD (23.0%) and the rural schools (18.3%), $p = 0.0035$. Subjects from the higher classes were more likely to be aware of heart attack (33.3%) as opposed to those from the lower classes (15.3%), $p < 0.001$.

Awareness of heart attack was found to be higher in subjects whose fathers had secondary or above level of education (30.9%) as compared to those whose fathers had lower education (20.0%), $p = 0.0225$. There was no significant difference between the level of awareness of heart attack as between mothers' level of education $p = 0.0556$, (Table 12).

Fourty seven (40.2%) out of the 117 subjects that were aware of heart attack seem to know the disease.

The knowledge of heart attack was slightly higher in males (46.3%) as compared to their females counterparts (34.9%), an insignificant difference, $p = 0.2108$. Knowledge of heart attack was almost similar in both age groups, 40.5% in the younger subjects and 40.0% in the older subjects, $p = 0.9557$.

Subjects from ULD schools had a slightly higher proportion of subjects that had knowledge of heart attack (43.8%), followed by the UHD schools (40.0%) while the rural schools had the lowest proportion of subjects that knew the disease (35.3%). This difference was not statistically significant, p = 0.7435.

Knowledge of heart attack was higher in the higher classes (43.2%) as compared to the lower classes subjects (33.3%). The difference was however not statistically significant, p = 0.3145.

Parents' level of education seem to have no influence on the knowledge of heart attack. These results are shown in table 13.

Table 12. Awareness of the main cardiovascular diseases by demographic characteristics

Characteristic	*Hypertension (N = 499)*		*Diabetes (N = 497)*		*Stroke (N = 487)*		*Heart Attack (N = 478)*	
	n	*%*	*N*	*%*	*n*	*%*	*N*	*%*
Total series	499	46.5	497	67.0	487	24.2	478	24.5
Gender								
Males	224	41.9*	223	65.0	218	20.6	214	25.2
Females	275	50.9	274	68.6	269	27.5	264	23.9
Age group (years)								
6 –11	232	34.9*	229	56.8**	226	19.5	224	16.5
12 – 15	267	56.6	268	75.7	261	28.7	254	31.5
School category								
†ULD	142	51.4	140	67.5*	139	38.1**	140	34.3
‡UHD	164	49.4	163	76.1	156	24.4	152	23.0
Rural	193	40.4	194	60.3	192	14.6	186	18.3
Class								
1 – 4	248	34.7**	244	60.7**	239	13.4**	235	15.3**
5 – 7	251	58.2	253	73.1	248	35.1	243	33.3
¶Father education								
Secondary+	226	56.2*	224	71.0	221	34.4**	220	30.9*
Below secodary	151	45.7	148	60.9	146	16.4	140	20.1
¶Mother's education								
Secondary+	174	55.2	173	69.4	168	32.1*	168	29.8*
Below secondary	193	49.2	189	68.5	191	21.5	182	20.9

$*p < 0.05$, $** p < 0.001$

ULD = Urban Low Density, UHD = Urban High Density

¶ Not all subjects knew their parents' education level

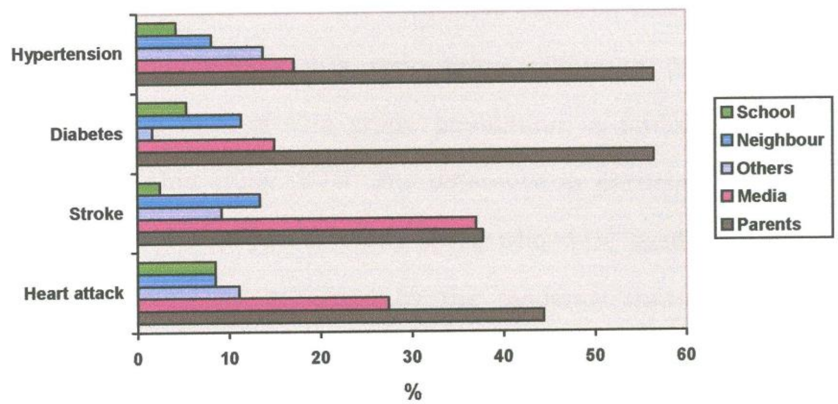
Table 13. Knowledge of cardiovascular diseases by demographic characteristics

Characteristic	*Hypertension (N=232)*		*Diabetes (N= 333)*		*Stroke (N= 119)*		*Heart Attack (N = 117)*	
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*
Total series	232	41.4	333	55.9	119	42.0	117	40.2
Gender								
Males	92	39.1	145	51.7	45	40.0	54	46.3
Females	140	42.9	188	59.0	74	43.0	63	34.9
Age group (years)								
6 – 11	81	38.3	130	56.9	44	40.9	37	40.5
12 – 15	151	43.0	203	55.2	75	42.7	80	40.0
School category								
ULD	73	58.9**	92	73.9**	53	52.8*	48	43.8
UHD	81	50.6	124	54.8	38	39.5	35	40.0
Rural	78	15.4	117	42.7	28	25.0	34	35.3
Class								
1 – 4	86	41.9	148	55.4	32	37.5	36	33.3
5 – 7	146	41.1	185	56.2	87	43.7	81	43.2
¶Father's education								
Secondary+	127	48.8*	159	69.2*	76	43.4	64	50.0
Below secondary	69	34.8	99	49.5	24	41.7	28	35.7
¶Mother's education								
Secondary+	96	50.0	120	68.3*	54	48.1	50	52.0
Below secondary	95	36.8	130	54.6	41	34.1	38	36.8

* $p < 0.05$, ** $p < 0.001$
ULD = Urban Low Density, UHD = Urban High Density
¶ Not all subjects knew their parents' education level
NB: Only subjects that were aware of the diseases were assessed for the level of knowledge

When asked about where they heard about the different cardiovascular diseases, the following responses were given;

Figure 7. Sources of information about cardiovascular diseases.



Parents were the main source of information of cardiovascular diseases, being mentioned by more than 50% of the respondents who were aware of hypertension and diabetes. It was also the main source of information with regard to stroke and heart attack. The second main source of information about cardiovascular diseases was media, which was more common as far as stroke and heart attacks were concerned. Of note is that school was the least mentioned source of information about cardiovascular diseases, being mentioned by less that 10% in all the cardiovascular diseases asked. The ``other´´ mentioned sources included hospital, in the streets, etc.

6. DISCUSSION

Seventy three percent of the consented children had complete data and were analysed. Girls were more willing to partcipate thus constituting 55% of this study population, a finding that is not unique to this study.[136,137] The difference in participation between those aged 6 –11 years versus those who were aged 12 –15 years could possibly be explained by the tendency that the younger subjects were more likely to have incomplete data, especially when it came to collecting blood samples, thus constituting only 46% of the study population. It is estimated that more than 95% of children who are school age children are at school, so this data roughly represent children in Tanzania.

Several studies have demonstrated that systolic and diastolic blood pressures have a strong, continuous, graded and etiological significant positive association with cardiovascular disease outcome.[51] In this study the mean blood pressure (both systolic and diastolic) was found to rise with age, consistent with earlier reports.[138,139] The mean (± SD) systolic and diastolic blood pressures were 108.22 ± 11.89 mmHg and 66.32 ± 8.69 mmHg respectively. These results are similar to those found by Hamidu et al[140] in a study done to determine the blood pressure profile of Nigerian children aged

5 – 16 years whereby the mean systolic and diastolic blood pressures were found to be 102.28 mmHg and 65.10mmHg respectively.

The prevalence of systolic, diastolic and both (systolic and diastolic) hypertension was found to be 11.4%, 8.1% and 3.9% respectively. This is different from the prevalences found earlier within Tanzanian adolescents aged 15 – 19 years,[31] whereby the prevalence of hypertension was found to be only 0.4%. This difference could be due to the fact that these reseachers used a cut off point for hypertension as blood pressure levels of ≥ 140/90 mmHg, regardless of the age, height and sex of the child/adolescent. The present results are however similar to those found by Chadha et al[141] in which the prevalence of hypertension (systolic, diastolic or both) among Indian children was found to be 11.9% in boys and 11.4% in girls aged 5 – 14 years. The similarity is detected yet these study populations are of different cultural background.

The area of residence of the subjects have been found to have an influence on their mean blood pressure levels. In this study, subjects from the urban areas had higher mean systolic and diastolic blood pressures, furthermore within the urban area, there was a significant difference in the mean blood pressure between those from Urban Low Density areas compared to those from Urban High Density areas.

These results are similar to those found in earlier studies within African communities[142,143] which show differentiation of blood pressure levels according to area of settlements. Areas of settlement are mainly determined by affluence and socio-economic status of the individual families. This has also a bearing on lifestyle. A significant difference was also found regarding parental educational status. Those children whose parents had secondary and above level of education, had higher mean levels for both systolic and diastolic blood pressures. This again can be explained by the difference in socio-economic status. Parents who are more educated are more likely to have good jobs thus having a higher income and live a more sedentary lifestyle than parents who are not educated or less educated.

Pediatric obesity (defined as body mass index in the 95th percentile or higher for age and sex) is a serious, chronic disease. Health care professionals should be concerned about overweight and obesity because of the well established relation between excess weight and such medical conditions as hypertension, hypercholesterolemia, type 2 diabetes, and to an increased incidence of musculoskeletal injuries[144]. Worldwide, the prevalence of obesity is reported to have doubled since mid 70's, most marked in the developed countries. In the US, Gortmaker et al[145] reported a 54% increase in childhood obesity from 1960 to 1980.

The prevalence of obesity in this study population was found to be 5.3%. It was 5.7% in females compared to 4.8% in males, an insignificant difference. Kane et al[146] found the prevalence of obesity to be 3% in a rural area of Thiadaye, Senegal. Among Portuguese children aged 6 – 15 years, the prevalence was found to be 7%.[157] The prevalence of obesity was higher in Indian children (aged 13 – 17 years) in which it was found to be 10%.[34] In Tanzanian adolescents aged 15-19 years, Kitange et al[31] found the prevalence of overweight to be in only 0.4% at the age of 15 years. This difference in the prevalence could be due to the differences in defining obesity in children and adolescents and methodology applied (they used the cut off points of obesity and overweight for adults).

The striking finding in this study was the differences in the prevalence of obesity in the three school settings, ie 16.9%, 1.8% and 0.0% in the ULD, UHD and the Rural schools respectively. This can be explained by the difference in the socioeconomic status of the parents in these school settings. The children in the ULD schools were more likely to eat more of excess energy and use less of their energy due to lack of physical activities. It is however interesting to note that the prevalence of obesity was higher in the younger subjects, thus it seems some of the obese children loose their weight as they grow older. However, this may not be trully harmless as Vanhala et al[147] showed that obesity at age of seven predicts the metabolic syndrome in adulthood. Whether this is true for the children who lose their weight before they become adults needs further investigation.

In contrast to the present results, McMurray and Collegues found that the rural children of North Carolina in the US had a 54.7% increased risk of obesity[158] when compared to the urban children. This fact can be explained by the natural history of socio-economic transitions.[159] The increase in obesity during childhood is multifactorial. Several studies have revealed that the sedentary lifestyle of today's children and adolescents is a major determinant. Also causally related to obesity is the availability of food rich in energy ``fast foods´´ around school premises, as well as familial obesity, socioeconomic status of parents, which makes children have enough money to spend in buying food rich in energy and soft drinks. Lack of physical fitness and activity levels have also been found to contribute.[148]

The health problems brought about by cigarette smoking can not be overemphasised. Most studies have shown a strong dose-response relation of cigarettes smoked to deaths from Cardiovascular diseases. In addition, smoking increases the risk of peripheral arterial occlusive disease, and various forms of cancer.[120]

In this study, cigarette smoking was found in only 1.2% of the subjects. These results are similar to earlier findings by Kitange et al[31] in which the prevalence of cigarette smoking was found in only 1.1% of the adolescents aged 15 years. Low prevalences were also found in India by Gupta et al[34], in which the prevalence was (0.4%). The results are however different from those found in most European and American children.[149,150]

The low prevalence of active cigarette smoking in these children can be explained by the fact that at this young age parents still have control of their childrens' behaviours, and culturally parents discourage smoking at young age. However, this trend can change any time from the present since there is an increased level of cigarette advertisement in posters, radios, and televisions. This can have an impact on children since most of them are now having access to these different media and some go to boarding schools at a young age and can pick on smoking habits from other students.

Surprisingly was the finding in this study of high prevalence (35%) of passive smoking in these young children. These findings suggest that even if the young children do not smoke by themselves, they cannot escape the sidestream smoke that is comming from their own parents and close relatives. So, with the high prevalence of smoking in the adult population (21% in Dar es Salaam and 28% in Morogoro, Kitange et al[32]) and due to lack of special restriction of smoking in public areas, the problem of smoking will still be there in these children.

Related to the prevention of cardiovascular diseases, daily physical activity in children has become a field of interest. Epidemiological studies have shown that physical activity appears to offer some degree of protection against cardiovascular diseases, hence a fundamental approach for prevention should begin at childhood.[113]

This study found most of the children (61%) to be active, engaging themselves in sports, doing farm and other house activities (8%), and most of them were going to school on foot as part of their daily activities. There was however a difference in the level of activities with regard to the different school settings. The subjects from ULD school were more likely to go to schools using motor vehicles as compared to those from the UHD and Rural schools, thus lacking walking as part of physical activity. However this study did not look on the actual levels of physical activities since this would have involved direct observation of the level of activities of the children, so no major conclusions can be drawn from these findings. Other activities which were more likely to be undertaken by the subjects from the rural schools are things like water fetching from distances, assisting in food harvest or collecting firewood. These activities are invariably done in rural and some less well off families in the urban settlements eventually leading to these children being more active than those from the affluent urban families.

Type 2 diabetes in children is an increasing problem worldwide, being more marked in the developed world. The problem goes hand in hand with the worldwide increase in the prevalence of obesity in children, coupled with today's sedentary lifestyle.

In this study the mean (SD) level of blood glucose was found to be 4.88 (0.74) mmol/l, with an insignificant difference between males (4.84 ± 0.63 mmol/l) and females (4.93 ± 0.64 mmol/l). This figure slightly exceed that found by other researchers[31] among Tanzanian adolescents aged 15 –19 years in which the mean level was 4.2 mmol/l with no difference between males and females.

The blood glucose levels were found to be slightly but significantly higher in the younger subjects as compared to the older ones (4.99 mmol/l versus 4.78 mmol/l), $p < 0.001$. This is in contrast to what was reported previously,[62,63] however these results show a significant difference in the mean blood glucose levels with regards to school settings. The most prosperous school setting, ULD had the highest mean level of blood glucose. This trend has also been observed by previous researchers.[34] The differences in the mean blood glucose levels in this study population can be explained by the differences in the levels of type 2 diabetes precursors in the different school settings. As reported earlier, the most prosperous subjects (those from ULD schools) were more likely to be obese and to have a sedentary lifestyle as compared to those from the UHD and the Rural subjects. The two (ie obesity and physical inactivity) are major determinants of glycaemic level. Generally, subjects from the affluent socio-economic background had an increased chance of having more caloric intake due to the type of food that is usually taken in the Urban social life, ie high energy foods such as chips, animal products and these children are more likely to take soft drinks, sugary foods like cakes, chocolates, etc, at home and while at school since they have enough money to spend.

Levels of serum cholesterol have been correlated with a person's subsequent risk of heart disease. Clinical studies have shown that serum cholesterol screening may reduce the risk of cardiovascular diseases in adults, and for high risk children and adolescents.[151] The issue on how much serum cholesterol concentration constitutes a risk for cardiovascular disease has long been debetable. The Framingham study indicated a continuous increase of risk of cardiovascular disease with serum cholesterol values above 4.65 mmol/l (180 mg/dl).[152] In countries in which the incidence of cardiovascular diseases is very low, the mean serum cholesterol values of adults are in the range of 3.62 - 4.14 mmol/l (140 – 160 mg/dl) but there is still a continuum of risk.[153] Furthermore, in animal model atherosclerosis does not develop when serum cholesterol levels are ≤ 4.65 mmol/l (180 mg/dl).[152] Previous studies have demonstrated that between the age of 5 – 18 years, the ideal mean serum cholesterol is 2.85 mmol/l (110 mg/dl) while the feasible mean is approximately 3.62 mmol/l (140 mg/dl).[154] In the present study we have shown that the mean serum total cholesterol to be 3.52 mmol/l in males and 3.61 mmols/l in females. These results are in keeping with those found by others[34] among Tanzanian adolescents in which the values were 3.46 mmol/l in males and 3.74 mmol/l in females. This still reflects a low risk profile from hypercholesterolemia.

In the present study, the mean values of total serum cholesterol were found to be significantly higher in the most affluent school children. Whether this difference in serum cholesterol levels is protective to the under previleged children (Rural) against atherosclerosis or whether the affluent children are exposed and therefore at risk of adult cardiovascular disease is a question that need to be part of a long term follow up study. The fact that these children have a favourable cholesterol profile does not exempt them from the occurance of atherosclerotic changes in their blood vessels later in life. In a recent publication by the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group[155] it was clearly shown that there is a substantial effect of the nonlipid risk factors on the extent and severity of coronary and aortic atherosclerosis even in the presence of a favourable lipoprotein profile. These results support the need for control of all cardiovascular risk factors.

Awareness and knowledge of the risk precursors to cardiovascular diseases is thought to be a key component of health decision making. However few studies have been done to determine the level of awareness and knowledge of cardiovascular diseases among children despite what the WHO recommends; i.e raising awareness of cardiovascular diseases and their precursors among children as a way of primary or primordial prevention.

This data revealed that the level of awareness of the main cardiovascular diseases was generally low in this study population and even in those who were aware of the diseases, their knowledge about the disease was also low. Diabetes was the one disease that many children were aware of (67%), followed by Hypertension (46.5%). Stroke and Heart attacks (24.2% and 24.5% respectively) were less known to the children. The low levels of awareness of cardiovascular diseases could possibly be reflecting the low prevalences of the diseases among this society thus they can be thought of as being not common.

The terminologies that are used to describe cardiovascular diseases in our native language are also not very popular. For instance, the Swahili word for hypertension is ``shinikizo la damu´´. Not so many people are aware of this terminology and people may sometimes talk of ``pressure´´, ``BP´´, meaning hypertension. The Swahili word for diabetes is ``Kisukari´´. This terminology is somehow more meaningful as it descibes the disease by itself, it is well understood by the general population and this can explain the fact that it was the disease that was known to many of the children. The word ``Kiharusi´´, which is a Swahili word for stroke is confusing to most of the children since it does not describe the disease and it has other meaning apart from the disease itself. Literally, there is no Swahili word for heart attack, and the word is most of the time confused with other heart problems, and even simple fainting attacks. Because the understanding of the disease terminologies was difficult, it is possible that some children thought they know the diseases while in actual sense they were confusing with something else. Thus the level of awareness and knowledge of these diseases could be lower.

Generally, the older subjects were more aware of diabetes (56.6% vs 34.9%) and hypertension (75.7% vs 56.8%) as compared to the young subjects. This difference was not found with stroke and heart attack, instead, parents' level of education and the school setting of the children determined the level of awareness of the diseases. Children from the ULD schools were found to be more aware of strokes (38.1%) and heart attacks (34.3%) as compared to those from the UHD (stroke 24.4%, heart attack 23.0%) and the Rural schools (stroke 14.6%, heart attack 18.3%). With regards to parents' level of education, those whose parents had secondary or above level of education were more likey to be aware of strokes and heart attack and there was no difference when diabetes and hypertension were asked about.

The differences in awareness of strokes and heart attack between the school settings can be explained by the fact that the children from the affluent families are more likely to watch television, listen to radio, etc as these were the second most common sources of information about stroke and heart attack mentioned. It is possible that parents of the affluent children were more likely to talk about these diseases as they were more likely to be affected by themselves or having a relative affected.

The level of knowledge of the cardiovascular diseases among the children aware of the diseases was generally similar for hypertension, stroke, and heart attack (41.4%, 42.0% and 40.2%) respectively. It was slight high in diabetes (55.9%). Collectively this data demonstrated that among the children who are aware of cardiovascular diseases only less than half of them know what the disease are in terms of cause (risk factors), symptoms and how the diseases can be prevented or avoided by change of lifestyle. It was found that the level of knowledge of the cardiovascular diseases is higher in the children from the affluent groups. This can be due to parental influence and access to different media of communication. This study did not look into the influence of teachers on the level of awareness and knowledge of cardiovascular diseases. This is important because children spend about a third of their day time at school, so teachers have to be aware of the different precursors of cardiovascular diseases for them to help instilling those healthy-related behaviours to children.

Experience has shown that raising awareness of cardiovascular diseases and their precursors pays off. In the new twist, the Phillipines[19] aimed a programme to prevent cardiovascular diseases not to adults but to pre-teen aged children starting from grade one of almentary school. The rationalle for its departure from the usual practice was that it is easier to insill healthy lifestyles in the young than to change bad habits in adulthood. Thus, even though most diseases of the heart and arteries occur after the middle age, the fight against them should begin with children. This programme proved to be very successful[19].

7. 0 CONCLUSION AND RECOMMENDATIONS

The following conclusions can be drawn from this study:

1. The prevalences of the conventional cardiovascular risk factors are generally low among children in this society.
2. There is a marked difference in the prevalences of cardiovascular risk factors among children from different socio-economic background, being very low in those from the rural and low income background and considerably high in those from the affluent socio-economic bakground.
3. There is generally low levels of awareness and knowledge of cardiovascular diseases and their precursors among children

The following recommendations are put forward:

1. Still there is an opportunity for primordial prevention of cardiovascular diseases in this society.
2. There is a need to educate children about cardiovascular diseases, their risk factors and the risky-related behaviours for cardiovascular diseases so as to raise awareness.
3. Schools should be used to raise awareness of cardiovascular diseases in children as this have shown to be effective in other societies.

4. Further studies are recommended to look on the level of awareness and knowledge of cardiovascular diseases among teachers and parents as this will have an impact on the children also.
5. It is recommended that these children be followed up into adulthood to see the trends of the levels of cardiovascular risk factors. The programme should be extended to larger community cohorts.

8. 0. REFERENCES

1. Davies M J (ed) 1993 Atherosclerosis. British Heart Journal 69:S1-S73.

2. Sytkowski PA, Kannel WB, D'Agostino RB. Changes in risk factors and the decline in mortality from cardiovascular disease. The Framingham heart study. N Engl J Med 1990;322:1635-41.

3. Breslow JL. Mouse Models of Atherosclerosis (Review). Science 1996 (May 3); 272 (5262):685 – 8.

4. Stary HC. Evolution and Progression of Atherosclerosis Lesions in Coronary Arteries of Children and Young Adults. Atherosclerosis, 1989;1:19.

5. Ross R. The pathogenesis of atherosclerosis-An update. N Engl J Med 1986;314:488 – 500.

6. Ip JH, Fuster V, Badmon L, Badimon J, Taubman MB, Chesebro J H. Syndromes of accelerated atherosclerosis : role of vascular injury and smooth muclse cell proliferation. J Am Coll Cardiol 1990 ;15 :1667 – 87.

7. American National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. Update on the 1987 task force report on high blood pressure in children and adolescents: a working group report from the National

High Blood Pressure Education Program. Pediatrics. 1996;98(4):649-658.

8. Brown MS, Goldstein JL. Lowering plasma cholesterol by raising LDL receptors. N Engl J Med 1981;305:515-517.

9. Azizi M, Guyene TT, Chatellier G, Wargon M, Menard J. Additive effects of lorsatan and enalapril on blood pressure and plasma active renin. Hypertension 1997;29:634-640.

10. Sacks FM, Pfeffer MA, Moye LA et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. Cholesterol and Recurrent Events Trial (CARE) investigators. N Eng J Med. 1996;335:1001-9.

11. Scandinavian Simvastatin Survival Group. Randomized trial of cholesterol lowering in 4444 patients with coronary heart disease: Scandinavian Simvastatin Survival Study (4S). Lancet 1994;344:1383-1389.

12. Jukema JW, Bruschke AVG, van Boven AJ, et al. Effects of lipid lowering by pravastatin on progression and regression of coronary artery disease in symptomatic men with normal to moderately elevated serum cholesterol levels: the Regression Growth Evaluation

Statin Study (REGRESS). Circulation 1995;91:2528-2540.

13. Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad base of initial cholesterol levels. N Eng J Med 1998;339:1344-57.

14. The West of Scotland Coronary Prevention Study Group. Prevention of Coronary Heart Disease with Pravastatin in men with hypercholesterolemia. N Eng J Med 1995;333:1301-1308.

15. Hansson L, Zanchetti A, Carruthers SG, et al. Effects of intensive blood-pressure lowering and low-dose aspirin in patients with hypertension: principal results of the Hypertension Optimal Treatment (HOT) randomised trial. HOT Study Group. Lancet 1998;351:1755-62.

16. Chockalingham A, Balaguer-Vintro I. Impending global pandemic of cardiovascular diseases. Barcelona, Prous Sciences/World Health Federation, 1999.

17. Dawber TR. The Framingham Study: the epidemiology of atherosclerotic disease. Cambridge, Massachusetts, Havard University Press, 1990.

18. Pathobiological Determinants of Atherosclerosis in Youth (PBDAY) Research Group. Relationship of atherosclerosis in young men to serum lipoprotein cholesterol concentrations and smoking. A pleriminary report from the Pathobiological Determinants of Atherosclerosis in Youth (PBDAY) Research Group. JAMA, 1990;264:3019-3024.

19. WHO Report on World Health Day, 7th April 1992. World Health Organization, CH-1211 Geneva 27, Switzerland

20. White PD. *Heart Disease,* 4th ed., p.956 – 957. New York: The MacMillan Company, 1951.

21. Campbell M. The main cause of increased death rate from diseases of the heart: 1920 to 1959. Br. Med J, 1963;3:712-717.

22. The impending global epidemic of cardiovascular diseases. Eur Heart J, Vol 21, issue 11, June 2000, 880-883.

23. Murray CJ, Lopez AD, eds. The Global Burden of Diseases: A comprehensive Assessment of Mortality and Disability From Diseases, Injuries and Risk Factors in 1990 and Projected to 2020. USA, Havard School of Health, 1996.

24. Watkins LO. Coronary heart disease risk factors in black populations in underdeveloped countries: The case for primordial prevention. Am Heart J 1984; 108:850-62.

25. Seftel HC. The rarity of coronary heart disease in South African Blacks. S Afr Med J. 1978; 54: 99-105.

26. Williams AO. Coronary atherosclerosis in Nigeria. Br Heart J 1971;33:95-100.

27. Miall WE, Bras G. Heart disease in the tropics. Br Med Bull 1972;28:79-86.

28. Wu ZK, Wu ZS, Yao CH. Epidemilogic studies of cardiovascular diseases in China. Chin Med J 1983;96:201-05.

29. Sarvotham SG, Berry JN. Prevalence of coronary heart disease in an urban population in Northern India. Circulation 1968;37:936-53.

30. Miller GJ, Beckles GLA, Maude G et al. Ethnicity and other characteristics predictive of coronary heart disease in a developing community: principal results of the St James survey, Trinidad. Int J Epidemiol 1989;18:808-17.

31. Kitange HM, Swai ABM, Masuki G, Kilima PM, Albert KGMM, McLarty DG. Coronary heart disease risk factors in Sub- Saharan Africa: Studies in Tanzanian

adolescents. J Epidemiol Cmmunity Health 1993;47:303-307.

32. Swai ABM, McLarty DG, Kitange HM, Kilima PM, Tatalla S, Keen N, Chuwa LM, Albert KGMM. Low prevalence of risk factors for coronary heart disease in rural Tanzania. Int. J. Epidemiol 1993;22:651-659.

33. Berenson GS, Srinivasan SR, Freedman DS, Radhakrishnamurthy B, Dalferes ER Jr. Atherosclerosis and its evolution in childhood. Am J Med Sci 1987;294:429-440.

34. 34.Gupta R. Goyle A, Kashyap S, Agarwal M, Consul R, Jain BK. Prevalence of atherosclerosis risk factors in adolescent school children. Indian Heart Journal. 1998;50(5);511-5.

35. Enos WF, Beyer JC, Holmes R. Pathogenesis of coronary disease in soldiers killed in Korea. JAMA 1955;148:912-914.

36. McNamara JJ, Molot MA, Stremple JF, Cutting RT. Coronary artery disease in combat casualties in Vietnam. JAMA 1971; 216:1185-1187.

37. Stary HC. Evolution and progression of atherosclerotic lesions in coronary arteries of children and young adults. Arteriosclerosis 1989;9(suppl 1):119-132.

38. Hirvonen J, Yla-Herttuala S, Laaksonen H, et al. Coronary intimal thickennings and lipids in Finnish children who died suddenly. Acta Paediatr Scand 1985;318(suppl):221-224.

39. Newman WP 111, Freedman DS, Voors AW, et al. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis: the Bogalusa Study. N Engl J Med 1986:314:138-144.

40. Freedman DS, Newman WP 111, Tracy RE, et al. Black – white differences in aortic fatty streaks in adolescence and early adulthood: the Bogalusa Heart Study. Circulation 1988;77:856-864.

41. Berenson GS, McMahan CA, Voors AW, et al. Cardiovascular risk factors in children. The early natural history of atherosclerosis and essential hypertension. New York: Oxford University Press, 1980.

42. Khoury PJ, Morrison JA, Kelly, et al. Clustering and interrelationships of coronary heart disease risk factors in schoolchildren, ages 6 – 19. Am J Epidemiol 1980;112:524-538.

43. Laurer RM, Conor WE, Leaverton PE, Reiter MA, Clarke WR. Coronary heart disease risk factors in children. J Pediatr 1975;86:697-706.

44. Webber LS, Cresanta JL, Voors AW, Berenson GS. Tracking of cardiovascular risk factor variables in school aged children. J Chron Dis 1983;36:647-660.

45. Weber LS, Cresanta JL, Croft JB, Srinivan SR, Berenson GS. Transitions of cardiovascular risk from adolescence to young adulthood- the Bogalusa Heart Study 11. Alterations in anthropometric blood pressure and serum lipoprotein values. J Chro Dis 1986;39:91-103.

46. Berenson GS, Srinivan SR, Nicklas TA, Webber LS. Cardiovascular risk factors in children and early prevention of heart disease. Clin Chem 1988;34:B115-B122.

47. Clarke WR, Schrott HG, Leaverton PE, Connor WE, Lauer RM. Tracking of lipids and blood pressure in school age children: The Muscatine Study. Circulation 1978;58:626-634.

48. Laskarzewiski P, Morrison JA, deGroot I, et al. Lipid and lipoprotein tracking in 108 children over a four-year period. Pediatrics 1979;64:584-591.

49. Anne McMunn, Paola Primatesta, Lualhati Bost. The Health of Young People 1995-97.

50. Kannel WB. Blood Pressure as a cardiovascular risk factor: prevention and treatment. JAMA 1996; 275:1571-1576.

51. Collins R, Peto R, MacMahon S, Hebert P, Fiebach NH, Eberlein KA, Godwin J, Qizilbash N, Taylor JO and Hennekens CH (1990) Blood Pressure, stroke, coronary heart disease. Part 2, Short term reductions in blood pressure: Overview of randomized drug trials in their epidemiological context. Lancent 335:827-838.

52. De Swiet M, Fayers P, Shinebourne EA. Blood Pressure in first 10 years of life: the Brompton study. Br Med J 1992;304:23-26.

53. Laure RM, Clarke WR. Childhood risk factors for high adult blood pressure: The Muscatine Study. Pediatrics 1989;84:633-641.

54. Nelso MJ, Ragland DR, Syme SL. Longitudinal Prediction of Adult Blood Pressure from Juvenile Blood Pressure Levels. Am J Epidemiol 1992;136:633-645.

55. Chen Y, Rennie DC, Reeder BA. Age-related associations between body mass index and blood pressure. The Humboltdt Study. Int J Obesity 1995;19:825-831.

56. Clarke WR, Woolson RF, Lauer RM. Changes in pendorosity and blood pressure in childhood: the Muscatine Study. Am J Epidemiol 1986;124:195-206.

57. Kaas Ibsen K. Factors influencing blood pressure in children and adolescents. Acta Paediatr Scand 1985;74:416-422.

58. National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. Update on the 1987 Task Force Report on High Blood Pressure in Children and Adolescents: A Working Group Report on High Blood Pressure Education Program. Pediatrics 1996;98:649-658.

59. Neel JV. Diabetes mellitus: a ``thrifty´´ genotype rendered detrimental by ``progress´´? Am J Hum Genet 1962;14:353-62.

60. Ohlson LO, Larsson B, Bjorntorp P, et al. Risk factors for type 2 (non-insulin dependent) diabetes mellitus: thirteen and one-half years of follow-up of the participants in a study of Swedish men born in 1913. Diabetologia 1988;31:798-805.

61. LaPorte RE, Matsushima M, Chang YF. Prevalence and incidence of insulin-dependent diabetes. In: Harris MI, Cowie CC, Stern MP, Boyko EJ. Reiher GE, Bennett PH,

eds. Diabetes in America. 2nd ed. Washington , DC: National Institutes of Health, NIDDK, 1995:37-46.

62. Onkamo P, Vaananen S, Karnoven M, Tuolmilehto J. Wordwide increase in incidence of type 1 diabetes: the analysis of data on published incidence trends. Diabetologia 1999:42:1395-403.

63. Fagot-Campagna A. Emergence of type 2 diabetes mellitus in children: the epidemiological evidence. J Pediatr Endocrinol Metabol (in press).

64. Fagot-Campagna A, Pettitt DJ, Engelgau MM, Rios Burrows N, Geiss LS, Valdez R, et al. Type 2 diabetes among North American children and adolescents: An epidemiological review and public health perspective. J Pediatr 2000;136:664-72.

65. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Over-weight prevalence and trends for children and adolescents. The National Health Examination Surveys. 1963 to 1991. Arch Pediatr Adolesc Med 1995;149:1085-91.

66. American Diabetes Association. Type 2 diabetes in children and adolescents. Diabetes Care 2000;23:381-9.

67. Lauer RM, Clarke WR. Use of cholesterol measurements in childhood for prediction of adult hypercholesterolemia: The Muscatine Study. JAMA 1990;264:3034-8.

68. Sprecher DL, Schaeter EJ, Kent KM, Gregg RE, Zech LA, Hoeg JM, McManus B, Roberts WC, Brewer HB Jr. Cardiovascular features of homozygous familial hypercholesterolemia: analysis of 16 patients. Am J Cardiol 1984;54:20-30.

69. Klag M J, Ford OE, Meal LA, He J, Whelton PK, Liang KY, Levine DM. Serum cholesterol in young men and subsequent cardiovascular diseases. N Engl J Med 1993;328:313-18.

70. Anderson KM, Caslelli WP, Levi D. Cholesterol and mortality: 30 years of follow up from the Framingham study. JAMA 1987;257:2176.

71. Schmidt HH, Hill S, Makariou EV, Feuerstein IM, Dugi KA, Hoeg JM. Relation of cholesterol year score to severity of calcific atherosclerosis and tissue deposition in homozygous familial hypercholesterolemia. Am J Cardiol 1996;77:575-80.

72. Freedman DS, Shear CL, Srinivasan SR, Webber LS, Berenson GS. Tracking of serum lipids and lipoproteins

in children over an 8 year period. The Bogalusa Heart Study. Prev Med 1985;14.203-16.

73. Hennekens CH, Jesse MJ, Klein BE, Goutley JE, Blurnenthal S. Cholesterol among children of men with Myocardial Infarction. Pediatrics 1976;58:211-17.

74. Aetna Coverage Policy Bulletins, 2000. Number 0525: Cholesterol Screening.

75. US National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. Pediatrics 1992;89(3 Pt 2):525-584.

76. Roche AF, Siervogel FM, Chumlea WB, Webb P. Grading body fittness from limited anthropometric data. Am J Clin Nutr. 1981;34:2831-2838.

77. Barlow, SE and Dietz, WH. Obesity evaluation and treatment: expert committee recommendations. Journal of Pediatrics 1998;102(3):e29.

78. Medalie JH, Papier CM, Goldbourt U, Herman JB. Major factors in the development of diabetes mellitus in 10,000 men. Arch Intern Med. 1975;135:811-817.

79. Mann GV. The influence of obesity on health. N Engl J Med. 1974;291:178-185, 226-232.

80. Barret-Conor EL. Obesity, atherosclerosis, and coronary artery disease. Am Intern Med. 1985;103:1010-1019.

81. Pi-Sunyer FX Medical hazards of obesity. Am Intern Med. 1993;119:655-660.

82. McGill HC Jr, McMahan A, Malcom GT, Oalmann MC, Strong JP. Relation of glycohemoglobin and adiposity to atherosclerosis in youth. Arterioscler Thromb Vasc Biol. 1995;15:432-440.

83. Dietz WH Jr, Gortmaker SL. Factors within the physical environment associated with childhood obesity. Am J Clin Nutr. 1984;39:619-624.

84. Leibel RL, Behary N, Friedman JF. Strategies for the molecular genetic analysis of obesity in humans. Crit Rev Food Sci Nutr. 1993;33:351-358.

85. Bradon F, Rodgers B, Wadsworth M, Davies J. Onset of obesity in a 36 year birth cohort study. Br Med J. 1986;293:299-303.

86. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. Prev Med. 1993;22:167-177.

87. Guo SS, Roche AF, Chumlea WC, Gardner JC, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35. Am J Clin Nutr. 1994;59:810-819.

88. Gidding SS. A perspective on obesity. Am J Med Sci. 1995;310(suppl 1):S68-71.

89. Stunkard A, Sorenson T, Hanis C, Teasedale T, Chakraborty R, Schull W, Shlsinger F. An adoption study of human obesity. N Eng J Med. 1986;314:193-198.

90. Stunkard A, Foch T, Hrubec Z. A twin study of human obesity. JAMA. 1986;256:51-54.

91. Borjeson M. The aetiology of obesity in children. Acta Paediatr. 1976;65:279-287.

92. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents: the National Health and Examination Surveys, 1963 to 1991. Arch Pediatr Adolesc Med. 1995;149:1085-1091.

93. Linder CW and RH DuRant. Exercise, serum lipids, and cardiovascular disease-risk factors in children. Pediatr. Clin. North. Am. 1982; 29:1341-1354.

94. Marrison, JA, CJ Glueck. Pediatric risk factors for adult coronary heart disease: Primary atherosclerosis prevention. Cardiovasc. Rev. Rep. 1981; 2:1269-1281.

95. Shear, CL, DS Freedman, GL Burke, DW Harsha, LS Webber, and GS Berenson. Secular trends of obesity in

early life: The Bogalusa heart study. Am. J. Pub. Health. 1988;78:75-77.

96. Strong, WB. Physical activity and children. Circulation 1990; 81:1697-1701.

97. Tell, GS, and OO Vellar. Physical fitness, physical activity, and cardiovascular risk factors in adolescents: The Oslo youth study. Prevent. Med. 1988; 17:12-24.

98. Treiber, FA, WB Strong, FW Arensman, and M Gruber. Relationships between habitual physical activity and cardiovascular responses to exercise in young children. In, Children and Exercise X111, SC Oseid and KH Carlson (Eds) Champaign, IL: Human Kinetics Publishers, 1989, pp 285-294.

99. Dietz, WH, Jr. Childhood obesity: Susceptibility, cause, and management. J. Pediatr. 1983; 61:813-819.

100. Pate RR, and JG Ross. Factors associated with health-related fitness. JOPERD. 1987; 58:93-96.

101. DuRant, RH, T. Baranowski, T Rhodes, B Guctin, et al. Association among serum lipid and lipoprotein concentrations for physical activity, physical fitness, and body composition in young children. J. Pediatr. 1993;123:185-192.

102. Williams, DP, SB Going, TG Lohman, DW Harsha, et al. Body fatness and risk for elevated blood pressure, total cholesterol and serum lipoprotein ratios in children and adolescents. Am J Public Health. 1992; 82:358-363.

103. Roccini, AP, V Katch, J Anderson, J Hinderliter, et al. Blood pressure in obese adolescents: Effects of weigt loss. Pediatrics 1988; 82:16-23.

104. Webber, LS, JL Cresanta, AW Voors, and GS Berenson. Tracking of Cardiovascular disease risk factor variables in school-age children. J Chron. Dis. 1983; 36 :647-660.

105. Must, A, PF Jacques, GE Dallal, CJ Bajema, and WH Dietz. Long-term morbidity and mortality of overweight adolescents. N. Engl. J. Med. 1992; 327:1350-1355.

106. Dennison BA, JH Straus, ED Mellits, and E Charney. Childhood physical fitness tests: Predictor of adult physical activity levels? Pediatrics 1998; 82:324-330.

107. Raitakari OT, KVK Porkka, S Taimela, R Talema, L Rasanen, and JSA Viikari. Effects of persistent physical activity and inactivity on coronary risk factors in children and young adults. Am J Epidemiol. 1994; 140:195-205.

108. Blair SN, HW Kohl 111, RS Paffenbarger, DG Clark, KH Cooper, and LW Gibbons. Physical fitness and all-cause mortality. JAMA 1989;262:2395-2401.

109. Gibbons LW, SN Blair, KH Cooper and M Smith. Association between coronary heart disease risk factors and physical fitness in healthy adult women. Circulation 1983;67:977-983.

110. Paffenbarger Jr. RS, RT Hyde, AL Wing and CC Hsieh. Physical activity, all-cause mortality, longevity of college alumni. N Engl J Med 1986; 314:605-614.

111. Pate RR, and JG Ross. Factors associated with health-related fitness. JOPERD. 1987; 58:93-96.

112. Sallis JF. Epidemiology of physical activity and fitness in children and adolescents. Crit. Rev. Food Sci. Nutr. 1993; 33:403-408.

113. Whitehead JR, CL Pemberton, and CB Corbin. Perspectives on the physical fitness of children: The case for a realistic educational approach. Pediatr. Exerc. Sci. 1990; 2:111-123.

114. Payne VG, and JR Morrow Jr. The effects of physical training on prepubescent VO2max: A meta-analysis. Res. Q. Exerc. Sport. 1993; 64 :305-313.

115. Pate RR, M Dowda, and JG Ross. Association between physical activity and physical fitness in American children. AJDC 1990 144:1123-1129,.

116. Morrow JR Jr. and PS Freedson. Relationship between habitual physical activity and aerobic fitness in adolescents. Pediatr. Exerc. Sci. 1994; 6:315-329.

117. Freedson, PS. Physical activity among children and youth. Can. J. Sport Sci. 1992; 17:280-283.

118. Pate RR. Physical activity assessment in children and adolescents. Crit. Rev. Food. Sci. Nutr. 1993; 33:321-326,.

119. Riopel DA, Bolerth RC, Coates TJ, Miller WW, Weideman WH, Hennekens CH. Coronary risk factor modification in children:smoking. Circulation 1986;74:1192A-1194A.

120. Fielding JE. Smoking: health effects and control. N Engl J Med 1985;313:491-498,555-565.

121. Gill JS, Shipley MJ, Tsementzis SA, et al. Cigarette smoking. A risk factor for hemorrhagic and nonhemorrhagic stroke. Arch Intern Med 1989;149:2053-2057.

122. Floderus B, Cederlof R, Frberg L. Smoking and mortality: a 21-year follow up based on Swedish Twin Registry. Int J Epidemiol 1988;17:332-340.

123. Haapanen A, Koskenvuo M, Kaprio J, Kesaniemi YA, Heikila K. Carotid arteriosclerosis in identical twins discordant for cigarette smoking. Circulation 1989;80:10-16.

124. Brown KS, Cherry WH, Frobes WF. The 1978 national survey of smoking habits of Canadian school children. Can J Public Health 1986;77:139-146.

125. Leventhal H, Glynn K, Fleming R. Is the smoking decision an ``informed choice´´? Effect of smoking risk factors on smoking beliefs. JAMA 1987;257:3373-3376.

126. Davidson DM. Carbon monoxide effects in persons with symptomatic coronary artery disease. Thesis, University of California, Irvine, 1987, 93 pp.

127. Cryer PE, Haymond MW, Santiago JV, Shah SD. Norepinephrine and epinephrine release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. N Engl. J Med 1976;295:573-577.

128. Laustiola KE, Lassila R, Kaprio J, Koskenvuo M. Decreased b-adrenergic receptor density and catecholamine response in male cigarette smokers. Circulation 1988;78:1234-1240.

129. Fielding JE, Phenow KJ. Health effects of involuntary smoking. N Engl J Med 1988;319:1452-1460.

130. Mattson ME, Boyd G, Byar EG, et al. Passive smoking on commercial airline flights. JAMA 1989;261:867-872.

131. US Department of Health and Human Services. The health consequences of involuntary smoking. A report of the surgeon general. Washington: Public Health Service, 1986. DHHS Publication no. (CDC) 87-8398.

132. Byrd JC, Shapiro RS, Schiedermayer DL. Passive smoking: a review of medical and legal issues. Am J Public Health 1989;79:209-215.

133. Davis JW, Shelton L, Watanabe IS, Arnold J. Passive smoking affects endothelium and plateletes. Arch Intern Med 1989;149:386-389.

134. Baugh JG, Hunter SM, Webber LS, Berenson GS. Developmental trends of first cigarette smoking experience of children: the Bogalusa Heart Study. Am J Public Health 1982;72:1161-1164.

135. Hunter SM, Baugh JG, Weber LS, Sklov MC, Berenson GS. Social learning effects on trial and adoption of cigarette smoking in children: the Bogalusa Heart Study. Prev Med 1982;11:29-42.

136. Melkiory C. Masatu, Knut-Inge Klepp, Gunnar Kvåle. Use of Health Services and Reported Satisfaction Among Primary School Adolescents in Arusha, Tanzania. J Adolesc Health 2001;28:278-287.

137. Aten MJ, Siegel DM, Roghmann KJ. Use of health services by urban youth: a school-based survey to assess differences by grade level, gender, and risk behaviour. J Adolesc Health 1996;19:258-66.

138. Obika L.F.O, Adedoyin M.A and Olowoyeye J.O. Patterns of paediatric blood pressure in rural, semi-urban and urban communities in Ilorin, Nigeria. Afr. J. Med. Sci. 1995;24:371-75.

139. Akinkugbe O.O., Akinkugbe F.M., et al. Biracial study of arterial pressures in the first and second decades of life. Brit. Med. J. 1977;1132-1134.

140. Hamidu L J, Okoro E O AND Ali M A. Blood pressure profile in Nigerian Children. East African Medical Journal 2000;77:180-184.

141. Chadha SL, Tandon R, Shekhawat S, Gopinath N. An epidemiological study of blood pressure in school children (5 -14 years) in Delhi. Indian Heart Journal 1999;51(2):178-82.

142. Abrahams DG, Alele CO and Barnard. The systemic blood pressure in a rural African community. West Afr J Med 1996;7:345-59.

143. Akinkugbe OO and Ojo OA. The systemic blood pressure in a rural African community. Trop geogr Med 1986;20:367- 371.

144. Deitz WH: Childhood obesity: susceptibility, cause and management. J Pediatr 1993;103:676-686.

145. Gortmaker S, Dietz W, Sobol A, et al: Increasing pediatric obesity in the United States. Am J Dis Child 1987;141:535-540.

146. Kane A, Ly M, Diouf ND, Dia AA, Diop PS, Diao M, et al. Arterial pressure and body mass index of children and

adolescents in a rural area of Thiadaye, Senegal. Dakar Medical. 1998;43(1):83-9.

147. Vanhala MJ, Vanhala PT, et al. Relative weight gain and obesity as a child predict metabolic syndrome as an adult. International Journal of Obesity and Related Metabolic Disorders. 1999;23(6)656-9.

148. Becque M, Katch V, Rocchini A, et al: Coronary risk incidence of obese adolescents: reduction by exercise plus diet intervention. Pediatrics 1988;81:605-612.

149. Byckling T, Sauri T. Atherosclerosis precursors in Finnish children and adolescents. XII. Smoking behaviour and its determinats in 12-18-year-old subjects. Acta Paediatr Scand Suppl 1985;318:195-203.

150. Tell GS, Vellar OD. Noncommunicable diseases risk factor intervention in Norwegian adolescents: the Oslo Youth Study. In:Hetzel B, Berenson GS (eds): Cardiovascular risk factors in childhood:epidemiology and prevention. New York: Elsevier, 1987:203-217.

151. American Academy of Pediatrics Committee on Nutrition. Cholesterol in childhood. Pediatrics 1998;101:141-6.

152. Kanel WB, Castelli WP, Gordon T, McNamara PM. Serum cholesterol, lipoproteins and the risk of coronary heart

disease: the Framingham study. Am Intern Med 1971;74:1-12.

153. Chen Z, Peto R, Collins R, MacMahon S, Lu J, Li W. Serum cholesterol concentration and coronary heart disease in population with low cholesterol concentrations. BMJ 1991;303:276-82.

154. WHO Expert Committee. Prevention of coronary heart disease. WHO Tech Rep Ser 1982;678.

155. Henry C, McGill Jr, MD, et al. Effects of Nonlipid Risk Factors on Atherosclerosis in Youth with a Favorable Lipoprotein Profile. Circulation 2001;102:1546-1550.

156. Taha AZ, Bella H. Heart disease risk factors: prevalence and knowledge in a primary care setting, Saudi Arabia. Saudi medical journal 1998;4(2):293-300.

157. Gerber ZR, Zielinsky P. Risk factors for atherosclerosis in children: an epidemiologic study. Arquivos Brasileiros de Cardiologia. 1997;69(4):231-6.

158. McMurray RG. Harrell JS. Bangdiwala SI. Deng S. Cardiovascular disease risk factors and obesity of rural and urban elementary school children. Journal of Rural Health. 1999;15(4):365-74.

159. Srinath RK. Cardiovascular diseases in the developing countries. HeartBeat 1998;2:1-5.