UNIVERSITI PUTRA MALAYSIA

PREVALENCE OF HYPERTENSION AND FACTORS ASSOCIATED AMONG SECONDARY SCHOOL STUDENTS IN BATANG PADANG DISTRICT PERAK

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ABSTRACT

Background: Hypertension (HPT) is the leading treatable risk factor for cardiovascular disease mortality as it has been widely reported in various regions of the world. Increased blood pressure levels during childhood strongly predict HPT in young adulthood. Among adolescents and young adults; elevated blood pressure is also associated with the presence of early atherosclerotic lesions.

Objective: To identify the prevalence and the associated factors of hypertension among secondary school students aged 12-17 in Batang Padang District, Perak.

Methods: A cross sectional study was conducted among 2131 secondary school students. Self – administered questionnaire was used to obtain information on socio demography, smoking status and physical activity level. Height, weight and blood pressure were taken and Body Mass Index (BMI) was calculated. The response rate was 87.1%. All data were analyzed using SPSS version 21.

Result: Out of 1857 respondents, 20.0% of them had hypertension. By using Chi-Square Test, it was shown that there were significant association between hypertension with gender, ethnicity, smoking status and nutritional status ($p<0.05$). However, there were no significant association between hypertension with age group, family history, and physical activity ($p>0.05$).

Conclusion: The overall prevalence of hypertension among secondary school students in Batang Padang District, Perak was 20.0% with significant association between hypertension with gender, ethnicity, smoking status and nutritional status ($p<0.05$). However, only ethnicity, gender, and obese status were found as the confounder for HPT ($r^2= 0.254$, $p< 0.05$), with male (AOR= 1.315), Chinese (AOR= 3.242), and obese (AOR= 7.852).

Key words: Hypertension, Prevalence, Secondary School Students

KELAZIMAN HYPERTENSI DAN FAKTOR-FAKTOR BERKAITAN DALAM KALANGAN PELAJAR SEKOLAH MENENGAH DI DAERAH BATANG PADANG,PERAK.

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ABSTRAK

Latar belakang: Hypertensi adalah faktor utama terhadap kadar kematian penyakit kardiovaskular yang boleh dirawat sebagaimana yang telah dilaporkan oleh negara-negara di dunia. Peningkatan paras tekanan darah semasa zaman kanak-kanak memberikan jangkaan hypertensi yang kukuh ketika awal dewasa. Dalam kalangan remaja dan golongan awal dewasa; peningkatan tekanan darah adalah berkaitan dengan kehadiran awal luka arterosklerotik.

Objektif: Untuk mengenal pasti kelaziman dan faktor-faktor yang berkaitan hypertensi dalam kalangan pelajar sekolah menengah berumur 12-17 di daerah Batang Padang, Perak.

Kaedah: Suatu kajian rentas telah dijalankan dalam kalangan 2131 responden. Borang soal selidik telah digunakan untuk mendapatkan maklumat tentang demografisosial, status merokok dan tahap aktiviti fizikal. Ketinggian, berat badan dan tekanan darah telah diambil dan indeks jisim badan dikira dengan menggunakan formula. Semua data dianalisis dengan menggunakan SPSS versi 21.0.

Keputusan: Daripada 1857 responden, 20.0% daripadanya didapati menghidap hypertensi. Dengan menggunakan ujian Chi-Square, telah ditunjukkan bahawa terdapat kaitan yang signifikan antara hypertensi dengan jantina, etnik, status merokok dan status nutrisi ($p<0.05$). Bagaimanapun, tiada kaitan yang signifikan antara hypertensi dengan kumpulan umur, sejarah keluarga yang menghidap hypertensi dan aktiviti fizikal ($p>0.05$).

Konklusi: Kadar kelaziman keseluruhan hypertensi dalam kalangan pelajar sekolah menengah di Daerah Batang Padang, Perak adalah 20.0% dengan kaitan yang signifikan antara hypertensi dengan jantina, etnik, status merokok dan status pemakanan ($p <0.05$). Namun, hanya etnik, jantina dan status obesiti telah didapati sebagai konfounder untuk hypertensi (r^2: 0.254 , $p<0.05$), iaitu lelaki (AOR= 1.315), Cina (AOR= 3.242), dan obes (AOR= 7.852).

Kata Kunci: Hypertensi, Kelaziman, Pelajar Sekolah Menengah

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LIST OF ABBREVIATIONS

CVD	:	Cardiovascular Disease
HPT	:	Hypertension
BP	:	Blood Pressure
SBP	:	Systolic Blood Pressure
DBP	:	Diastolic Blood Pressure
WHO	:	World Health Organization
BMI	:	Body Mass Index
DASH	:	Dietary Approaches to Stop Hypertension
CARDIA	:	Coronary Artery Risk Development in Young Adult
DALYs	:	Disability-Adjusted Life Years
PAQ-C	:	Physical Activity Questionnaire for Children
SPSS	:	Statistical Package for Social Sciences
AOR	:	Adjusted Odd Ratio
SD	:	Standard Deviation
CDC	:	Centres for Disease Control and Prevention
GSHS	:	Global School Health Survey

CHAPTER 1: INTRODUCTION

1.1 Background

Cardiovascular disease (CVD) is the number one cause of death throughout the world and kills more people each year than any other disease. It is responsible for 25% of all deaths worldwide.[1] CVD mortality is likely to continue to increase in developing countries, if no appropriate action is taken.[2] Hypertension (HPT) is the leading treatable risk factor for CVD mortality as it has been widely reported in various regions of the world.[3-13] It is ranked third as a cause of disability-adjusted life-years and is a leading risk factor for mortality and 1.56 billion people are expected to have HPT by 2025.[9,14] It causes more than seven million deaths every year worldwide.[12]

In Malaysia, the prevalence of HPT amongst adults aged 30 years and above has increased from 32.9% in 1996[15] to 40.5% in 2004[16] to 42.6% in 2006[17] and slightly decrease to 42.2% in 2011[18], which is high. HPT is an established risk factor and contributes substantially to CVD incidence and premature mortality.[19]

Increased blood pressure (BP) levels during childhood strongly predict HPT in young adulthood.[20-21] Among adolescents and young adults; elevated BP is also associated with the presence of early atherosclerotic lesions.[22] In another study carried out in Putrajaya, Malaysia by Rampal *et al.* in 2010, the prevalence of pre- HPT and HPT among the male was 16.2% and 12.9% respectively. The prevalence of pre- HPT and HPT in the female was 5.8% and 10.2% respectively. The overall prevalence of pre- HPT and HPT was 11.1% and 11.6% respectively, which is high.[23]

1.2 Problem Statements

In Malaysia, cardiovascular disease has been the leading cause of death for the past 40 years.[16] HPT is the leading treatable risk factor for CVD mortality as it has been widely reported in various regions of the world.[3-13] National Health and Morbidity Survey 2011 includes adults aged 18 and above, showed that the prevalence of HPT was 32.7%.[18] Increased BP levels during childhood strongly predict HPT in young adulthood.[20-21] In another study carried out in Putrajaya, Malaysia by Rampal *et al.* in 2010, the prevalence of pre- HPT and HPT among the male was 16.2% and 12.9% respectively. The prevalence of pre- HPT and HPT in the female was 5.8% and 10.2% respectively. The overall prevalence of pre- HPT and HPT was 11.1% and 11.6% respectively, which is high.[23]

1.3 Objectives

1.3.1 General Objective

The general objective of this study was to determine the prevalence of HPT and factors associated among secondary school students aged 12-17 in Batang Padang District, Perak.

1.3.2 Specific Objectives

The specific objectives of this study were:

i. To compare the mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) by age, gender and ethnicity.

ii. To determine the prevalence of HPT among secondary school students aged 12-17 in Batang Padang District, Perak.

iii. To determine the association between HPT and associated factor (socio- demographic characteristics, physical inactivity, smoking, obesity).

1.4 Hypothesis:

The alternative hypotheses were:

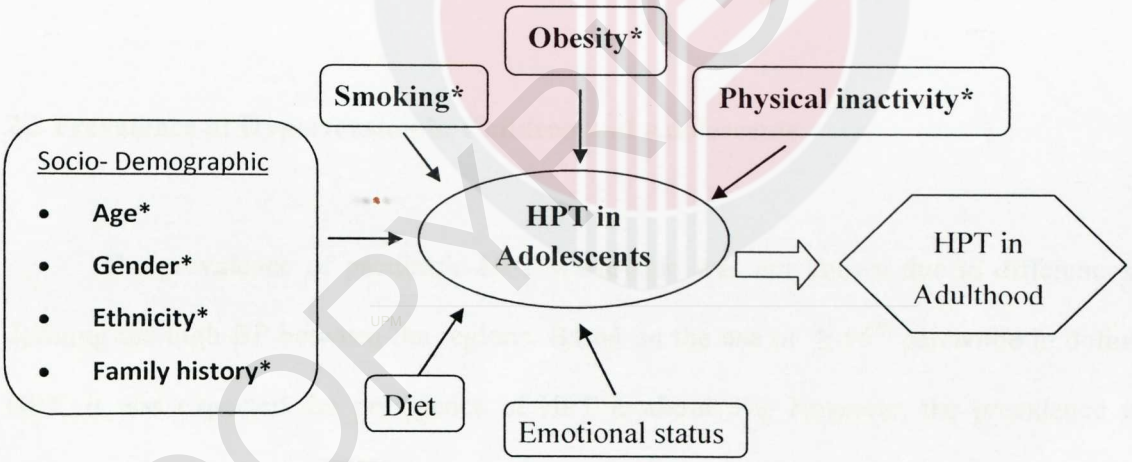
H_1: There is an association between HPT and socio- demographic characteristics.

H_2: There is an association between HPT and smoking status.

H_3: There is an association between HPT and obesity.

H_4: There is an association between HPT and physical inactivity.

1.6 Conceptual Framework



***The factors that concerned in this research.**

Figure 1.1 Risk factors of hypertension in adolescents.

CHAPTER 2: LITERATURE REVIEW

2.1 Prevalence of Hypertension

According to a report by the World Health Organization (WHO) in 2009, the leading global risk for mortality in the world was HPT which accounts for 13% death worldwide.[13] It became one of the risks responsible for raised in chronic diseases such as heart disease. A study on global burden of HPT showed that there was a total of nearly one billion people experienced HPT in 2000 which more than half of it account in Asia, while the number was estimated to increase up to 1.56 billion people in the year of 2025 which the Asian was estimated to contribute about 580 million of the cases.[9] In Malaysia, the prevalence of HPT had increase from 32.9% in 1996[15] to 42.6% in 2006[17] among adults age 30 years old above.

2.2 Prevalence of Hypertension in Children and Adolescents

The prevalence of paediatric HPT worldwide was not known due to differences in defining the high BP between the regions. Based on the use of $\geq 95^{th}$ percentile in defining HPT, it was expected the prevalence of HPT is about 5%. However, the prevalence was expected to be lower that 5% due to effects of accommodation and regression to mean of repeated measures.[24] The prevalence of high BP had been rise among US children and adolescent since 1963-2002. The rising trend of the BP among them was indicated due to obesity and some include racial factor.[25]

A study conducted among children and adolescents aged 3-18 years old in Ohio shown that the prevalence of HPT is 3.6% with the age, height and obesity contributes to the

diagnosis.[26] Another study in Seychelles among those in age group 5-16 years old shown that the prevalence of elevated BP in boys and girls were 9.1% and 10.1% respectively with significant association with the body mass index (BMI).[27] In a study conducted in Texas among 1066 children aged 8-13 years old, the prevalence of high BP was reported to be 20.6% and was reported to be higher in that overweight and Hispanic group.[28]

2.3 Classification of Hypertension

Table I : Classification of hypertension in children and adolescent.[4,29]

Classification of Hypertension in Children and Adolescents	
	SBP or DPB Percentile*
Normal	$<90^{th}$
Pre-HPT	90^{th} to $<95^{th}$ or if BP exceeds 120/80 even if $<90^{th}$ up to $<95^{th}$ percentile**
Stage 1 HPT	$95^{th} - 99^{th}$ plus 5 mmHg
Stage 2 HPT	99^{th} plus 5 mmHg
*For gender, age, and height measured on at least 3 separate occasions; if SBP and DBP categories are different, categorize by the higher value. **This occurs typically at 12 years old for SBP and at 16 years old for DBP.	

2.4. Risk Factors

2.4.1 Non - Modifiable Risk Factors

Age

Aging was identified as a possible risk factor for the progression of HPT. Elevated blood level in increasing age might be due to result of aging process in organ system. Advanced aging of the cardiovascular system contributed to the presence of a varied-phenotype in elderly HPT, such as morning HPT and nocturnal HPT.[30] A study by Rampal *et al.* showed that the prevalence of HPT in Malaysia increased as age increased. The prevalence of HPT for subjects aged ≥15 years old was 27.8% while the prevalence of HPT among those aged ≥30 years old had increased from 32.9% in 1996 to 40.5% in 2005. For those aged ≥50 years old, the prevalence of HPT was said to be higher in female during the study.[16] Prevalence of

HPT by age groups which were above 18 years in 2011 is 8.1% in 18-19 age group, 11.8% in 20-24 age group, 14.3% in 25-29 age group, 22.2% in 30-34 age group, 27.2% in 35-39 age group, 35.8% in 40-44 age group, 44.0% in 45-49 age group, 51.6% in 50-54 age group, 61.0% in 55-59age group, 68.7% in 60-64 age group, and 74.1% in 65-69 age group.[31] This indicated that the prevalence of HPT increased with age.

Gender

Previous review concluded that androgen hormone might contribute to the differences of BP between genders. Boys tended to have higher BP compare to girls after the onset of the puberty due to the increased in the level of the androgen. However, post- menopause women experienced increased in BP which was most likely due to decreasing effect of estrogen hormone.[32] A previous study conducted in the sub- district of Dengkil, Selangor in respondents aged 15-60 years old and above, shown that the prevalence of HPT in male is higher compared to female (31.7%:23.5%) with the association between the two variables was significant.[33] Among the adolescent in Malaysia, the prevalence of pre- HPT and HPT in female was 5.8% and 10.2% respectively while the prevalence of pre-HPT and HPT in male was 16.2% and 12.9% respectively. Thus, the result showed that that was an association between HPT and gender.[23]

Ethnicity

Most studies in United Kingdom and United States reported a higher prevalence and lower awareness of HPT in black than in white people. Black people were reported to have higher prevalence than either group of white people of different social status.[34] Another study in

2002 showed elevated BP level after first screening was highest among Hispanics which account 25% while lowest was among Asians which total up to 14%.[35] In Malaysia, a study conducted in 2008 shown that for respondent aged ≥15 years, the Chinese had the highest prevalence of HPT which account for 30.6% followed by Malays (26.7%) and the Indians (25.1%). The indigenous people from the state of Sarawak, the Sarawak Bumiputera had a higher prevalence (31.1%) compared to other ethnic group.[16]

Family History

Various studies had shown that people with family history of HPT had a risk of developing HPT.[36-37] Based on a screened cohort in Okinawa Japan, the chances of getting HPT was higher for those with family history of HPT with the number of first- degree family members with HPT, which was quantitatively correlated with the risk of developing the condition. The positive family history of HPT might lead to an increased of HPT through genetic heritability of factors associated with HPT, inheritance of susceptibility to the effects of such factors and family sharing of lifestyle.[38] In a study involving adolescent in Malaysia, the prevalence of HPT was higher among those who had family history (12%) as compared to those who had no family history of HPT (11.3%). However, the difference was not statistically significant ($p>0.05$).[23]

2.4.2 Modifiable Risk Factors

Diet

The main modifiable risk causes of high BP were diet, which include high salt intake, excessive alcohol intake. Usually the BP following the rise in age was rise steadily with the

cumulative effect of the stated factors except in the largely absent of obesity, high physical activity and low salt intake.[39] A study about the Dietary Approaches to Stop HPT (DASH) diet which included reduced dietary sodium showed that the diet lowered the BP at high, intermediate, and low levels of sodium intake.[40]

Stress

In susceptible individual, stress caused a transient, sympathetically mediated rise in blood pressure. Repetitive exposures to stress were possibly result in habituation, with a consequent blunting of the systemic response and associated cardiodynamic changes.[41] A CARDIA study which involved follow-up in more than 4100 normotensive black and white man and women, shown that large BP changes in response to acute stressors (cold pressor, star tracing and video game tasks) predicted incident of HPT.[42]

Obesity

Obesity had become an important medical problem in children and adolescents and one of the outcome related to childhood obesity include HPT.[43] The link between obesity and HPT might be partly mediated by the sympathetic nervous system hyperactivity. The state of the hyperactivity might included the increased in heart rate and BP variability, increased in plasma level of catecholamines and increased in peripheral sympathetic nerve traffic which all of it included the cardiovascular, neurohormonal and neural manifestations.[44] In Malaysia, the prevalence of 'at risk of overweight' and overweight was 12.5% and 11.7% respectively.[23] In a study carried out by Rampal *et al.* in 2005 among 3,333 school children aged 13-17 years in the Klang district showed that the prevalence of 'at risk of overweight'

and 'overweight' was 11.4% and 8.2% respectively. The overall prevalence of overweight student aged 13-17 was 8.2% with a result of moderate direct significant relationship between overweight with SBP and DBP.[45]

Physical Inactivity

Physical inactivity was recognized as an important risk factor for multiple causes of death and chronic morbidity and disability. Account for the worldwide population, physical inactivity contributed for 21.5% of ischemic heart disease and 11% of ischemic stroke. It also caused 3.3% death all around the world and 19 million disability-adjusted life years (DALYs) worldwide.[46] A study in 2007 for the US adults population stated that adults with self-reported HPT reported being less physically active than those who were not hypertensive. They were also reported to be less likely met the current physical activity recommendations.[47] From a study conducted among Finnish man and women in 2003, regular physical activity was associated with a significantly reduced risk of HPT in both man and women.[48] The decreased of BP from the physical activity might be due to decreased in stroke volume and contractility of the heart after exercise,[49] combined with the decreased in vascular resistance which caused by the decreased in sympathetic activity.[50]

Smoking

Smoking was associated with arterial stiffness[51] and chronic low-grade inflammation[52] which both were related to HPT. Study in population-based sample of Vietnamese men in 2005 showed that those in a group, smokers had only a marginally higher prevalence of HPT than those who never smoke. Those in a group of ex- smoker however, have higher prevalence of

HPT compared to the current and never- smoke group.[53] An experimental study showed that an acute effect of the administration of tobacco or nicotine in man and animals was to produce small increased in BP and heart rate which was presumably due to the released of catecholamines.[54]

2.5. Complication

2.5.1 Hypertension in Adulthood

Increased BP levels during childhood strongly predict HPT in young adulthood.[20-21] In a previous study conducted by Sun *et al.*, it was concluded that children with SBP above the criterion values established in the study were at risk of HPT and metabolic syndrome in later life. The children of age group of 5-7 years old, with repeated measurements of SBP exceeding criterion values was found to have higher relative risk of adult HPT than older children and adolescents. Hence, it was suggested that the preventive and intervention program should begin as early as 5 years of age.[55]

CHAPTER 3: METHODOLOGY

3.1 Study Location

The study was carried out in Secondary Schools in Batang Padang District, which is located in the central part of Perak. Batang Padang District covers a total of approximately 271, 200 hectares, occupying almost 13 per cent of the total area of the entire state. It has a population of approximately 177, 000 people, mainly rural farmers and villagers spread out over the six territorial subdivisions or 'mukims'. There are 17, 382 secondary students distributed in 24 secondary schools.

3.2 Study Design

A cross sectional study design was used.

3.3 Study Population

The study population was a list of all 24 secondary schools in Batang Padang District.

3.4 Sample Size Estimation

The formula for hypothesis testing of two group comparison was used.[56]

$$n = \frac{\{z_{1-\frac{\alpha}{2}}\sqrt{2\tilde{P}(1-\tilde{P})} + Z_{1-\beta}\sqrt{P_1(1-P_1)+P_2(1-P_2)}\}^2}{(P_1-P_2)^2}$$

where

n = sample size,

$z_{1-\frac{\alpha}{2}}$ = Z statistic for level of confidence of 95% = 1.96

$Z_{1-\beta}$ = Z statistic for 80% power = 0.842

P_1 = prevalence of HPT in boys of 12.9%= 0.129[23]

P_2 = Prevalence of HPT in girls of 10.2%= 0.102[23]

$$\widetilde{P} = \frac{P_1 + P_2}{2} = 0.1155$$

n= 2200 which is the minimum required sample size

Adjusting for 10% non response:

n = 2200 + 10% of 2200 = 2420

Total sample size needed was 2420.

3.5 Sample Selection

The sample selection was done by using random cluster sampling method. Four schools were selected from the sampling frame using table of random numbers.

3.6 Sample Population

The sample population consisted of students from the 4 schools (SMK Buyong Adil, SMK Bidor, SMK Chenderiang, and SMJK Katholik) that were selected.

3.7 Sample Unit

The sample unit was every student in the classes that had been selected randomly, beginning from Form 1 until Form 5 from selected schools.

3.8 Research Instruments

A self- administered structured pretested questionnaire was used to collect data. BP was taken using an automatic electronic BP sets (HBP- 1300 Professional BP Monitor manufactured by Omron Healthcare Europe). Weight was taken using an automatic electronic weight balance (Tanita - HD 319 Lithium Digital Bathroom Weight Scale), while height was taken using a standing height rule (Charder - HM 200P Portable Stadiometer Height Measurement Scale).

3.9 Data Collection

3.9.1 Questionnaire

A validated pretested questionnaire in bilingual, Malay and English language was used for data collection. The bilingual questionnaire was made from the original English version and tried to ensure that the changes in the words to get across the same meanings. The questionnaire was divided into three sections;

Section A: Socio-Demographic data (gender, age, ethnicity, religion, family history, educational level of parents)

It was a self-administered instrument. It developed to access their age, gender, ethnicity, religion, and family history. Item age was calculated by subtracting the date of birth from the date of data collection of respondents by a closed end question in filling in the blank format. Gender, ethnicity, religion, family history, and educational level of parents were a multiple choice format question. It administered after the first taking of blood pressure. To prevent missing data, students was explained that the research is essentially to make sure they haven't missed any of the questions.

Section B: Physical Activity Questionnaire for Children & Adolescents[57-58]

It was a self-administered, 7- day recall instrument. It developed to assess general levels of physical activity for secondary school students. These included sports or dance that made them sweated or made their legs felt tired, or games that made them breathed hard, like skipping, running, climbing, and others. It administered after the first taking of blood pressure. This was the modified version of the Physical Activity Questionnaire for Children (PAQ-C) developed by Kowalski *et al.,* 2004 which contained both Physical Activity Questionnaire for Children and for Adolescents. Some of the sports that are not commonly done in Malaysia were deleted. It assessed the physical activity level over the past seven days. It consisted of nine items which scored in a five point likert scale to get the mean physical activity score. It also had an additional 10th question which assessed the unusual activity that prevented them from performing their usual physical activity. Each respondent had a score of 1-5 for each item and the mean was obtained. The mean was the final physical activity score. A score of 1 to 2.99 was classified as low physical activity and a score of 3 to 5 was classified as high physical activity.[57] When it administered, it was stressed that it was not a test or exams and explained to them that we were interested in actual activity during the last 7 days, so that they can answered the questions honestly. To prevent missing data, students was explained that the research assistants were not looking at their activity levels, but rather just making sure they haven't missed any of the questions.

Section C: Smoking questionnaire (Modified pretested version of wellness & quality of life questionnaire by Prof. Dr. Lekhraj Rampal Year 2013)

It was a self-administered instrument. It developed to assess general levels of smoking status for secondary school students. It administered after the first taking of blood pressure. There consisted of nine items in the questionnaire. Item of first started smoking, reason for starting

and quitting cigarette and smoking status were multiple choice format question. Duration of taking and stopping cigarette were closed end question in filling in the blank format. When it administered, it stressed that it was not a test or exams and explained to them that we were interested in actual smoking status, so that they can answered the questions honestly. To prevent missing data, students was explained that the research assistants were not looking at their smoking levels, but rather just making sure they haven't missed any of the questions.

3.9.2 Anthropometric Measurements (Height and Weight)

Tanita - HD 319 Lithium Digital Bathroom Weight Scale, which is an automatic electronic weight balance, was used to measure the weight of secondary students. Before a student's weight being measure, he/she was asked to take out all the stuff on body included shoes and socks, exclude clothing. Then, he/she was asked to stand on the balance until the reading stop only can let him/her for the height measurement. Height measurement was using a Charder - HM 200P Portable Stadiometer Height Measurement Scale to measure by taking off their shoes and made sure the hair part was excluded in measuring height.

3.9.3 Blood Pressure Measurements

To prevent any random errors that can be occur, we used HBP- 1300 Professional BP Monitor manufactured by Omron Healthcare Europe, which is an automatic electronic BP set to measure their blood pressure. We measured the BP of their brachial artery of left hand in the sitting position with the left forearm horizontal on a table, where the instrument was same level with the heart of student. When applied the cuff, we made sure that one finger was enough to pass beneath the cuff. Then, start/stop button was pressed and waited for the result

of BP showed on the screen. Two readings were taken in an interval of five minutes apart. Then the average of the 2 readings was recorded. All efforts were done to minimize the factors which might affect the BP such as anxiety, fear, stress, crying, laughing, and recent activity.[59] The students should avoided stimulant drugs or foods, had been sitting quietly for 5 minutes, and seated with his or her back supported, feet on the floor and right arm supported, cubital fossa at heart level.[60-61] Observer bias or digit preference can be minimized by the using of automatic devices.[62] Even under standard resting conditions, BP level was varies. Therefore, an average of multiple BP measurements can gave a more precise characterization of a person's BP level. Gender, age, and height were necessary to determine the normal range of BP in children and adolescents.[29]

3.10 Data Analysis

Data was analysed by using Statistical Package for Social Sciences Programmes (SPSS) for Windows, version 21.0. Descriptive characteristics of the respondents were obtained as mean, median, frequency & percentage. Chi square test was used to test association between the socio-demographic variables (gender, age, ethnics, and family history), physical activity level, smoking status, and nutritional status and HPT status. Kruskal- Wallis test was used as a non- parametric test to compare means of more than 2 groups like the age group and ethnicity. Wilcoxon Mann- Whitney was used as a non parametric test to compare means of 2 groups like the gender. Logistic Regression was used to control for confounder and determined the adjusted odd ratio (AOR). The level of significance was fixed at p less than 0.05.

3.11 Definition of Terms

Blood Pressure

With referring to the BP Table, Normal BP was defined as having average SBP and DBP < 90^{th} percentile; high normal/pre- HPT - average SBP or DBP $\geq 90^{th}$ and < 95^{th} percentile; HPT - average SBP or DBP $\geq 95^{th}$ percentile for gender, age and height.[4,29]

Age

Age was calculated by subtracting the date of birth from the date of data collection of respondents and took the complete year age without any rounding off value. Hence, age was categorized as 12, 13, 14, 15, 16, and 17 years old.

Gender

Gender of respondents was classified as male and female as answered in the questionnaire.

Family History

Family history was classified as with or without family history of HPT as answered in the questionnaire.

Ethnicity

Ethnicity was classified into Malay, Chinese, Indian, and others as answered in the questionnaire.

Physical Activity

We had a score from 1 to 5 for each of the items used in the physical activity composite score of the questionnaire, and we took the mean of those items, which resulted in the final physical activity summary score. After that, we divided the summary score by 9. A score of 1 to 2.99 indicated low physical activity, whereas a score of 3 to 5 indicated high physical activity.[57]

Obesity

Nutritional status was classified according to BMI. BMI was obtained by dividing the weight of each respondent in kilogram by the height in meter squared.[63-64] This was then compared with WHO Growth Reference (z- score curve) for 5 to 19 years by age and gender.[63,65] According to the chart, we found the intersection point,x of respondent's age and BMI. The age was rounded off to the nearest one month using date of birth and date of data collection as reference. $x \leq -2\ standard\ deviation(SD)$ was categorized as underweight, $-2 \leq x < +1\ SD$ was categorized as normal, $+1 \leq x < +2\ SD$ was categorized as overweight, and $x \geq +2\ SD$ was categorized as obese. Obese status was classified underweight and normal as non- obese, while overweight and obese as obese.

Smoking Status

Respondents who reported never having smoked 100 cigarettes were categorized as Never Smoker, while respondents who reported smoking at least 100 cigarettes in their lifetime were categorized as Ever Smoker.[66]

Height

With referring to the standard CDC height- age chart for 2 to 20 years to get the height percentile for respondents by gender, which were 5th percentile, 10th percentile, 25th percentile, 50th percentile, 75th percentile, 90th percentile and 95th percentile.[67]

CHAPTER 4: RESULTS

4.1 Response Rate

Out of the 2131 selected secondary school students between the age of 12-17 years old from selected 4 schools, 1857 (87.1%) of them took part in the study, giving a response rate of 87%. Non- response was for those students who were absent during the day of the data collection and also those who did not give the consents to participate in the research which accounted for 13%.

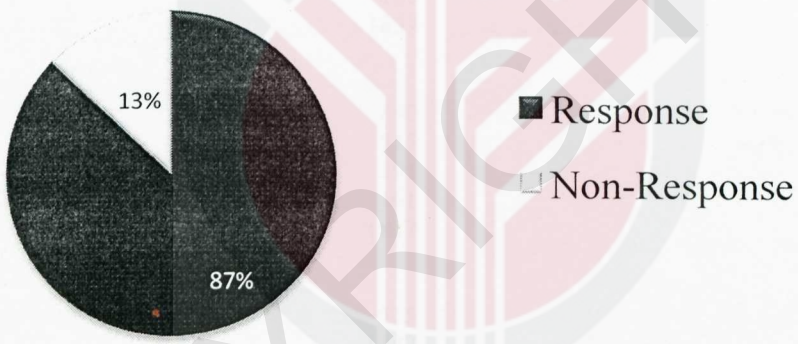


Figure 4.1 Distribution of response rate.

4.2 Socio-Demographic Characteristics

The mean age of the male and female respondents was 15.0 years old. Table II summarizes the socio-demographic characteristics of the respondents. There were 820 (44.2%) male respondents out of total 1857 respondents, while the rest (55.8%) were female respondents. Age group of 13 and 14 were approximately the highest with 388 (20.9%) and 390 (21.0%) respondents respectively, while age group 12 were the least with 169 respondents (9.1%). The rest were 375 (20.2%) of age group 15, 346 (18.6%) of age group 16, and 189 (10.2%) of

age group 17. Majority were Malays (47.9%) followed by Chinese (27.7%), and Indian (15.7%). The rest 8.7% (161 respondents) were composed by others ethnic. Out of the 1857, 515 (27.7%) respondents had a family history of HPT.

Table II : Socio-demographic characteristics of the respondents.

Factors	n	Percentage
Gender		
Male	820	44.2
Female	1037	55.8
Total	1857	100.0
Age		
12	169	9.1
13	388	20.9
14	390	21.0
15	375	20.2
16	346	18.6
17	189	10.2
Total	1857	100.0
Ethnicity		
Malay	890	47.9
Chinese	515	27.7
Indian	291	15.7
Others	161	8.7
Total	1857	100.0
Family History		
Yes	515	27.7
No	1342	72.3
Total	1857	100.0

4.3 Blood Pressure Measurements

According to the Shapiro- Wilk test of normality, p=0.000 for both mean SBP and mean DBP, which were less than the alpha value that had fixed (p=0.05). Hence, the null hypothesis was rejected indicated the mean SBP and mean DBP were not distribute normally.

Table III and VI show the mean SBP and DBP of the respondents. The mean SBP and DBP for 1857 respondents was 114.9 and 65.1 mmHg respectively. The mean SBP of male [117.8 (95% CI= 116.8 - 118.8)] mmHg was slightly higher than that of female [112.6 (95%

CI= 111.8 - 113.4)] mmHg. However, the mean DBP of male [64.6 (95% CI= 64 - 65.2)] mmHg was slightly lower than that of female [65.5 (95% CI= 65 - 66)] mmHg. A significant association was found between gender and mean SBP (p=0.000), but not in mean DBP (p=0.05).

The mean SBP and DBP were increased down the age group except in age group 15 where it declined slightly and same as previous age group 14 respectively. There is a statistically significant different between the mean SBP and mean DBP, and the age group (p=0.000); (p=0.000). By pairwise comparisons with Mann Whitney U test, there is a significant association in all the age group pairs for mean SBP except in pairs of age group (12- 13), (14- 15), (14- 16), (14- 17) and (16- 17). In mean DBP, there is also a significant association in all age group pair except in pairs of age group (12- 13), (14- 15), and (16- 17).

Table IV and VII show that the mean SBP among male and female was increased with age except at the age of 15 where it declined. For male, there is a statistically significant different between the mean SBP and the age group (p=0.000), but there is no for female (p= 0.128). For the mean DBP among male, the reading is shown increased with age except for the age of 15 where it declined while the mean DBP among female increased with age. A significant different is found between mean DBP and age group for both male and female (p=0.000); (p= 0.002).

By referring to Table III and VI, Chinese respondents had the highest mean SBP [119.9 (95% CI= 118.7- 121.1)] mmHg and mean DBP [68.2 (95% CI= 67.5- 69.0)] mmHg among all the ethnicity and followed by Malay with mean SBP [113.5 (95% CI= 112.6- 114.4)] mmHg and mean DBP [64.5 (95% CI= 64.0- 65.0)] mmHg. Indians had the lowest

mean DBP value [62.8 (95% CI= 61.9- 63.7)] mmHg while other ethnicity had the lowest mean SBP value [111.4 (95% CI= 109.3- 113.4)] mmHg. There is a statistically significant different between the SBP and DBP and the ethnicity (p=0.000); (p=0.000). By pairwise comparisons with Mann Whitney U test, there is a significant association in all the ethnicity pairs for SBP except in pairs of ethnicity (Malay- Indian) and (Indian- Others), while for DBP, all the ethnicity pairs were significant associated except in pairs of ethnicity (Malay- Others) and (Indian- Others).

In Table V and VIII, the mean SBP for Malay and Chinese were fluctuated down the age group, while the mean DBP of them were increased down the group except there was a decline in age group 15. In addition, the mean SBP and DBP for Indian were increased down the group. Moreover, the mean SBP and DBP for Others were also increased down the group except at age group 16 with a decline. There is a significant association in all the age group ($p<0.05$) with the mean SBP and mean DBP for all ethnicity, except in Indians for mean SBP ($p=0.053$).

Table III : Mean SBP of the respondents.

Factors	Mean	S.D	95% CI	Median	df	p
Overall	114.9	13.6		114.0		
Gender						
Male	117.8	14.6	116.8- 118.8	117.0	1	0.000*
Female	112.6	12.4	111.8- 113.4	111.5		
Age						
12	110.0	12.8	108.0- 111.9	109.0	5	0.000*
13	112.1	13.2	110.8- 113.4	110.5		
14	116.1	13.5	114.7- 117.4	115.8		
15	114.6	12.9	113.3- 115.9	113.0		
16	117.3	13.5	115.8- 118.7	116.0		
17	118.9	14.7	116.8- 121.1	118.0		
Ethnicity						
Malay	113.5	13.2	112.6- 114.4	112.5	3	0.000*
Chinese	119.9	13.8	118.7- 121.1	118.5		
Indian	112.3	12.8	110.8- 113.8	112.0		
Others	111.4	13.1	109.3- 113.4	109.5		

S.D = Standard deviation, CI= Confidence interval, df= degree of freedom, *= significant at p< 0.05

Table IV : Mean SBP according to age and gender.

Gender/Age(Years)	n	Systolic Blood Pressure (mmHg)					
		Mean	95% CI	Median	SD	df	p
Male							
12	64	108.8	105.5- 112.1	105.8	13.3	5	0.000*
13	179	112.9	110.8- 115.0	110.5	14.1		
14	185	119.9	117.9- 121.9	118.5	13.7		
15	157	117.8	115.5- 120.1	117.0	14.4		
16	144	121.6	119.4- 123.8	121.5	13.2		
17	91	123.7	120.6- 126.8	123.0	15.0		
Total	820	117.8	116.8- 118.8	117.0	14.6		
Female							
12	105	110.7	108.2- 113.1	111.0	12.6	5	0.128
13	209	111.4	109.7- 113.1	111.0	12.4		
14	205	112.7	110.9- 114.4	111.5	12.4		
15	218	112.3	110.8- 113.8	110.3	11.3		
16	202	114.2	112.4- 116.0	113.8	12.9		
17	98	114.6	111.9- 117.2	112.5	13.1		
Total	1037	112.6	111.8- 113.4	111.5	12.4		
Both Gender							
12	169	110.0	108.0- 111.9	109.0	12.8	5	0.000*
13	388	112.1	110.8- 113.4	110.5	13.2		
14	390	116.1	114.7- 117.4	115.8	13.5		
15	375	114.6	113.3- 115.9	113.0	12.9		
16	346	117.3	115.8- 118.7	116.0	13.5		
17	189	118.9	116.8- 121.1	118.0	14.7		
Total	1857	114.9	114.3- 115.5	114.0	13.6		

S.D = Standard deviation, CI= Confidence interval, df= degree of freedom, *= significant at p< 0.05

Table V : Mean SBP according to age and ethnicity.

Ethnicity/Age(Years)	n	Systolic Blood Pressure (mmHg)					
		Mean	95% CI	Median	SD	df	p
Malay							
12	114	110.6	108.2- 113.0	108.8	12.8	5	0.000*
13	185	110.5	108.6- 112.4	109.0	13.1		
14	180	114.7	112.8- 116.5	114.0	12.5		
15	178	113.3	111.4- 115.1	113.0	12.6		
16	159	116.6	114.6- 118.6	116.0	12.8		
17	74	116.5	113.0- 120.0	114.8	15.2		
Total	890	113.5	112.6- 114.4	112.5	13.2		
Chinese							
12	22	113.9	108.7- 119.0	113.5	11.6	5	0.003*
13	102	117.6	115.1- 120.0	117.5	12.5		
14	126	121.4	119.0- 123.9	120.0	13.8		
15	105	117.8	115.3- 120.4	115.5	13.2		
16	98	121.0	118.0- 123.9	120.5	14.7		
17	62	124.5	120.9- 128.2	121.8	14.4		
Total	515	119.9	118.7- 121.1	118.5	13.8		
Indian							
12	18	106.0	99.9- 112.2	105.5	12.3	5	0.053
13	45	110.1	106.0- 114.1	110.5	13.5		
14	56	110.2	106.8- 113.6	109.3	12.7		
15	61	112.5	109.5- 115.4	110.5	11.5		
16	64	114.7	111.6- 117.8	115.0	12.5		
17	47	115.6	111.8- 119.5	113.0	13.2		
Total	291	112.3	110.8- 113.8	112.0	12.8		
Others							
12	15	104.4	96.9- 111.9	99.5	13.5	5	0.038*
13	56	108.7	105.5- 112.0	106.3	12.0		
14	28	112.8	108.1- 117.5	114.0	12.1		
15	31	115.4	109.9- 120.8	110.5	14.9		
16	25	113.4	108.1- 118.7	113.0	12.8		
17	6	117.5	108.9- 125.9	116.8	8.1		
Total	161	111.4	109.3- 113.4	109.5	13.1		

S.D = Standard deviation, CI= Confidence interval, df= degree of freedom, *= significant at $p < 0.05$

Table VI : Mean DBP of the respondents.

Factors	Mean	S.D	95% CI	Median	df	p
Overall	65.1	8.5		64.5		
Gender						
Male	64.6	8.8	64.0- 65.2	64.5	1	0.050
Female	65.5	8.3	65.0- 66.0	64.5		
Age						
12	61.9	7.7	60.8- 63.2	62.0	5	0.000*
13	63.5	8.6	62.7- 64.4	63.0		
14	65.3	8.5	64.5- 66.2	65.0		
15	65.3	7.9	64.5- 66.1	65.0		
16	66.7	8.8	65.7- 67.6	66.0		
17	67.8	8.6	66.6- 69.1	66.5		
Ethnicity						
Malay	64.5	8.1	64.0- 65.0	64.3	3	0.000*
Chinese	68.2	8.9	67.5- 69.0	67.5		
Indian	62.8	7.6	61.9- 63.7	62.0		
Others	63.0	8.8	61.6- 64.3	63.0		

S.D = Standard deviation, CI= Confidence interval, df= degree of freedom, *= significant at $p< 0.05$

Table VII : Mean DBP according to age and gender.

Gender/Age(Years)	n	Diastolic Blood Pressure (mmHg)					
		Mean	95% CI	Median	SD	df	p
Male							
12	64	60.5	58.5- 62.4	59.5	7.8	5	0.000*
13	179	62.2	61.0- 63.5	61.5	8.5		
14	185	65.2	63.9- 66.4	65.0	8.9		
15	157	64.4	63.1- 65.7	65.0	8.2		
16	144	66.9	65.4- 68.4	67.0	9.1		
17	91	68.1	66.4- 69.8	67.5	8.2		
Total	820	64.6	64.0- 65.2	64.5	8.8		
Female							
12	105	62.9	61.4- 64.4	63.5	7.5	5	0.002*
13	209	64.6	63.5- 65.8	63.5	8.6		
14	205	65.5	64.4- 66.6	65.0	8.2		
15	218	65.9	64.9- 66.9	64.5	7.6		
16	202	66.5	65.3- 67.7	65.5	8.5		
17	98	67.6	65.8- 69.4	66.0	9.0		
Total	1037	65.5	65.0- 66.0	64.5	8.3		
Both Gender							
12	169	62.0	60.8- 63.2	62.0	7.7	5	0.000*
13	388	63.5	62.7- 64.4	63.0	8.6		
14	390	65.3	64.5- 66.2	65.0	8.5		
15	375	65.3	64.5- 66.1	65.0	7.9		
16	346	66.7	65.7- 67.6	66.0	8.8		
17	189	67.8	66.6- 69.1	66.5	8.6		
Total	1857	65.1	64.8- 65.5	64.5	8.5		

S.D = Standard deviation, CI= Confidence interval, df= degree of freedom, *= significant at $p< 0.05$

Table VIII : Mean DBP according to age and ethnicity.

Ethnicity/Age(Years)	n	Diastolic Blood Pressure (mmHg)					
		Mean	95% CI	Median	SD	df	p
Malay							
12	114	62.3	60.9- 63.8	61.5	7.8	5	0.000*
13	185	63.5	62.3- 64.7	63.0	8.4		
14	180	64.3	63.1- 65.4	64.5	7.8		
15	178	64.2	63.2- 65.3	64.5	7.1		
16	159	66.6	65.3- 67.9	65.5	8.1		
17	74	67.2	65.2- 69.3	65.8	8.9		
Total	890	64.5	64.0- 65.0	64.3	8.1		
Chinese							
12	22	65.4	62.7- 68.1	65.5	6.1	5	0.029*
13	102	66.2	64.6- 67.8	65.0	8.0		
14	126	68.4	66.8- 70.0	68.3	9.2		
15	105	68.3	66.7- 69.9	67.0	8.3		
16	98	69.2	67.2- 71.1	69.0	9.7		
17	62	70.5	68.1- 72.9	70.0	9.5		
Total	515	68.2	67.5- 69.0	67.5	8.9		
Indian							
12	18	58.0	54.4- 61.6	56.3	7.2	5	0.007*
13	45	61.1	58.8- 63.4	60.5	7.7		
14	56	62.6	60.6- 64.6	62.0	7.5		
15	61	62.8	61.0- 64.6	62.0	6.9		
16	64	63.9	61.7- 66.0	63.8	8.6		
17	47	65.1	63.4- 66.9	65.0	5.9		
Total	291	62.8	61.9- 63.7	62.0	7.6		
Others							
12	15	59.3	55.4- 63.2	59.5	7.0	5	0.019*
13	56	60.7	58.1- 63.3	60.0	9.8		
14	28	63.8	60.8- 66.7	65.3	7.6		
15	31	65.9	62.6- 69.2	65.5	9.1		
16	25	64.4	61.6- 67.1	66.0	6.6		
17	6	68.7	61.5- 75.9	67.8	6.9		
Total	161	63.0	61.6- 64.3	63.0	8.8		

S.D = Standard deviation, CI= Confidence interval, df= degree of freedom, *= significant at $p< 0.05$

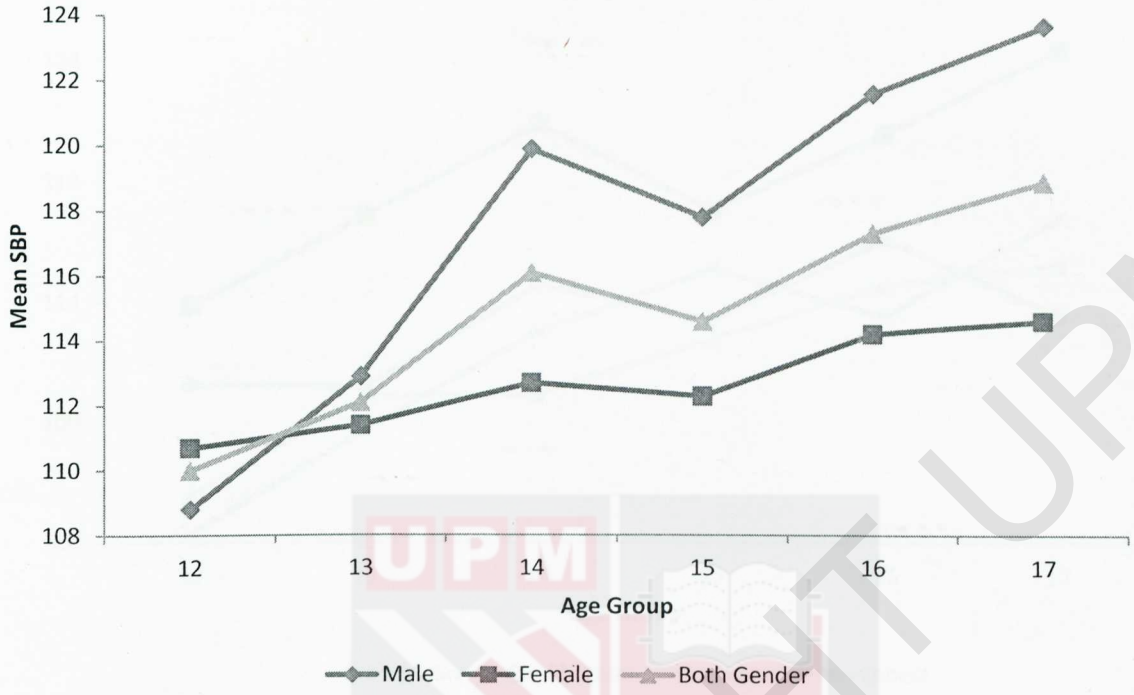


Figure 4.2 Mean SBP by age group for gender.

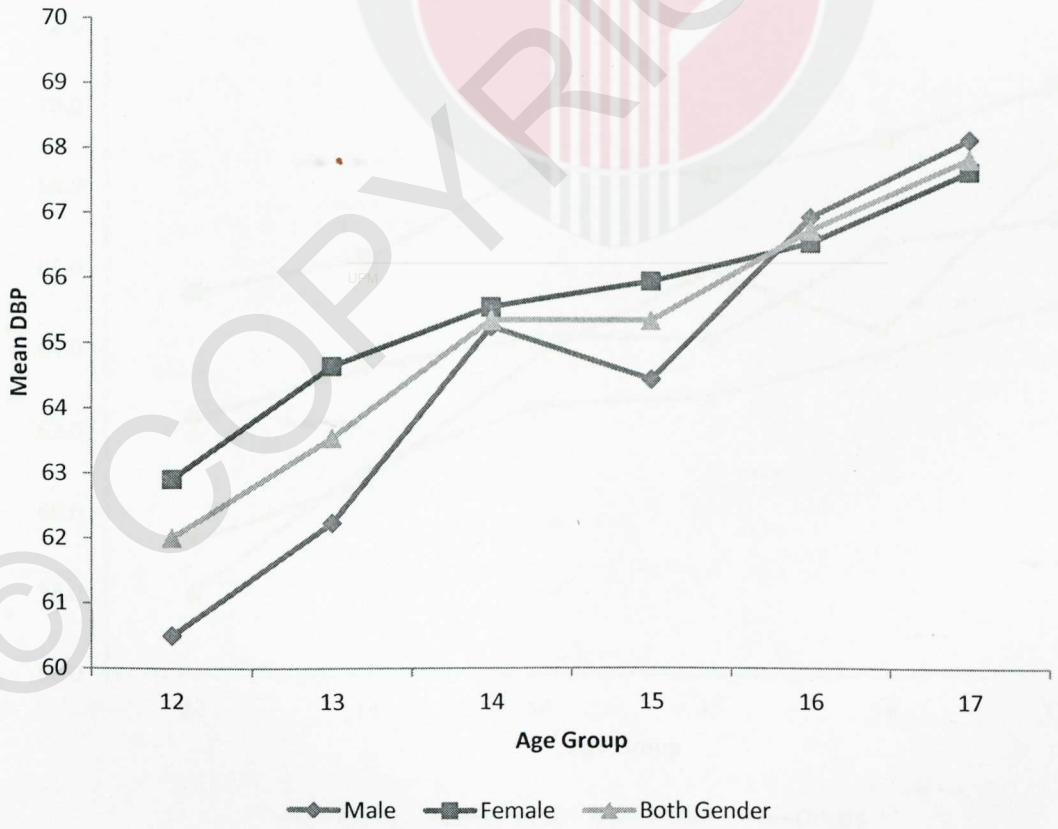


Figure 4.3 Mean DBP by age group for gender.

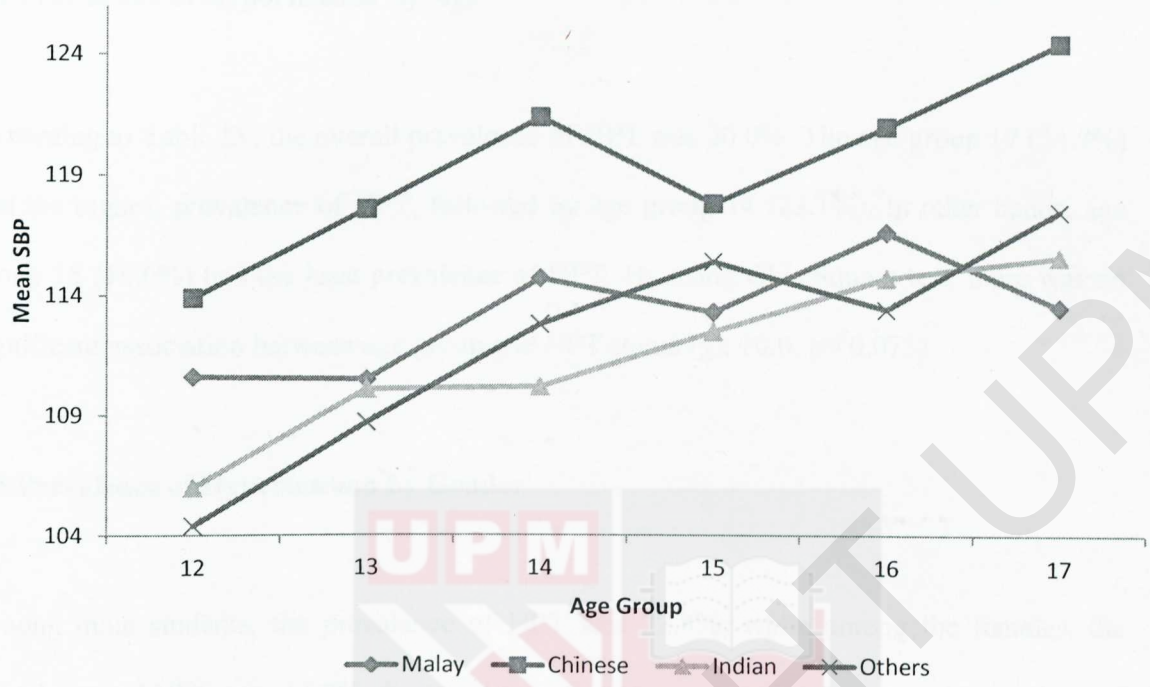


Figure 4.4 Mean SBP by age group for ethnicity.

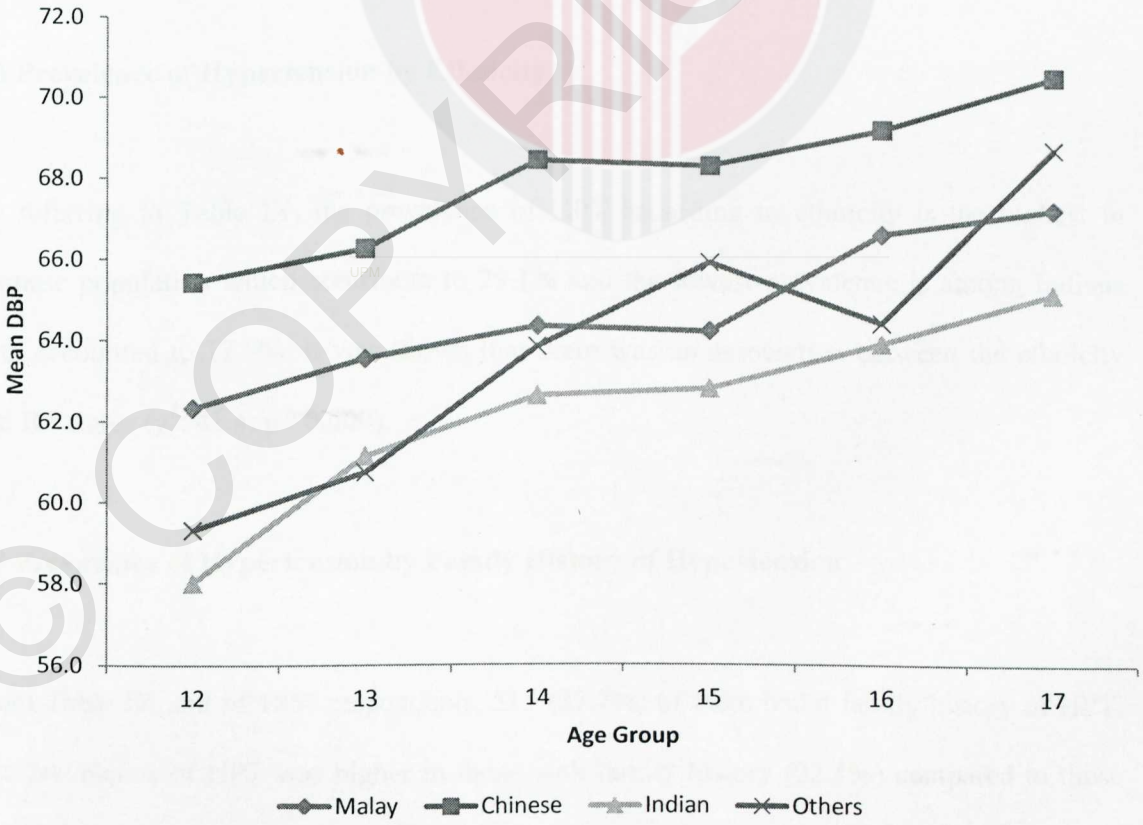


Figure 4.5 Mean DBP by age group for ethnicity.

4.4 Prevalence of Hypertension by Age

According to Table IX, the overall prevalence of HPT was 20.0%. The age group 17 (24.9%) had the highest prevalence of HPT, followed by age group 14 (23.1%). In other hands, age group 15 (16.0%) had the least prevalence of HPT. By using Chi- Square test, there was no significant association between age group and HPT status (χ^2: 10.0, p= 0.075).

4.5 Prevalence of Hypertension by Gender

Among male students, the prevalence of HPT was 22.8%; while among the females, the prevalence of HPT was 17.7%. It was shown that the association between the gender and HPT status was significant. (χ^2: 7.3, p= 0.007).

4.6 Prevalence of Hypertension by Ethnicity

By referring to Table IX, the prevalence of HPT according to ethnicity is the highest in Chinese population which contribute to 29.1% and the lowest prevalence is among Indians with accounted to 12.7%. It was shown that there was an association between the ethnicity and BP status (χ^2: 41.4, p= 0.000).

4.7 Prevalence of Hypertension by Family History of Hypertension

From Table IX, out of 1857 respondents, 515 (27.7%) of them had a family history of HPT. The prevalence of HPT was higher in those with family history (22.1%) compared to those

with no family history of HPT (19.2%). However, the association between HPT and family history of HPT is not significant (χ^2: 2.1, p= 0.15).

4.8 Prevalence of Hypertension by Physical Activity Level

The prevalence of HPT among those who with low physical activity was higher (20.1%) compared to those who with high physical activity (19.3%). However, it was shown that the association between physical activity level and HPT status was not significant (χ^2: 0.1, p= 0.744).

4.9 Prevalence of Hypertension by Smoking Status

The prevalence of HPT was higher among those who never smoking (20.5%), as compared to those who ever smoking was only 13.7%. In spite of that, association between smoking status and HPT status was significant (χ^2: 3.9, p= 0.048). Among HPT, 94.6% of them were those who never smoke.

4.10 Prevalence of Hypertension by Nutritional Status

The prevalence of HPT was increased down the nutritional status, in which from underweight (6.6%) until obese (56.8%). Among those who with normal nutritional status, only 10.8% of getting HPT, while 36.5% for overweight nutritional status. Significant association was shown between nutritional status and HPT status (χ^2: 323.3, p= 0.000). The highest percentage of HPT was found on normal group and obese group.

4.11 Prevalence of Hypertension by Age in Male and Female

Table X shows that for male, the highest prevalence of HPT was those from the age group of 14 which was 29.7%. By referring to Table XI, the highest prevalence of HPT in female is those from the age group of 17 (21.4%) followed by those in age group of 12 (21.0%) while the lowest prevalence of HPT is among the group of 15 (14.2%). In males, significant association was shown between age group and HPT status (χ^2: 11.8, p= 0.038), while there was no significant association for females (χ^2: 4.8, p= 0.443).

4.12 Prevalence of Hypertension by Ethnicity in Male and Female

Chinese (34.8%) in male had the highest prevalence of HPT among ethnicity, while Others (9.4%) was the lowest. The prevalence of HPT in female according to ethnicity was the highest in Others (24.7%) followed by Chinese (18.6%) while the lowest was among Malay ethnic (8.8%). It was shown that the association between ethnicity and HPT status was significant for both male and female (χ^2: 29.5, p=0.000) ; (χ^2: 18.4, p=0.000).

4.13 Prevalence of Hypertension by Family History of Hypertension in Male and Female

From Table X and XI, it was shown that those with family history of HPT had higher prevalence of HPT (26.9%; 18.9%) than those with no family history of HPT (21.4% ; 17.3%). However, it was shown that the association of family history of HPT with HPT status is not significant (χ^2: 2.7, p= 0.101) ; (χ^2: 0.4, p= 0.530).

4.14 Prevalence of Hypertension by Physical Activity Level in Male and Female

The prevalence of HPT was higher in those with low level of physical activity (23.3%; 18.1%) compare to those with high level of physical activity (21.5%; 14.0%) for both male and female. However, the association between physical activity level with HPT was shown as not significant (χ^2: 0.3, p= 0.578) ; (χ^2: 1.0, p= 0.319).

4.15 Prevalence of Hypertension by Smoking Status in Male and Female

By referring to Table X and XI, the prevalence of HPT in male and female were shown as higher in those who never smoke (24.5%; 17.9%) than those who ever smoke (14.3%; 7.7%). In males, significant association was shown between smoking status and HPT status (χ^2: 6.5, p= 0.011). However, it is shown that the association between smoking status and HPT status is not significant for female (χ^2: 0.9, p= 0.340).

4.16 Prevalence of Hypertension by Nutritional Status in Male and Female

The prevalence of HPT in male and female were increased down the nutritional status from the underweight (7.7%; 4.7%) until obese (61.3%; 52.1%). Those in normal group contribute to 12.4% and 9.8% of the prevalence of HPT among male and female respectively while the overweight group is account up to 38.0% and 35.3%. It is shown that the association of nutritional status and HPT status is significant (χ^2: 157.6, p=0.000) ; (χ^2: 161.3, p=0.000).

Table IX : Association between socio-demographic factors, physical activity level, smoking status, and nutritional status and hypertension.

Factors	Hypertension status		χ^2	df	*p*
	Hypertensive	Not Hypertensive			
	n	n			
Overall	371 (20.0%)	1486 (80.0%)			
Gender					
Male	187 (22.8%)	633 (77.2%)	7.3	1	0.007*
Female	184 (17.7%)	853 (82.3%)			
Age Group					
12	31 (18.3%)	138 (81.7%)	10.0	5	0.075
13	71 (18.3%)	317 (81.7%)			
14	90 (23.1%)	300 (76.9%)			
15	60 (16.0%)	315 (84.0%)			
16	72 (20.8%)	274 (79.2%)			
17	47 (24.9%)	142 (75.1%)			
Ethnicity					
Malay	160 (18.0%)	730 (82.0%)	41.4	3	0.000*
Chinese	150 (29.1%)	365 (70.9%)			
Indian	37 (12.7%)	254 (87.3%)			
Others	24 (14.9%)	137 (85.1%)			
Family History					
Yes	114 (22.1%)	401 (77.9%)	2.1	1	0.15
No	257 (19.2%)	1085 (80.8%)			
Physical Activity Level					
Low	309 (20.1%)	1227 (79.9%)	0.1	1	0.744
High	62 (19.3%)	259 (80.7%)			
Smoking Status					
Yes	20 (13.7%)	126 (86.3%)	3.9	1	0.048*
No	351 (20.5%)	1360 (79.5%)			
Nutritional Status					
Underweight	8 (6.6%)	113 (93.4%)	323.3	3	0.000*
Normal	134 (10.8%)	1106 (89.2%)			
Overweight	95 (36.5%)	165 (63.5%)			
Obese	134 (56.8%)	102 (43.2%)			

χ^2 = Chi square, *= significant at $p < 0.05$

Table X : Prevalence of hypertension for socio- demographic factors, physical activity level, smoking status, and nutritional status in male.

Factors	Hypertension Status		χ^2	df	*p*
	Hypertensive	Non Hypertensive			
	n	n			
Age Group					
12	9 (14.1%)	55 (85.9%)	11.8	5	0.038*
13	37 (20.7%)	142 (79.3%)			
14	55 (29.7%)	130 (70.3%)			
15	29 (18.5%)	128 (81.5%)			
16	31 (21.5%)	113 (78.5%)			
17	26 (28.6%)	65 (71.4%)			
Ethnicity					
Malay	78 (20.2%)	308 (79.8%)	29.5	3	0.000*
Chinese	79 (34.8%)	148 (65.2%)			
Indian	24 (16.8%)	119 (83.2%)			
Others	6 (9.4%)	58 (90.6%)			
Family History					
Yes	56 (26.9%)	152 (73.1%)	2.7	1	0.101
No	131 (21.4%)	481 (78.6%)			
Physical Activity Level					
Low	138 (23.3%)	454 (76.7%)	0.3	1	0.578
High	49 (21.5%)	179 (78.5%)			
Smoking Status					
Yes	19 (14.3%)	114 (85.7%)	6.5	1	0.011*
No	168 (24.5%)	519 (75.5%)			
Nutritional Status					
Underweight	6 (7.7%)	72 (92.3%)	157.6	3	0.000*
Normal	62 (12.4%)	440 (87.6%)			
Overweight	46 (38.0%)	75 (62.0%)			
Obese	73 (61.3%)	46 (38.7%)			

χ^2 = Chi square, *= significant at $p < 0.05$

Table XI : Prevalence of hypertension for socio- demographic factors, physical activity level, smoking status, and nutritional status in female.

Factors	Hypertension Status		χ^2	df	*p*
	Hypertensive	Non Hypertensive			
	n	n			
Age Group					
12	22 (21.0%)	83 (79.0%)	4.8	5	0.443
13	34 (16.3%)	175 (83.7%)			
14	35 (17.1%)	170 (82.9%)			
15	31 (14.2%)	187 (85.8%)			
16	41 (20.3%)	161 (79.7%)			
17	21 (21.4%)	77 (78.6%)			
Ethnicity					
Malay	13 (8.8%)	135 (91.2%)	18.4	3	0.000*
Chinese	18 (18.6%)	79 (81.4%)			
Indian	82 (16.3%)	422 (83.7%)			
Others	71 (24.7%)	217 (75.3%)			
Family History					
Yes	58 (18.9%)	249 (81.1%)	0.4	1	0.530
No	126 (17.3%)	604 (82.7%)			
Physical Activity Level					
Low	171 (18.1%)	773 (81.9%)	1.0	1	0.319
High	13 (14.0%)	80 (86.0%)			
Smoking Status					
Yes	1 (7.7%)	12 (92.3%)	0.9	1	0.340
No	183 (17.9%)	841 (82.1%)			
Nutritional Status					
Underweight	2 (4.7%)	41 (95.3%)	161.3	3	0.000*
Normal	72 (9.8%)	666 (90.2%)			
Overweight	49 (35.3%)	90 (64.7%)			
Obese	61 (52.1%)	56 (47.9%)			

χ^2 = Chi square, *= significant at $p < 0.05$

4.17 Logistic Regression of Hypertension

Logistic Regression analysis shows that the predictors for HPT were gender, ethnicity, and obesity status. The results show that males had 1.32 times higher risk of being hypertensive as compared to females (AOR= 1.315, p= 0.047). The results also show that the Chinese students had 3.24 higher risk of being of being hypertensive as compared to Indian students (AOR= 3.242, p= 0.000). Those students who were obese had around 8 times higher risk of being of being hypertensive when compared to the non- obese group (AOR= 7.852, p=

0.000). According to Table XII, the Nagelkerke R Square shows that about 25% of the variation in the outcome variable (HPT) is explained by this logistic model.

Table XII : Logistic regression of socio- demographic factors, physical activity level, smoking status, and obese status.

Factors	B	S.E	Wald	AOR	95% CI	df	p	r^2
Gender			3.944			1	0.047*	
Female				1.000				
Male	0.274	0.138		1.315	1.004- 1.723			
Ethnicity			42.038			3	0.000*	
Indian				1.000				
Malay	0.344	0.217	2.510	1.411	0.922- 2.159	1	0.113	
Others	0.385	0.310	1.548	1.470	0.801- 2.697	1	0.213	
Chinese	1.176	0.223	27.776	3.242	2.093- 5.021	1	0.000*	
Obese Status			241.865			1	0.000*	
Non- Obese				1.000				
Obese	2.061	0.133		7.852	6.056- 10.181			
Age Group			7.441			5	0.190	
12				1.000				
13	-0.106	0.263	0.163	0.899	0.537- 1.506	1	0.687	0.254
15	-0.097	0.270	0.129	0.907	0.534- 1.541	1	0.719	
16	0.206	0.265	0.603	1.228	0.731- 2.064	1	0.438	
14	0.207	0.259	0.636	1.230	0.740- 2.044	1	0.425	
17	0.410	0.294	1.948	1.507	0.847- 2.682	1	0.163	
Family History of HPT			1.149			1	0.284	
No				1.000				
Yes	0.154	0.143		1.166	0.880- 1.545			
Physical Activity Level			0.455			1	0.500	
Low				1.000				
High	0.121	0.180		1.129	0.794- 1.605			
Smoking Status			3.060			1	0.080	
Ever				1.000				
Never	0.495	0.283		1.641	0.942- 2.857			

S.E = Standard error, df= degree of freedom, *= significant at $p< 0.05$, AOR= Adjusted odd ratio, CI= Confidence interval

CHAPTER 5: DISCUSSION AND CONCLUSIONS

5.1 DISCUSSION

According to a report by the WHO in 2009, the leading global risk for mortality in the world is HPT which accounts for 13% death worldwide.[13] A study on global burden of HPT shows that there was a total of nearly one billion people experienced HPT in 2000 which more than half of it accounted in Asia, while the number was estimated to increase up to 1.56 billion people in the year of 2025 which the Asian is estimated to contribute about 580 million of the cases.[9] In 2010, the first leading risk factor for global disease burden was high BP (7·0% [95% CI 6·2- 7·7] of global DALYs), especially in most of Asia, most of Latin America, North Africa and Middle East, and central Europe.[68] Reduce HPT would increase global life expectancy by nearly 5 years.[13]

In Malaysia, cardiovascular disease has been the leading cause of death for the past 40 years.[16] Among countries in the Southeast Asian region, the prevalence of HPT in Malaysia (42.2%)[18] is the highest compared to Singapore (26.6%), Indonesia (23.0%), and Thailand (20.5%).[69] National Health and Morbidity Survey 2011 includes adults aged 18 and above, showed that the prevalence of HPT was 32.7%.[18] In Malaysia, it has been estimated that there are 4.8 million Malaysian residents who have HPT.[70]

The prevalence of paediatric HPT worldwide is not known due to differences in defining the high BP between the regions. Based on the use of ≥ 95th percentile in defining HPT, it is expected the prevalence of HPT is about 5%. However, the prevalence is expected to be lower that 5% due to effects of accommodation and regression to mean of repeated

measures.[24] The prevalence of high BP has been rise among US children and adolescent since 1963-2002. The rising trend of the BP among them was indicated due to obesity and some include racial factor.[25]

Various studies showed that male had a higher BP.[71,79] In Katona *et al.* study, male had significantly higher SBP and DBP (ΔSBP= 11 mmHg; ΔDBP = 2 mmHg, $p< 0.001$) relative to girls.[79] In our study, the mean SBP of male [117.8 (95% CI= 116.8 - 118.8)] mmHg was slightly higher than that of female [112.6 (95% CI= 111.8 - 113.4)] mmHg. However, the mean DBP of male [64.6 (95% CI= 64 - 65.2)] mmHg was slightly lower than that of female [65.5 (95% CI= 65 - 66)] mmHg. A significant association was found between gender and mean SBP (p=0.000), but not in mean DBP (p=0.05).

The mean SBP and DBP were increased down the age group except in age group 15 where it declined slightly and same as previous age group 14 respectively. There is a statistically significant different between the mean SBP and mean DBP, and the age group (p=0.000); (p=0.000). Kaur study showed that an age associated increased in mean SBP and DBP.[72] Similar finding were also noticed in several studies.[72-78] In van der Sande *et al.* and Agyemang *et al.* studies, the mean SBP and DBP significantly increase with age for both male and female.[77-78] Our study showed that the mean SBP among male and female was increased with age except at the age of 15 where it declined. For male, there is a statistically significant different between the mean SBP and the age group (p=0.000), but there is no for female (p= 0.128). For the mean DBP among male, the reading is shown increased with age except for the age of 15 where it declined while the mean DBP among female increased with age. A significant different is found between mean DBP and age group for both male and female (p=0.000); (p= 0.002).

Aging is identified as a possible risk factor for the progression of HPT. Elevated blood level in increasing age might be due to result of aging process in organ system.[30] The risk of developing HPT increases with age. In older age groups it is more common than not; based on data from the Framingham study, the lifetime risk of HPT is estimated to be 90 percent for people with normal BP at age 55 or 65 who live to be age 80-85, respectively.[6,19]

Taken out from the previous national study, the prevalence of HPT in Malaysia increased as age increased. The prevalence of HPT for subjects aged ≥15 years old was 27.8% while the prevalence of HPT among those aged ≥30 years old had increased from 32.9% in 1996 to 40.5% in 2005.[16] Based on our study in overall subject, the age group 17 (24.9%) had the highest prevalence of HPT while the age group 15 (16.0%) had the least prevalence of HPT. However, there was no significant association between age group and HPT status (χ^2: 10.0, p= 0.075). By looking according to gender, the highest prevalence of HPT among male is those from the age group of 14 which was 29.7% with significant association was shown between age group and HPT status (χ^2: 11.8, p= 0.038), while in female is those from the age group of 17 (21.4%) with no significant association shown between age group and HPT status (χ^2: 4.8, p= 0.443).

Previous review concluded that androgen hormone may contribute to the differences of BP between genders. It is found that when the level of androgen was increasing after the onset of the puberty, boys tend to have higher BP compare to girls.[32] In a study conducted among adolescents in Hungary, it was found that boys had significantly higher SBP and DBP relative to girls. The mean difference was found to be significant for both SBP and DBP.[79] In a global study on the burden of HPT, it was found that the relation between sex and prevalence of HPT varied by world region; four regions had higher prevalence in men and

four had higher prevalence in women. It was found that at young ages the prevalence of HPT were higher in men than in women, whereas in older people they were higher in women than in men.[9]

A previous study conducted in the sub- district of Dengkil, Selangor in respondents aged 15-60 years old and above, shown that the prevalence of HPT in male is higher compared to female (31.7% ; 23.5%) with the association between the two variables is significant.[33] Comparing with other study among the adolescent in Malaysia, it is shown that the prevalence of HPT in male was higher compared to female which was 12.9% and 10.2% respectively and with the result also showed that there was significant association between HPT and gender.[23] The results in our current study also do shows that that was an association between HPT and gender (χ^2: 7.3, p= 0.007) with the prevalence of HPT in male is higher compared to female (22.8% ; 17.7%).The logistic regression between these variables shows that the gender was correlate with the HPT (AOR= 1.315, p= 0.047).

Many studies had done and shown that different ethnics accounted to different prevalence of HPT.[34-35,80] A study conducted in 2008 shown that for respondent aged ≥15 years, the Chinese had the highest prevalence of HPT which account for 30.6% followed by Malays (26.7%) and the Indians (25.1%). The indigenous people from the state of Sarawak, the Sarawak Bumiputera had a higher prevalence (31.1%) compared to other ethnic group.[16] The prevalence of HPT was the highest in Chinese population which contribute to 29.1% followed by Malay (18.0%) and Others (14.9%), while Indians with the least accounted to 12.7%. It was shown that there was an association between the ethnicity and HPT (χ^2: 41.4, p=0.000). However, some studies that had done in Malaysia show no significant association between HPT and ethnicity.[33,81]

Chinese (34.8%) in male had the highest prevalence of HPT, while Others (9.4%) was the lowest. The prevalence of HPT in female according to ethnicity is the highest in Others (24.7%) followed by Chinese (18.6%) while the lowest is among Malay ethnic (8.8%). It is shown that the association between ethnicity and HPT status is significant for both male and female (χ^2: 29.5, p=0.000) ; (χ^2: 18.4, p=0.000). Ethnicity was found to be the confounder for HPT (p= 0.000). However, only Chinese showed significant associated with HPT, which is around 3 times the occurrence of HPT when compared to Indians (AOR= 3.242, p= 0.000). There are several possible reasons including genetic, life-style, and cultural factors that influenced the BP in certain ethnicity.[82] Most of the studies found HPT to be significantly higher in Blacks than Whites.[83] Ethnicity may be an effect modifier in the association of body size with BP.[84-86] On the other hand, Harding *et al.* found only minor ethnic-specific effects for BP in a multi-ethnic cohort of United Kingdom adolescents.[87] Ethnic-specific associations were found for body size with BP. Biological (differences in body composition[88-90]), behavioural and environmental factors, including diet and nutrition (especially salt intake[91]), may play a role in an explanation for these differences.

Various studies had shown that people with family history of HPT have a risk of developing HPT.[36-37] Several studies showed significant association between HPT and family history of HPT.[92-95] A total of 29.9% people who had positive family history of HPT also had HPT [35.0% (n=86) women, 19.1% (n=22) men]. People who had positive family history 2.23 times (95%CI: 1.62- 3.07 ; p=0.000) more risk to develop HPT than who did not have this factor.[92] Therefore, a positive history of HPT in first-degree relatives causes higher risk for HPT.[92,96-98]

Compared a previous study on HPT in adolescent[23] with our current study, the prevalence of HPT was higher in those with family history (12.0% ; 22.1%) compared to those with no family history of HPT (11.3% ; 19.2%). However, the difference was not statistically significant ($p>0.05$) for both the study. For both male and female, it is shown that those with family history of HPT have higher prevalence of HPT (26.9% ; 18.9%) than those with no family history of HPT (21.4% ; 17.3%). However, it is shown that the association of family history of HPT with HPT status is not significant ($p>0.05$). Based on a screened cohort in Okinawa Japan, the chances of getting HPT is higher is those with family history of HPT with the number of first-degree family members with HPT was quantitatively correlated with the risk of developing the condition. The positive family history of HPT may lead to an increased of HPT through genetic heritability of factors associated with HPT, inheritance of susceptibility to the effects of such factors and family sharing of lifestyle.[38]

Account for the worldwide population, physical inactivity contributed for 21.5% of ischemic heart disease and 11% of ischemic stroke. It also causes 3.3% death all around the world and 19 million DALYs worldwide.[46] A study in 2007 for the US adults population stated that adults with self-reported HPT reported being less physically active than those who were not hypertensive.[47] From a study conducted among Finnish man and women in 2003, regular physical activity was associated with a significantly reduced risk of HPT in both man and women.[48] Participating in adequate physical activity helps reduce blood pressure, ensure a healthy blood profile.[99] Physical inactivity accumulate with age will raise the BP.[13]

However, in our study, the prevalence of HPT among those who with low physical activity was higher (20.1%) compared to those who with high physical activity (19.3%). In addition, it was shown that the association between physical activity level and HPT status

was not significant (χ^2: 0.1, p= 0.744). It was same go to both male and female (χ^2: 0.3, p= 0.578) ; (χ^2: 1.0, p= 0.319). In our study, female (91.0%) also significantly to have low physical activity level than male (72.2%) (χ^2: 113.637, p=0.000). According to a survey done by Beckles, male students (20.6%) are found to be significantly more likely than female students (14.1%) to be physically active for a total of at least 60 minutes per day on all 7 days during the past 7 days. Female students (82.0%) are significantly more likely than male students (72.8%) to participate in insufficient physical activity. In the 13- 15 age groups physical inactivity was higher among the females than male students.[100]

Smoking is associated to HPT.[51-52] Acute effect of smoking on vasoconstriction and increase pulse rate were shown,[101-103] while its chronic effect on BP is much less certain. Study in population- based sample of Vietnamese men in 2005 shows that those in a group, smokers had only a marginally higher prevalence of HPT than those who never smoke.[53] However, in our study, the prevalence of HPT was higher among those who never smoking (20.5%), as compared to those who ever smoking was only 13.7%. In spite of that, association between smoking status and HPT status was significant (χ2: 3.9, p= 0.048). There are some studies showed that smokers had lower BP compared with non- smokers.[104-110] There was a significant association between HPT and smoking, but the prevalence of HPT was higher in non- smokers than the smokers (p=0.000).[92] Although smokers had higher prevalence of HPT, but there was no significant association between HPT and smoking in the previous study by Mohd Yunus *et al.*(OR=1.31, 95% CI=0.87-1.98).[33]

The prevalence of HPT in male and female were shown as higher in those who never smoke (24.5% ; 17.9%) than those who ever smoke (14.3% ; 7.7%). There are significant associated 16.2% of male smokers and 1.3% of female smoker (χ2: 141.582, p=0.000). Male

smoked significant more cigarettes than the female (p=0.000).[33,111-112] The prevalence of smoking among male youths was between 17-36%[113-115] and 1-5% among females in the same group[113,116]. In the Global School Health Survey (GSHS) 2007, 10.7% of males compared to 5.3% of females smoked cigarettes on one or more days during the past 30 days.[100] However, only male shows significant association between smoking status and HPT status ($\chi 2$: 6.5, p= 0.011). Logistic regression between these variables shows that the smoking status was not correlate with the HPT (r^2: 0.247 , AOR: 1.569, p= 0.108).

Smoking will increase the blood cholesterol level directly but not BP.[13] Halimi *et al.* reported that a positive association of smoking with HPT disappeared after adjustment for body mass index (BMI) among volunteers aged 20–69 years.[117] A cross sectional study was conducted by Li *et al.* among Mongolian population to examine the effect of smoking on BP and HPT stratified by BMI. Among subjects with BMI<25 kg/m^2, adjusted mean DBP in all smokers were lower than that in non-smokers ($p<0.05$); among subjects with BMI≥25 kg/m^2, mean SBP decreased with amount of smoking and linear trend was statistically significant ($p<0.05$). Multivariate AORs of HPT for three smoking groups were all not statistically significant. Therefore, smoking was a risk factor of HPT and elevated BP cannot be supported in their study.[118] There were also some prospective studies showing that there is no significant positive association or there is a negative association between smoking and BP.[105,119-120]

Dochi *et al.* also reported a positive association of smoking with BP in a cohort including 8251 male Japanese workers in a steel company.[121] These findings implied that the relation of BP with smoking was a complex issue, because smoking itself was a social behavior associated with many other factors. Primatesta *et al.*'s study showed that light

smokers (1–9 cigarettes/day) tended to have lower BP than never smokers among women, and current smokers had significantly lower BMI than never smokers.[122] Age–gender adjusted prevalence of overweight and central obesity was lower in smokers than that in non-smokers.[118]

Obesity had become an important medical problem in children and adolescents and one of the outcome related to childhood obesity include HPT.[43] It is found that obesity accumulate with age will raise the BP.[13] The link between obesity and HPT may be partly mediated by the sympathetic nervous system hyperactivity which may include the increased in heart rate and BP variability, increased in plasma level of catecholamines and increase in peripheral sympathetic nerve traffic which all of it include the cardiovascular, neurohormonal and neural manifestations.[44] Richard in his article concluded that obesity predisposes to HPT and alters the course of hypertensive CVD.[123]

Based on a study among school children in China, the prevalence of HPT was 1.5-2.2 folds in overweight and obese children compared to normal weight children.[124] In a study carried out by Rampal *et al.* in 2005 among 3,333 school children aged 13-17 years in the Klang district showed that the prevalence of 'at risk of overweight' and 'overweight' was 11.4% and 8.2% respectively. The overall prevalence of overweight student aged 13-17 was 8.2% with a result of moderate direct significant relationship between overweight with SBP and DBP.[45] In our current study, the prevalence of HPT in male and female who are obese are 61.3% and 52.1% respectively, and those who are overweight are account up to 38.0% and 35.3% respectively with significant association of nutritional status and HPT status (χ^2: 157.6, p=0.000) ; (χ^2: 161.3, p=0.000). It is found that, obese status is one the confounder for HPT (p= 0.000) besides ethnicity and gender. There was around 8 times the occurrence of HPT in

obese group than non- obese group (AOR= 7.852, p= 0.000). The Nagelkerke R Square shows that about 25% of the variation in the outcome variable (HPT) is explained by this logistic model.

5.2 LIMITATION OF STUDY

As our study was conducted among secondary school students, "white- coat effect" cannot be avoided because the BP was taken twice at a single visit. The respondents' BP may be elevated during the measurement taken due to nervousness and anxiety on the procedure of taking blood pressure. 24-hour ambulatory measurements or self- monitored BP measurement will have a more accurate measurement by excluding any subjective or measuring biases. Besides, differences between day and night also can be evaluated. However, when the study is done on a large population, this may not be practical.

5.3 RECOMMENDATION

By looking at our study, the state of HPT already existed among secondary school student. Thus, this condition must be managed properly to avoid the condition from worsening as they get older. First, we recommend that the higher authorities such as Ministry of Health and Ministry of Education to collaborate together and finding a way to increase the awareness of the importance of having the knowledge on BP and HPT among school children. This will help the student to increase their understanding on HPT in early stages of their life and hopefully encourage them to lead a healthy lifestyle. The existing module on physical activity and dietary programme could also be improved from time to time by referring to new study and research that conclude on a better module to be implemented in school environment.

At the school level, The Parent Teachers Association should fully make use of their connection with each other in planning a way to gain the student interest in continuing to practice the given health module even when the students are at their respective home. They can also discuss about planning a yearly program together on the health learning and practice as they understand the student better and have more insight on what the student are dealing with everyday. Lastly, routine BP measurements should be carried out every year at school. Those students who are diagnosed as pre-hypertensive and hypertensive should be referred to physician for a proper management of their condition.

5.4 CONCLUSION

The overall prevalence of HPT was 20.0%. By using Chi- Square test, there was no significant association between age group and HPT status (χ^2: 10.0, p= 0.075). Male students (22.8%) had higher the prevalence of HPT than females (17.7%). It was shown that the association between the gender and HPT status was significant. (χ^2: 7.3, p= 0.007). Chinese (29.1%) had the highest prevalence of HPT, while Indians (12.7%) the least. It was shown that there was an association between the ethnicity and BP status (χ^2: 41.4, p= 0.000). It was shown that the association between ethnicity and HPT status was significant for both male and female (χ^2: 29.5, p=0.000) ; (χ^2: 18.4, p=0.000). The prevalence of HPT was higher in those with family history (22.1%) compared to those with no family history of HPT (19.2%). However, the association between HPT and family history of HPT is not significant (χ^2: 2.1, p= 0.15). The prevalence of HPT among those who with low physical activity was higher (20.1%) compared to those who with high physical activity (19.3%). However, it was shown that the association between physical activity level and HPT status was not significant (χ^2: 0.1, p= 0.744). The prevalence of HPT was higher among those who never smoking (20.5%), as

compared to those who ever smoking was only 13.7%. In spite of that, association between smoking status and HPT status was significant (χ^2: 3.9, p= 0.048). In males, significant association was shown between smoking status and HPT status (χ^2: 6.5, p= 0.011). However, it is shown that the association between smoking status and HPT status is not significant for female (χ^2: 0.9, p= 0.340). The prevalence of HPT was increased down the nutritional status, in which from underweight (6.6%) until obese (56.8%). Significant association was shown between nutritional status and HPT status (χ^2: 323.3, p=0.000).

Out of seven variables that being studied, only gender, ethnicity and obese status were found to be the predictors of HPT. In gender, males had 1.32 times higher risk of being hypertensive as compared to females (AOR= 1.315, p= 0.047). Chinese students had 3.24 higher risk of being of being hypertensive as compared to Indian students (AOR= 3.242, p= 0.000). Those students who were obese had around 8 times higher risk of being of being hypertensive when compared to the non- obese group (AOR= 7.852, p= 0.000). The Nagelkerke R Square shows that about 25% of the variation in the outcome variable (HPT) is explained by this logistic model.

REFERENCES

1. World Health Organization. *Global status report on non- communicable diseases 2010.* Geneva, Switzerland: World Health Organization; 2011.
2. Whelton PK, He J, Muntner P. Prevalence, awareness, treatment and control of hypertension in North America, North Africa and Asia. *Journal of Human Hypertension.* 2004; 18(8): 545-551.
3. Wolf-Maier K, Cooper RS, Banegasm JR, et al. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *The Journal of the American Medical Association.* 2003; 289: 2363-2369.
4. Hypertension Guideline Working Group. *Clinical Practice Guidelines Management of Hypertension (3rd Edition).* Ministry of Health Malaysia. http://www.moh.gov.my/v/cd. Last updated February, 2008. Accessed April 10, 2013.
5. Lee DS, Massaro JM, Wang TJ, et al. Antecedent blood pressure, body mass index, and the risk of incident heart failure in later life. *Hypertension.* 2007; 50(5): 869–876.
6. Cutler JA, Sorlie PD, Wolz M, et al. Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988-1994 and 1999-2004. *Hypertension.* 2008; 52(5):818-827.
7. Fields LE, Burt VL, Cutler JA, et al. The burden of adult hypertension in the United States 1999 to 2000: A rising tide. *Hypertension.* 2004; 44(4):398-404.
8. Gu D, Reynolds K, Wu X, et al. Prevalence, awareness, treatment, and control of hypertension in China. *Hypertension.* 2002; 40(6):920-927.
9. Kearney PM, Whelton M, Reynolds K, et al. Global burden of Hypertension: Analysis of worldwide data. *Lancet.* 2005; 365(9455):217-223.
10. Lawes CM, Vander Hoorn S, Rodgers A. Global burden of blood-pressure-related disease, 2001. *Lancet.* 2008; 371(9623):1513-1518.
11. Lewington S, Clarke R, Qizilbash N, et al. Age-specific relevance of usual blood pressure to vascular mortality: A meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet.* 2002; 14(360) :1903-1913.
12. Ezzati M, Lopez AD, Rodgers A, et al. Selected major risk factors and global and regional burden of disease. *Lancet.* 2002; 360: 1347–1360.

13. World Health Organization. *Global health risks: Mortality and burden of disease attributable to selected major risks*. Geneva, Switzerland: World Health Organization; 2009.
14. Whitworth JA, World Health Organization, International Society of Hypertension Writing Group. World Health Organization/ International Society of Hypertension statement on management of hypertension 2003. *Journal of Hypertension*. 2003; 21: 1983–1992.
15. Institute for Public Health. *Second National Health and Morbidity Survey 1996*. Kuala Lumpur, Malaysia: Ministry of Health Malaysia; 1997.
16. Rampal L, Rampal S, Azhar MZ, et al. Prevalence, awareness, treatment and control of hypertension in Malaysia: A national study of 16,440 subjects. *Journal of the Royal Institute of Public Health*. 2008; 122: 11–18.
17. Institute for Public Health. *Third National Health and Morbidity Survey 2006*. Kuala Lumpur, Malaysia: Ministry of Health Malaysia; 2008.
18. Institute for Public Health. Hypertension. In: *National Health and Morbidity Survey 2011 (NHMS 2011). Vol. II: Non- Communicable Diseases*. Kuala Lumpur, Malaysia: Ministry of Health Malaysia; 2011: 21.
19. Vasan RS, Larson MG, Leip EP, et al. Impact of high-normal blood pressure on the risk of cardiovascular disease. *The New England Journal of Medicine*. 2011; 345: 1291-1297.
20. Nelson MJ, Ragland DR, Syme SL. Longitudinal prediction of adult blood pressure from juvenile blood pressure levels. *American Journal of Epidemiology*. 1992; 136: 633-645.
21. Bao W, Threefoot SA, Srinivasan SR, et al. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood. *American Journal of Hypertension*. 1995; 8: 657-665.
22. Newman WP, Freedman DJ, Voors AW, et al. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis: the Bogalusa Heart Study. *The New England Journal of Medicine*. 1986; 314: 138-144.
23. Rampal L, Ng KC, Nur Izzati I, et al. Prevalence of hypertension among Malay adolescents in Putrajaya secondary schools, Malaysia, 2010. *Malaysian Journal of Medicine and Health Sciences*. 2011; 7(2): 53-60.
24. Falkner B. Hypertension in children and adolescents: epidemiology and natural history. *Pediatric Nephrology*. 2010; 25(7): 1219–1224.
25. Dzietham DR, Liu Y, Bielo MV, et al. High Blood pressure trend in children and adolescents in national surveys, 1963 to 2002. *Circulation*. 2007; 116(13):1488-1496.

26. Hansen ML, Gunn PW, Kaelber DC. Underdiagnosis of Hypertension in Children and Adolescents. *JAMA*. 2007; 298(8):874-879.

27. Chiolero A, Madeleine G, Gabriel A, et al. Prevalence of elevated blood pressure and association with overweight in children of a rapidly developing country. *Journal of Human Hypertension*. 2007; 21:120-127.

28. Urrutia-Rojas X, Egbuchunam CE, Sejong B, et al. High blood pressure in school children: prevalence and risk factors. *BMC Pediatrics*. 2006; 6:32.

29. National Heart, Lung, and Blood Institute. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Journal of Pediatrics*. 2004; 114:555-576.

30. Fukutomi M, Kario K. Aging and Hypertension. *Expert Review of Cardiovascular Therapy*. 2010; 8(11):1531-1539.

31. Kaur G, Hiong TG, Feisul M, et al. The epidemiology of hypertension in Malaysia: current status. Paper presented at: 15th National Institutes of Health Scientific Meeting incorporating NHMS 2011 and GATS conference; June 12-14, 2012; Subang Jaya, Selangor.

32. Reckelhoff JF. Gender differences in the regulation of blood pressure. *Hypertension*. 2001; 2037:1199 –1208.

33. Mohd Yunus A, Sherina MS, Nor Afiah AZ, et al. The prevalence of hypertension and smoking in the subdistrict of Dengkil, Selangor. *Malaysian Journal of Public Health Medicine*. 2003; 3(2):5-9.

34. Brown MJ. Hypertension and Ethnic Group 2006. *British Medical Journal*. 2006; 332(7545): 833–836.

35. Sorof JM, Lai D, Turner J, et al. Overweight, ethnicity, and the prevalence of hypertension in school-aged children. *Pediatrics*. 2004; 113(3):475-482.

36. Anand NK, Tandon L. Prevalence of hypertension in school going children. *Indian Pediatrics*. 1995; 33: 377-381.

37. van der Sande Marianne AB, Walraven Gijs EL, Milligan Paul JM, et al. Family history: an opportunity for early interventions and improved control of hypertension, obesity and diabetes. *Bulletin of the World Health Organization*. 2001; 79(4): 321-328.

38. Tozawa M, Oshiro S, Iseki C, et al. Family history of hypertension and blood pressure in a screened cohort. *Hypertension Research*. 2001; 24(2): 93-98.

39. World Health Organization. *The World Health Report 2002: Reducing Risks, Promoting Healthy Life*. Geneva, Switzerland: World Health Organization; 2002.

40. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on Blood Pressure of Reduced Dietary Sodium and the Dietary Approaches to Stop Hypertension (DASH) Diet. *The New England Journal of Medicine*. 2001; 344:3-10.
41. Mustacchi P. Stress and Hypertension. *The Western Journal of Medicine*. 1990; 153:180-185.
42. Matthews KA, Katholi CR, McCreath H, et al. blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation*. 2004; 110:74-78.
43. Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations. The maternal and child health bureau, health resources and services administration and the department of health and human services. *Pediatrics*. 1998; 102:E29.
44. Sorof J, Daniels S. Obesity hypertension in children: a problem of epidemic proportions. *Hypertension*. 2002; 40:441-447.
45. Rampal L, Sherina MS, Rampal S, et al. Prevalence of overweight among secondary school students in Klang district, Selangor. *Malaysian Journal of Nutrition*.2007; 13: 1-8.
46. World Health Organization. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Geneva, Switzerland: World Health Organization; 2004.
47. Churilla JR, Ford ES. Comparing physical activity patterns of hypertensive and nonhypertensive US Adults. *American Journal of Hypertension*. 2010; 23(9): 987-993.
48. Gang H, Barengo NC, Tuomilehto J, et al. Relationship physical activity of and body mass index to the risk of hypertension: a prospective study in Finland. *Hypertension*. 2004; 43:25-30.
49. Ketelhut R, Losem CJ, Messerli FH. Is a decrease in arterial pressure during long-term aerobic exercise caused by a fall in cardiac pump function?. *American Heart Journal*. 1994; 127(3):567–571.
50. Bond VJr, Franks BD, Tearney RJ, et al. Exercise blood pressure response and skeletal muscle vasodilator capacity in normotensives with positive and negative family history of hypertension. *Journal of Hypertension*. 1994; 12(3):285–290.
51. Kim JW, Park CG, Hong SJ, et al. Acute and chronic effects of cigarette smoking on arterial stiffness. *Blood Pressure*. 2005; 14:80–85.

52. Bazzano LA, He J, Muntner P, et al. Relationship between cigarette smoking and novel risk factors for cardiovascular disease in the United States. *Annals of Internal Medicine*. 2003; 138:891–897.
53. Thuya AB, Leigh Blizzard L, Schmidt MD, et al. The association between smoking and hypertension in a population-based sample of Vietnamese men. *Journal of Hypertension*. 2010; 28:245–250.
54. United States Department of Health, Education, and Welfare. *Public health service: Smoking and health: A report of the surgeon general. DHEW Publication No (PHS) 79-50066*. Washington, DC: Government Printing Office, 1979, pp 4-57.
55. Sun SS, Grave GD, Siervogel RM, et al. Systolic blood pressure in childhood predicts hypertension and metabolic syndrome later in life. *Pediatrics*. 2007; 119(2): 237-246.
56. Aday LA, Cornelius LJ. *Designing and Conducting Health Survey*. California, San Francisco: Jossey-Bas, 2006.
57. Kowalski KC, Crocker PRE, Donen RM. *The Physical Activity Questionnaire for Older Children (PAQ-C) and Adolescents (PAQ-A) Manual*. Canada, Saskatchewan: College of Kinesiology, 2004.
58. The IPAQ group. *International Physical Activity Questionnaire (October 2002) Long Last 7 Days Self- Administered Format for Use with Young and Middle- Aged Adults (15-69 Years)*. International Physical Activity Questionnaire. https://sites.google.com/site/theipaq/questionnaire_links. Last updated October, 2002. Accessed April 16, 2013.
59. Sharma BK, Sagar S, Wahi PL, et al. Blood pressure in schoolchildren in Northwest India. *American Journal of Epidemiology*. 1991; 134: 1417-1426.
60. Mourad A, Carney S, Gillies A, et al. Arm position and blood pressure: a risk factor for hypertension?. *Journal of Human Hypertension*. 2003; 17:389-395.
61. Netea RT, Lenders JW, Smits P, et al. Both body and arm position significantly influence blood pressure measurement. *Journal of Human Hypertension*. 2003; 17:459-462.
62. Butani L, Morgenstern BZ. Are pitfalls of oxcillometric blood pressure measurements preventable in children?. *Pediatric Nephrology*. 2003; 18: 313–318.
63. World Health Organization. *The WHO Reference 2007*. World Health Organization. http://www.who.int/growthref/en/. Last updated 2007. Accessed April 22, 2013.
64. Centers for Disease Control and Prevention (CDC). *Healthy Weight - it's not a diet, it's a lifestyle!*. Centers for Disease Control and Prevention (CDC).

http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html. Last updated September 13, 2011. Accessed April 22, 2013.

65. de Onis M, Onyango AW, Borghi E, et al. Development of a WHO growth reference for school-aged children and adolescents. *Bulletin of the World Health Organization*. 2007; 85:660–667.
66. Centers for Disease Control and Prevention (CDC). *Disability and Health Data System (DHDS)-Health Topic Data Guide: Smoking Status*. Centers for Disease Control and Prevention (CDC). http://dhds.cdc.gov/guides/healthtopics/indicator?i=smokingstatus. Last updated February 6, 2013. Accessed July 29, 2013.
67. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: Methods and development. National Center for Health Statistics. *Vital and Health Statistics*. 2002; 11(246): 50-51.
68. Stephen Lim S, Theo V, Abraham DF, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990—2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*. 2012; 380(9859): 2224-2260.
69. Patricia MK, Megan W, Kristi R, et al. Worldwide Prevalence of Hypertension: A Systematic Review. *Journal of Hypertension*. 2004; 22(1):11-19.
70. Gazianoa TA, Bitton A, Anand S, et al. The Global Cost of Nonoptimal Blood Pressure. *Journal of Hypertension*. 2009; 27(7): 1472–1477.
71. de Munter JSL, Agyemang C, van Valkengoed IG, et al. Sex difference in blood pressure among South Asian diaspora in Europe and North America and the role of BMI: a meta-analysis. *Journal of Human Hypertension*. 2011; 25: 407- 417.
72. Kaur M. Blood Pressure Trends and Hypertension among Rural and Urban Jat Women of Haryana, India. *Collegium Antropologicum*. 2012; 36(1): 139- 144.
73. Benjamin AI, Cade J, Cowan B, et al. Blood Pressure Profile and Prevalence of Hypertension in Punjabi Women. *Health and Population – Perspectives and Issues*. 1995; 18(1): 1- 11.
74. Kaur J. Correlation of biophysical and psychosocial factors with blood pressure among urban and rural jat sikh women. *Nursing and Midwifery Research Journal*. 2007; 3(1): 20- 29.
75. Sidhu S, Badaruddoza, Kaur A. Blood Pressure in Rural and Urban Adult Healthy Females of Jat Sikh Community in Puniab, North India: An Epidemiologic Profile. *Medical Journal of Malaysia*. 2004; 59(2): 233- 241.

76. Singal P, Sethi HK, Sharma M. Changes in blood pressure and some related variables with ageing in rural and urban Jat Sikh females. *Human Growth – A Multidisciplinary Approach*. 1999; 87-94.

77. van der Sande MA, Milligan PJ, Nyan OA, et al. Blood pressure patterns and cardiovascular risk factors in rural and urban gambian communities. *Journal of Human Hypertension*. 2000; 14(8): 489- 496.

78. Agyemang C, William KR, Ellis OD, et al. Blood pressure patterns in rural, semi- urban and urban children in the Ashanti region of Ghana, West Africa. *BMC Public Health*. 2005; 5:114.

79. Katona E, Zrinyi M, Lengyel S, et al. The prevalence of adolescent hypertension in Hungary – The Debrecen Hypertension Study. *Blood Pressure*. 2011; 20: 134–139.

80. Hoog MLAD, Eijsden MV, Stronks K, et al. Association between Body Size and Blood Pressure in Children from Different Ethnic Origins. *Cardiovascular Diabetology*. 2012; 11:136.

81. Kandiah N, Rampal L, Paranjothy S, et al. A Community- based Study on the Epidemiology of Hypertension in Selangor. *Medical Journal of Malaysia*. 1980; 34: 211-220.

82. Sorel JE, Ragland DR, Syme SL. Blood pressure in Mexican Americans, whites, and blacks. The Second National Health and Nutrition Examination Survey and the Hispanic Health and Nutrition Examination Survey. *American Journal of Epidemiology*. 1991; 134(4): 370-378.

83. Kurian AK, Cardarelli KM. Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. *Ethnicity and Disease*. 2007; 17(1): 143-152.

84. Rosner B, Prineas R, Daniels SR, et al. Blood pressure differences between blacks and whites in relation to body size among US children and adolescents. *American Journal of Epiedemiology*. 2009; 151:1007–1019.

85. Gopinath B, Baur LA, Garnett S, et al. Body mass index and waist circumference are associated with blood pressure in preschool-aged children. *Annals of Epidemiology*. 2011; 21:351–357.

86. Ke L, Brock KE, Cant RV, Li Y, Morrell SL. The relationship between obesity and blood pressure differs by ethnicity in Sydney school children. *American Journal of Hypertension*. 2009; 22:52–58.

87. Harding S, Whitrow M, Lenguerrand E, et al. Emergence of ethnic differences in blood pressure in adolescence: the determinants of adolescent social well-being and health study. *Hypertension.* 2010; 55:1063–1069.

88. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis among different ethnic groups. *International journal of obesity and related metabolic disorders.* 1998; 22:1164–1171.

89. Nightingale CM, Rudnicka AR, Owen CG, et al. Patterns of body size and adiposity among UK children of South Asian, black African-Caribbean and white European origin: Child Heart And health Study in England (CHASE Study). *International Journal of Epidemiology.* 2011; 40:33–44.

90. Ujcic-Voortman JK, Bos G, Baan CA, et al. Obesity and body fat distribution: ethnic differences and the role of socio-economic status. *Obesity Facts.* 2011; 4:53–60.

91. He FJ, Macgregor GA. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. *Hypertension.* 2006; 48:861–869.

92. Dogan N, Toprak D, Demir S. Hypertension prevalence and risk factors among adult population in Afyonkarahisar region: a cross sectional research. *The Anatolian Journal of Cardiology.* 2012; 12: 47-52.

93. Reddy SS, Prabhu GR. Prevalence and risk factors of hypertension in adults in an urban Slum, Tirupati, A.P. *Indian Journal of Community Medicine.* 2005; 30: 84-86.

94. Stein K, Bradshaw D, Norman R, et al. Determinants and treatment of hypertension in South Africans: the first Demographic and Health Survey. *South African Medical Journal.* 2008; 98: 376-380.

95. Goldstein IB, Shapiro D, Guthrie D. Ambulatory blood pressure and family history of hypertension in healthy men and women. *American Journal of Hypertension.* 2006; 19: 486-491.

96. Delles C, McBride MW, Graham D, et al. Genetics of hypertension: from experimental animals to humans. *Biochimica et Biophysica Acta.* 2010; 1802: 1299-1308.

97. Lee WK, Padmanabhan S, Dominiczak AF. Genetics of hypertension: from experimental models to clinical applications. *Journal of Human Hypertension.* 2000; 14: 631-647.

98. Dudley CR, Giuffra LA, Reeders ST. Identifying genetic determinants in human essential hypertension. *Journal of the American Society of Nephrology.* 1992; 3: 2-8.

99. Warburton DER, Nicol CW, Bredin SSD. Health benefits of physical activity: the evidence. *Canadian Medical Association Journal.* 2006; 174 (6): 801-809.

100.Beckles, M.P. *Global School Health Survey 2007 Tobago Report.* Tobago, Republic: Ministry of Health Republic; 2007.

101.Aronow WS, Dendinger J, Rokaw SN. Heart rate and carbon monoxide level after smoking high-, low-, and non-nicotine cigarettes. *Annals of Internal Medicine*. 1971; 74:697–702.

102.Benowitz NL, Jacob P, Jones RT, et al. Interindividual variability in the metabolism and cardiovascular effects of nicotine in man. *Journal of Pharmacology and Experimental Therapeutics*. 1982; 221:368–372.

103.Benowitz NL, Kuyt F, Jacob P. Influence of nicotine on cardiovascular and hormonal effects of cigarette smoking. *Clinical Pharmacology and Therapeutics*. 1984; 36:74–81.

104.Green MS, Jucha E, Lz Y. Blood pressure in smokers and non- smokers: Epidemiologic findings. *American Heart Journal*. 1986; 111: 932–940.

105.Okubo Y, Suwazono Y, Kobayashi E, et al. An association between smoking habits and blood pressure in normotensive Japanese men: A 5-year follow-up study. *Drug and Alcohol Dependence*. 2004; 73:167–174.

106.Rosengren A, Wilhelmsen L. Alcoholic registration and cardiovascular morbidity and mortality – A prospective study in middle aged Swedish men. *Acta Medica Scandinavica. Supplementum*. 1987; 717:87–92.

107.Imamura H, Tanaka K, Hirae C, et al. Relationship of cigarette smoking to blood pressure and serum lipids and lipo-protein in men. *Clinical and Experimental Pharmacology and Physiology*. 1996; 23:397–402.

108.Seltzer CC. Effect of smoking on blood pressure. *American Heart Journal*. 1974; 87:558–564.

109.Lee DH, Ha MH, Kim JR, et al. Effects of smoking cessation on changes in blood pressure and incidence of hypertension: A 4-year follow-up study. *Hypertension*. 2001; 37:194–198.

110.John U, Meyer C, Hanke M, et al. Smoking status, obesity and hypertension in a general population sample: A cross-sectional study. *The Quarterly journal of medicine*. 2006; 99:407–415.

111.Lim KH, Amal NM, Hanjeet K, et al. Prevalence and factors related to smoking among secondary school students in Kota Tinggi district, Johor, Malaysia. *Tropical Biomedicine*. 2006; 23(1): 75-84.

112.Rampal L, Rampal S, Azhar MZ, et al. A National Study on the Prevalence and Factors Associated with Smoking Amongst Malaysians Aged 18 Years and Above. *Malaysian Journal of Medicine and Health Sciences.* 2008; 4(2): 41-53.

113.Thambypillai V. Smoking among urban Malaysian school children. *Journal of the Malaysian Society of Health.* 1985; 21(7): 818-823.

114.Shamsuddin K, Haris MA. Family influence on current smoking habits among secondary school children in Kota Bharu, Kelantan. *Singapore Medical Journal.* 2000; 41(4): 167-171.

115.Naing NN, Ahmad Z, Musa R, et al. Factors related to smoking habits of male adolescents. *Tobacco Induces Diseases.* 2004; 2(3): 133- 140.

116.Ahmad Z, Jaafar R, Musa R. Cigarettes smoking among Malaysian youth: problem and prospects. Proceedings of the Malaysian Society of Health 21st Symposium; December 6-7, 1997; Kuala Lumpur.

117.Halimi JM, Giraudeau B, Vol S, et al. The risk of hypertension in men: Direct and indirect effects of chronic smoking. *Journal of Hypertension.* 2002; 20:187–193.

118.Li HM, Tong WJ, Wang AL, et al. Effects of cigarette smoking on blood pressure stratified by BMI in Mongolian population, China. *Blood Pressure.* 2010; 19: 92-97.

119.Nagahama K, Inoue T, Suwazono Y, et al. Hyperuricemia as a predictor of hypertension in a screened cohort in Okinawa, Japan. *Hypertension Research.* 2004; 27:835–841.

120.Wang W, Lee ET, Fabsitz RR, et al. A longitudinal study of hypertension risk factors and their relation to cardiovascular disease: The strong heart study. *Hypertension.* 2006; 47:403–409.

121.Dochi M, Sakata K, Oishi M, et al. Smoking as an independent risk factor for hypertension: A 14-year longitudinal study in male Japanese workers. *Tohoku Journal of Experimental Medicine.* 2009; 217:37–43.

122.Primatesta P, Falaschetti E, Gupta S, et al. Association between smoking and blood pressure: Evidence from the health survey for England. *Hypertension.* 2001; 37:187–193.

123.Richard NR. Obesity-Related Hypertension. *The Ochsner Journal.* 2009; 9:133–136.

124.Lu X, Shi P, Luo CY, et al. Prevalence of hypertension in overweight and obese children from a large school-based population in Shanghai, China. *BMC Public Health.* 2013; 13:24.

Appendix 1 : Gantt Chart

Activity	Weeks															
	1	2	5	12	13	14	15	16	17	18	19	20	21	22	23	24
Proposal preparation																
Submission of Proposal to Module Coordinator																
Submission of ethic form																
Data collection																
Data analysis																
Report writing																
Submission final report / scientific article																
Final presentation																
Correction of final report /scientific article & Submission of log book and final report																

Appendix 2 : Budget Planning

Questionnaire Photocopy	RM100
Proposal	RM50
Report	RM50
Binding	RM15
Hardcover	RM35
Stationary	RM10
Transportation	RM100
Total	**RM360**

Appendix 3 : Questionnaire

Identification Number: .....

UNIVERSITI PUTRA MALAYSIA

BERILMU BERBAKTI

QUESTIONNAIRE

TITLE: PREVALENCE OF HYPERTENSION AND FACTORS ASSOCIATED AMONG SECONDARY SCHOOL STUDENTS IN BATANG PADANG DISTRICT PERAK

Reminder: This is not a test or exams. There is no right or wrong answer. Please answer the questions honestly

SECTION A:

Socio-demographic Characteristics

Instructions: Choose only one answer for each question. Tick the relevant box.

S/N.	Questions and Filters	Coding categories
1	Gender	☐ Male ☐ Female
2	Date of Birth	..... Day Month Year
3	Ethnicity	☐ Malay ☐ Chinese ☐ Indian ☐ Others
4	Religion	☐ Islam ☐ Buddha ☐ Hindu ☐ Christian ☐ Others ☐ I don't have
5	Form	☐ Form 1 ☐ Form 2 ☐ Form 3 ☐ Form 4 ☐ Form 5 ☐ Form 6 lower ☐ Form 6 upper ☐ Transition
6	Educational level of Father	☐ No formal education ☐ Primary School ☐ Secondary School ☐ Higher institution
7	Educational level of Mother	☐ No formal education ☐ Primary School ☐ Secondary School ☐ Higher institution
8	Family history of hypertension	☐ No ☐ Yes

SECTION B:

Physical Activity Questionnaire

We are interested to find out about your level of physical activity from ***the last 7 days*** (in the last week). These includes sports or dance that make you sweat or make your legs feel tired, or games that make you breathe hard, like skipping, running, climbing, and others.

S/N	Question & Filters	Coding category				
1	Physical activity in your spare time: Have you done any of the following activities in the past 7 days? If yes, how many times? (For Q 1.1 to 1.13, Tick only one per question)	Number of times in the past 7 days				
		0	1-2	3-4	5-6	>7
1.1	Football	☐	☐	☐	☐	☐
1.2	Volleyball	☐	☐	☐	☐	☐
1.3	Badminton	☐	☐	☐	☐	☐
1.4	Jogging or running	☐	☐	☐	☐	☐
1.5	Hockey	☐	☐	☐	☐	☐
1.6	Basketball	☐	☐	☐	☐	☐
1.7	Tennis	☐	☐	☐	☐	☐
1.8	Rugby	☐	☐	☐	☐	☐
1.9	Swimming	☐	☐	☐	☐	☐
1.10	Bicycling	☐	☐	☐	☐	☐
1.11	Walking	☐	☐	☐	☐	☐
1.12	Dancing	☐	☐	☐	☐	☐
1.13	Others (specify).....	☐	☐	☐	☐	☐
2	In the last 7 days, during your physical education (PE) classes, how often were you very active (running, jumping, throwing)? (Tick one only.)	☐ I never do physical education ☐ Almost never ☐ Sometimes ☐ Quite often ☐ Always				
3	In the last 7 days, what did you do most of the time *at recess*? (Tick one only.)	☐ Sit down (talking, reading, doing schoolwork) ☐ walk around ☐ Run or play a little bit ☐ Run around and play quite a bit ☐ Run and play much most of the time				

4	In the last 7 days, what did you normally do *at lunch* (besides eating lunch)? (Tick one only.)	☐ Sit down (talking, reading, doing schoolwork) ☐ walk around ☐ Run or play a little bit ☐ Run around and play quite a bit ☐ Run and play much most of the time
5	In the last 7 days, on how many days *right after school*, did you do sport, dance, or play games in which you were very active? (Tick one only.)	☐ None ☐ 1 time last week ☐ 2 or 3 times last week ☐ 4 times last week ☐ 5 times last week
6	In the last 7 days, on how many *evenings* did you do sports, dance, or play games in which you were very active? (Tick one only.)	☐ None ☐ 1 time last week ☐ 2 or 3 times last week ☐ 4 or 5 times last week ☐ 6 or 7 times last week
7	*On the last weekend*, how many times did you do sports, dance, or play games in which you were very active? (Tick one only.)	☐ None ☐ 1 time ☐ 2- 3 times ☐ 4- 5 times ☐ 6 or more times
8	Which *one* of the following describes you best for the last 7 days? Read *all five* statements before deciding on the *one* answer that describes you.	☐ All or most of my free time was spent doing things that involve little physical effort ☐ I sometimes (1-2 times last week) did physical things in my free time (e.g. played sports, went running, swimming, bike riding) ☐ I often (3-4 times last week) did physical things in my free time ☐ I quite often (5-6 times last week) did physical things in my free time ☐ I very often (7 or more times last week) did physical things in my free time
9	Mark how often you did physical activity (like playing sports, games, doing dance, or any other physical activity) for each day last week.	
10	Were you sick last week, or did anything prevent you from doing your normal physical activities? (Tick one.)	☐ Yes ☐ No If yes what prevented you……

	None	Little	Medium	Often	Very often
Monday					
Tuesday					
Wednesday					
Thursday					
Friday					
Saturday					
Sunday					

SECTION C:

Smoking Questionnaire

S/N.	Questions & Filters	Question Response and code Skip to
1	Have you ever smoked?	☐ Yes ☐ No → Q9
2	How long have you been smoking?	Months ..... Years .....
3	How old were you when you first started smoking?	Age in years .....
4	Why did you start smoking?	☐ My friends asked me to try ☐ I think it is stylish to smoke ☐ To release tension ☐ My parents smoke, so I smoke too ☐ Try for fun ☐ Others, specify.....
5	Are you still (currently) smoking?	☐ Yes → Q9 ☐ No → Q9 ☐ Quitted → Q6
6	If you are ex-smoker at what age did you stop smoking?	Age in years .....
7	If you ex-smoker, why did you quit smoking?	☐ Friend' s advice ☐ Parents told me to stop ☐ My health life ☐ I am ill ☐ I feel nice for not smoking ☐ Others, specify.....
8	How long have you quit smoking?	Months ..... Years .....
9	In schools, do you think that smoking should be allowed in all areas, some areas or not allowed at all?	☐ All area ☐ Some area ☐ Not allowed

SECTION D:

Anthropometric Measurements

S/N	Parameter	1st reading	2nd reading	Average
1	Weight(kg)			
2	Height(cm)			
3	Systolic Blood Pressure (mmHg)			
4	Diastolic Blood Pressure (mmHg)			

Pengarah
Jabatan Pelajaran Negeri Perak

Ketua Jabatan
Faculty of Medicine And Health Sciences
Universiti Putra Malaysia

Appendix 5 : Approval Letter from Department of Education Perak

JABATAN PELAJARAN PERAK
JALAN TUN ABDUL RAZAK
30640 IPOH,
PERAK DARUL RIDZUAN.

Telefon : 05-501 5000 Faks : 05-527 7273 Portal : www.pelajaranperak.gov.my

"1MALAYSIA : RAKYAT DIDAHULUKAN PENCAPAIAN DIUTAMAKAN"

Ruj. Kami : J. Pel. Pk (AM)5114/4 Jld.11 (٤٤)
Tarikh : ٢٥ Februari 2013

PROF. DR. LEKHRAJ RAMPAL,
Faculty of Medicine And Health Sciences,
Universiti Putra Malaysia,
43400 UPM Serdang,
Selangor Darul Ehsan.

Tuan,

KEBENARAN UNTUK MENJALANKAN KAJIAN DI SEKOLAH DI BAWAH JABATAN PELAJARAN PERAK

Sukacitanya perkara di atas di rujuk dan surat tuan, Rujukan : UPM/FPSK/LR/01/2012, bertarikh 15 Februari 2013 dan surat dari Bahagian Perancangan Dan Penyelidikan Dasar Pendidikan, Kementerian PelajaranMalaysia, Rujukan : KP(BPPDP)603/5/Jld.05(3)), bertarikh 14 Mei 2012 adalah berkaitan.

2. Sehubungan dengan itu, dimaklumkan bahawa Jabatan Pelajaran Perak **tiada halangan untuk membenarkan pihak tuan meneruskan tempoh sehingga Oktober 2014 bagi menjalankan kajian "Effectiveness Of A Behavioural Modification School Based Intervention Program To Reduce NCD Risk Factors Among School Children – A Randomized Controlled Study"** seperti dinyatakan dalam surat tuan dengan syarat-syarat berikut :-

2.1 Pihak tuan perlu mendapatkan kebenaran terlebih dahulu daripada Pegawai Pelajaran Daerah dan Pengetua sekolah untuk menggunakan sampel kajian;

2.2 Kajian yang dijalankan hendaklah tidak mengganggu proses pengajaran dan pembelajaran sekolah;

2.3 Pihak tuan bertanggungjawab menjaga keselamatan dan kebajikan guru-guru yang terlibat dalam kajian ini;

Appendix 6 : Approval Letter from Batang Padang District Education Office

PEJABAT PELAJARAN DAERAH BATANG PADANG
JALAN PAHANG,
35000 TAPAH, PERAK DARUL RIDZUAN.
PORTAL :http://www.ppdbatangpadang.edu.my

No. Tel : 605-4011363
No. Fax : 605-4013527

"Perak Sentiasa Di Puncak Kecemerlangan"

Ruj. Tuan :
Ruj. Kami :PPD.BP. 01/16/018 (47)
Tarikh : **9 JULAI 2012**

PROF. DR. LEKHRAJ RAMPAL
Faculty of Medicine And Health Sciences,
Universiti Putra Malaysia
43400 UPM Serdang

Tuan/Puan,

KEBENARAN MENJALANKAN KAJIAN DI SEKOLAH

Dengan hormatnya saya merujuk perkara di atas. Surat surat JPN Perak J.Pel.PK.(AM)5114/4.Jld.9 (11) bertarikh 4 JUN 2012 adalah berkaitan.

2. Dengan ini dimaklumkan bahawa Pejabat Pelajaran Daerah Batang Padang **TIADA HALANGAN** membenarkan pihak tuan/puan menjalankan kajian bertajuk **"EFFECTIVENESS OF A BEHAVIOURAL MODIFICATION SCHOOL BASED INTERVENTION PROGRAM TO REDUCE NCD RISK FACTORS AMONG SCHOOL CHILDIEN-A RANDOMIZED CONTROLLED STUDY"** melibatkan guru sekolah sebagai sampel kajian dengan syarat-syarat berikut:

2.1 pihak tuan perlu mendapatkan kebenaran terlebih dahulu daripada Pengetua sekolah untuk menggunakan sampel kajian;

2.2 kajian yang dijalankan hendaklah tidak menganggu proses pengajaran dan pembelajaran di sekolah;

2.3 pihak tuan/puan bertanggungjawab menjaga keselamatan dan kebajikan guru-guru yang terlibat dalam kajian ini;

2.4 pihak tuan/puan hendaklah bertanggungjawab menanggung semua kos kajian termasuk pengangkutan, makan dan minum peserta yang terlibat;

2.5 guru-guru tidak boleh dipaksa terlibat dengan kajian ini;

Attachment 2

JKEUPM Ref No. : FPSK_Mei (13)37A

Members of the JKEUPM who reviewed the documents:

Prof. Dato' Dr. Lye Munn Sann

Date of approval: 31/5/2013

Endorsed at JKEUPM Meeting on **6/9/2013**, attended by:

NAME	DESIGNATION	GENDER	TICK IF PRESENT
Prof. Dr. Norlijah Othman	Paediatrics& Dean, Faculty of Medicine and Health Sciences	Female	√
Prof. Dr Zamberi Sekawi	Medical Microbiologist & Deputy Dean of Research and Internationalization, Faculty of Medicine and Health Sciences	Male	√
Prof. Dato' Dr. Lye Munn Sann	Medical Statistician, Dept of Community Health, Faculty of Medicine and Health Sciences	Male	
Prof. Dr. Tengku AizanAbd Hamid	Gerontologist & Director, Institute of Gerontology	Female	√
Prof. Dr. Lekhraj Rampal	Medical Statistician, Dept of Community Health, Faculty of Medicine and Health Sciences	Male	√
Prof. Dr. Elizabeth George	Pathologist, Dept of Pathology, Faculty of Medicine and Health Sciences	Female	√
Prof. Dr. Lim ThiamAun	Anesthesiologist, Dept of Surgery, Faculty of Medicine and Health Sciences	Male	√
Prof. Dr. Wan Omar Abdullah	Medical Parasitologist, Dept of Medical Microbiology and Parasitology, Faculty of Medicine and Health Sciences	Male	
Prof. Dr. Patimah Ismail	Professor of Biomedicine, Dept of Biomedical Sciences, Faculty of Medicine and Health Sciences	Female	√
Assoc. Prof. Dr. Johnson Stanslas	Pharmacologist, Dept of Medicine, Faculty of Medicine and Health Sciences	Male	√
Assoc. Prof Dr. Mansor Abu Talib	Assoc. Professor of Guidance and Counselling, Dept of Human Development and Family Studies, Faculty of Human Ecology	Male	
Assoc. Prof. Dr. Noritah Omar (Lay Person)	Assoc. Professor of English Language, Dept of English Language, Faculty of Communication and Modern Languages	Female	
Dr. RojanahKahar (Lay Person)	Lecturer of Dept of Human Development and Family Studies, Faculty of Human Ecology	Female	√
Tan Sri Dato' Napsiah Omar (Lay Person)	Chairman, National Population and Family Development Board	Female	