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The relationship between socioeconomic status and hypertension in South African adolescents

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UNIVERSITY OF THE WITWATERSRAND



FACULTY OF HEALTH SCIENCES

SCHOOL OF PUBLIC HEALTH

The Relationship between Socioeconomic Status and Hypertension in South African Adolescents.

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A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, in partial fulfilment of the requirements for the degree of Master of Science in Epidemiology in the field of Epidemiology and Biostatistics.

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Declaration

I **Zisiwe Mahlati** declare that this research report is my work. I submit this work for the degree of MSc Epidemiology (Epidemiology and Biostatistics) at the University of the Witwatersrand, Johannesburg. This research report has never been submitted before in any other university nor for any other examination.



19th May 2021

Dedication

I dedicate this work to the Lord Jesus Christ who made it possible for me to be part of this program at the prestigious University of the Witwatersrand. I also dedicate this work to my mother Thelma Nomgcobo Mahlati who has been with me through thick and thin.

Abstract

Background: Hypertension is a non-communicable condition marked by consistently elevated systolic and diastolic blood pressures. Hypertension is associated with adverse outcomes including various manifestations of morbidity e.g. cardiovascular disease, and disability, and mortality. Literature suggests that hypertension may be associated with socioeconomic status (SES), in addition to a range of other explanatory variables. Yet, pathways of this relationship in adolescence is not clear due to a paucity of data on hypertension and its correlates in this population, particularly in low- and middle-income countries (LMIC). Therefore, this study aimed to examine the relationship between SES and hypertension and identify the possible pathways between SES as exposure and the onset of hypertension among adolescents in South Africa.

Methods: The primary data accessed for this study were from the South African Demographic and Health Survey of 2016. A secondary analysis of the data entailed a crude estimation of the prevalence of hypertension and a survey-weighted investigation of correlates of hypertension among adolescents. For this secondary analysis, SES was categorized into five quintiles namely; poorest, poorer, middle, richer, and richest. Hypertension was defined as BP greater than the 95th percentile for sex, height and age in adolescents aged 17 years and younger. Among those aged 18 and 19 years, hypertension was defined as SBP above 140 mmHg and/or DBP above 90mmHg. Logistic regression models and generalized structural equation modelling (SEM) were then employed to examine the relationship between SES and hypertension in adolescence.

Results: The overall prevalence of hypertension in the study was 26% and was higher in males (28.3%) compared to females (24.6%). The relationship between SES and hypertension was not statistically significant. After adjusting for potential confounders age, sex, BMI, and level of education, the relationship between SES and hypertension remained one of no statistical significance. However, the results still suggested an increased risk of hypertension by 17% and 18% among the lowest two SES quintiles while the highest SES quintile suggested a 32% decreased risk of hypertension. From the GSEM results it was observed that age and BMI were the only factors that influenced hypertension with OR = 0.72 (95% CI: 0.63 - 0.82) and OR = 1.06 (95% CI:1.02 - 1.11) respectively.

Conclusion: This study reported a high prevalence of hypertension among adolescents in South Africa. Surprisingly, there was no association between SES and hypertension. This

study emphasizes a need for further social epidemiology research on hypertension in this age group, preferably with other proxies of socioeconomic status besides the wealth index. There is a need for such data to then influence health policies to address the high burden of hypertension in adolescents.

Keywords: Hypertension, socioeconomic status, adolescents, South Africa, Demographic and Health Surveys.

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List of abbreviations

BMI	Body mass index
BP	Blood pressure
CVD	Cardiovascular disease
DHS	Demographic and Health Survey
DPB	Diastolic blood pressure
GSEM	Generalized structural equation modelling
LICM	Low- and Middle-Income Countries
NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio
SBP	Systolic blood pressure
SA	South Africa
SEM	Structural equation modelling
SES	Socioeconomic status
SSA	Sub-Saharan Africa

1.0 Introduction

This chapter presents the outline and structure of the research report. The chapter provides detail on the background of the study, the literature review, the problem statement, justification for the study, the research question, the aim of the study, and the objectives of the study.

1.1 Background

Hypertension is a condition characterized by sustained high levels of blood pressure, that is, systolic blood pressure (SBP) equal or above 140 mmHg and or diastolic blood pressure (DBP) equal or above 90mmHg in adults (1). In children and adolescents, the criteria for hypertension differs slightly and is defined based on age, sex, and height-specific references. It is defined as the average SBP and/ or DBP blood pressure above or equal to the 95th percentile for age, sex, and height (2). Hypertension is a significant cause of premature mortality and disability globally and is a leading risk factor for cardiovascular disease (3).

In 2010, the global prevalence of hypertension was estimated to be 31.1% in adults aged 20 and above. When stratified by high and low to middle-income countries, the prevalence estimates were found to be higher in the low-and middle-income countries (LIMSs) with 31.5% and lower in the high-income countries with the prevalence of 28.5% (4). In 2013, the number of people living with hypertension globally was estimated to be 1.13 billion where two-thirds of those people are in low- and middle-income countries (LIMCs) (5). A 2013 World Health Organization (WHO) report suggested that globally that one billion adults had hypertension and that more than nine million deaths annually could be attributed to hypertension. With hypertension increasing in adults worldwide, there is also an observed parallel increase in hypertension in the young population of ages from 15 years to 30 (5).

In Africa, across the continent, the WHO estimated that the age-standardized percentage of people living with hypertension was 46% in the year 2013 (5). In South Africa (SA), the prevalence of hypertension among adults was found to be higher than 50% in the year 2017, it was estimated that 42-54% of South Africans are living with hypertension and that the number are expected to increase exponentially (6). The prevalence of hypertension in children and adolescents was thought to be approximately 10% in 2013 (7). Additionally, in a study that was conducted among Grade 12 learners in SA in 2018, in their final year of high school, there

was a high prevalence of pre-hypertension among the learners (29.7%) and the prevalence of hypertension was 13.7% (8).

A few studies have estimated the prevalence of hypertension in both high and low to middle-income settings. Further, these studies have attempted to examine the correlates of hypertension prevalence. For example, a systematic review and meta-analysis of the prevalence of hypertension in urban and rural regions of India led by Anchala and colleagues suggested that the prevalence of hypertension among adults in India in urban areas was significantly higher than in rural parts. Although the authors suggest that they only managed to access a few studies conducted in rural parts of India of which most were cross sectional design, this is an interesting finding. (9). Manus and colleagues who conducted a cross sectional study to estimate the prevalence of hypertension in Madagascar found a significant relationship between age, BMI, and hypertension (10). Shen and colleagues in a Chinese study on prevalence and risk factors of hypertension and pre-hypertension showed that sex, age, education, BMI, job position, ethnicity, hyperuricemia, diabetes, family history of hypertension, frequent drinking, and obesity or overweight were correlates of hypertension (11).

There are several risk factors for hypertension and these can be either modifiable or non-modifiable. Modifiable risk factors are those that can be reduced or managed with changed behaviour and include excessive salt intake, sedentary lifestyle, or lack of exercise, high fat diet, harmful use of alcohol, use of tobacco, socioeconomic status, and stress. Non-modifiable risk factors are those that an individual is unable to change. These include sex, race, age, family history of hypertension, growth-rate, metabolic factors, and genetic composition (12). In a meta-analysis conducted by Leng and colleagues, low socioeconomic status, potentially a modifiable risk factor, was found to be related to hypertension, especially among those with low levels of education. This was particularly true for high-income country settings with a stronger relationship between socioeconomic status (measured by income, education, and occupation) and hypertension seen among women (13).

However, in another high-income setting, in Japan, Satoh and colleagues conducted a nationally representative cross-sectional study of over 2000 adult participants. They suggested a prevalence estimate of 48.9 % of hypertension. They found that SES was not significantly associated with poor awareness of one's hypertension nor treatment and control of the condition after adjusting for a range of confounding variables (age, sex, BMI, smoking, alcohol consumption, regular physical exercise, and a range of comorbid conditions). SES was not

clearly associated with unawareness, nor treatment, nor poor control of hypertension (14). Another study by Mathews and colleagues suggested that hypertension was associated with psychological stress in young adults (15) and the Satoh study suggested that being unmarried or living alone (possible proxies for stress) was associated with a higher risk of hypertension.

Nutrition and diet are known to be crucial contributors. However, other factors also contribute such as socioeconomic status (SES), growth rate, cultural influences, metabolic and genetic factors, pubertal stage, racial and ethnic predispositions, and overweight and obesity (16). Some of these risk factors are acquired by behaviour and are caused by lifestyle changes during adolescence. Adolescence is a developmental stage from 10 to 19 years, where life is presented with rapid growth and development of body, mind, and social relationship with behaviour changes such as sexual maturity and independence. This stage comes with more exposure to risky behaviours such as unsafe sex, and substance abuse. At this age, these risk factors are well tolerated and are barely perceived by the adolescents as harmful due to their premature state of neurological development (17,18). A systematic review and meta-analysis in 25 African studies revealed an 8.1 % prevalence of hypertension in South African children and adolescents (19).

1.2 Literature review

A literature review was undertaken to examine epidemiological studies of hypertension in adults and adolescents with a view to understanding gaps in the field. Several themes emerged from my searches and are captured below. These include: 1.2.1. Prevalence and correlates of Hypertension in adults and children. 1.2.2 Social correlates of Hypertension in adults and children. 1.2.3 Biological correlates of Hypertension in adults and children, and 1.2.4 Behavioural correlates of Hypertension.

1.2.1. Prevalence and correlates of Hypertension in adults and children

In addition to studies estimating the prevalence of hypertension among adults in high and low to middle-income countries described above, some epidemiological studies have been undertaken to examine the prevalence of hypertension in children and adolescents (19–21). These have been conducted in high and low to middle-income countries contexts and they include a spectrum of meta-analyses and/or systematic reviews, and cross-sectional studies.

Many of these also include attempts to estimate correlates of hypertension such as gender , age and socioeconomic status (22).

Meta-analyses have aimed to estimate a pooled prevalence of hypertension in children and adolescents. A meta-analysis conducted by Akbari and colleagues who work in Iran, a low to middle-income country context (published in 2017) suggests that the pooled prevalence of hypertension in Iranian children was 8.9% and that hypertension was higher in male children, a result that was statistically significant (23). Another recent systematic review and meta-analysis suggested that the global prevalence of childhood hypertension has increased from 1994 to 2018. This study showed that in 2015, hypertension prevalence was 4.32% in children aged 6 years, 3.25% among those who are 19 years while this peaked among those who are 14 years with a prevalence of 7.89% (24). The prevalence of childhood hypertension in SA had increased substantially in a short period of about a decade. A systematic review of childhood hypertension in SA suggested that the prevalence of hypertension ranges from 7.5 to 22.3% in 2008 (25).

A study conducted in Uganda to assess prevalence, awareness, and control of hypertension revealed a linear relationship between age and hypertension. In the age range of 15-24 years, women had a lower prevalence of hypertension with men far more likely to have hypertension (26). When they examined the difference between rural and urban settings, men in urban settings had a higher prevalence of hypertension compared to men in rural settings, as in the study in India cited earlier (9). However, there was no difference observed between women in rural and urban settings (26).

Similar results were observed in Ouagadougou, a city in Burkina Faso. The prevalence of hypertension was significantly higher in those who lived in formal settings compared with those in informal settings. However, this difference was no longer evident after adjusting for age. Other independent risk factors observed were; being an unmarried woman, recent migration from rural to urban, obesity, and physical inactivity (27).

1.2.2. Social correlates of Hypertension in adults and children

There has been an observed difference in the prevalence of hypertension between males and females. Literature has shown that men tend to have higher blood pressure and more hypertensive than women (28). In a study that explored sex differences in blood pressure during adolescence, girls had lower SBP and DBP than boys. This difference was seen when they were

both at rest and during challenges. The composition of the body and insulin resistance were found to play a part in these differences (29). Similar findings were obtained in a pilot study of African American adolescent males and females where the mean SBP for males was higher than that of females (30).

A review of the impact of SES on hypertension suggested that low SES was associated with elevated levels of blood pressure in adults. This association was reported to be weak in this study (31). A study that analysed socioeconomic determinants of hypertension in South Africa showed that it is a complex issue in the country. SES was defined by two variables education and income, which gave contrasting results between gender. Higher SES in women was associated with lower BP while in men it resulted in higher BP (32).

Conflicting evidence on the association between SES and hypertension has been observed in literature with some studies finding no association. For example, a study conducted in Europe suggested that higher SES was found to have a protective effect on hypertension, but only in univariate analysis. However, using education as a proxy to measure SES, after adjusting for age and sex, education had no association with hypertension (33). In addition to this, studies investigated the effect of SES change from low to high between childhood and adolescence to observe whether there is an association between SES mobility, blood pressure, and hypertension. Kagura and colleagues explored this relationship and found no association between SES change and hypertension (34). The same results were found in a similar study by Hallal and colleagues where there was no relationship between blood pressure and socioeconomic trajectories from birth to 11 years of age (35).

It is important to note that SES can be measured by a number of proxies including financial, occupational or educational variables (36,37). The education level of parents may be correlated with their potential income earnings, access to resources, and social standing. Furthermore, it is a reflection of one's level of understanding of health and essential health practices that will influence their child's health status (38,39). One study conducted to explore the effect of parental education attainment on adolescent blood pressure revealed that low parental education is associated with elevated SBP and DBP. However, this association was mediated by the mother's body weight during pregnancy and the adolescent's body weight (40).

BP levels in adolescence may be influenced by their parent's SES in different pathways. These pathways include maternal education level, paternal occupation status inter-linked to the level of urbanization of the place of residence taking into consideration age, sex, and weight status

(41). Data from a cohort study of low-income population adolescents revealed that most adolescents with normal weight have higher levels of BP. This cohort study also suggested that a large number of adolescents in low-income had a high prevalence of hypertension and many were undiagnosed. Dietary habits of adolescents in this low-income population such as rare consumption of fast foods and engaging in regular exercise did not yield the expected low BP but instead, the opposite was reported (42).

Contrasting results were obtained in another study which showed no significant disparities in the prevalence of hypertension among adolescents from low to middle, and high-income settings (43). Another set of results was obtained in a smaller sample size study of black adolescents' SES and BP reactivity at Virginia Commonwealth University, in the United States of America. It was reported that those who lived in poorer neighbourhoods had lower BP only when their parents' level of education was higher. Furthermore, those who lived in poorer neighbourhoods but whose parents' income was higher had lower BP (44). The different findings in these studies reveal that BP complexities and income disparities need to be explored further for future use in preventive studies especially in the adolescence stage of life.

1.2.3. Biological correlates of Hypertension in adults and children

Body mass index (BMI) is an important risk factor for hypertension in adolescents. In a very large cohort of 714, 922 healthy teenagers, BMI was found to have a positive significant association with both SBP and DBP. This association was found in both female and male participants in the study and the results from a dose-response curve also showed that BMI was independently associated with an additional risk of higher blood pressure levels (45). Similar results were obtained in another adolescent's studies where with every increase in BMI percentiles, there was a moderate increase in both SBP and DBP (46). Elevated BMI had an association with an elevated risk of hypertension even when adjusting for various factors that could be potential confounders (47).

The increasing risk of hypertension with increasing BMI may sometimes present differences when it comes to sex. In one study within the normal range, BMI increase in males is associated with hypertension while this may not be found in females (48). Another study yielded opposite results from the previous study. Changes in BMI, particularly increase, seem to be related to an increase in the risk of hypertension. The increased risk of high blood pressure was seen to be four times higher in women who were obese and those who were overweight. However, in

males, the increased risk of high blood pressure was two times higher in only those who were overweight (49).

1.2.4. Behavioural correlates of hypertension

Physical activity has been found to be associated with a decrease in BP. Increased levels of physical activity have been associated with decreased blood pressure and this sometimes is not dependent on the intensity of the activity but rather the volume of the activity (50). A cohort study that was conducted to explore the cross sectional and longitudinal association between BP and physical activity (objectively measured) also showed a similar trend that increased physical activity is associated with lower BP. It showed that additional and more intensive physical per day and is associated with decreased BP for both SBP and DBP. However, there was an association between physical activity and SBP (51).

Conflicting results were found in another study that examined the association between physical activity, fitness, and features of the metabolic syndrome, there was no association between physical activity and BP (52).

Literature seems to give conflicting evidence of the relationship between smoking and hypertension. One study in Europe observed that among women, smoking lowered SBP on current smokers than those who never smoked and those stopped smoking, the accuracy on the measurements on smoking as a habit, and other risk factors may have biased the results (53). However, there has been evidence that shows a dose-response relationship between cigarette smoking and hypertension although the relationship was not significant in men (54). Because of this and similar findings, evidence supports that to have better BP, cigarette smoking should be forbidden rather than continued (55).

In sub-Saharan Africa, a four-country cross-sectional study that investigated the burden of hypertension revealed that smoking was among the risk factors associated with hypertension. Results from that study showed a crude OR of the use of filtered tobacco which was significantly higher than those who had stopped smoking, and those who used unfiltered tobacco (56). Literature is scarce on the effects of smoking and hypertension among adolescents; hence it gives enough reason for further studies to investigate the relationship.

An association between the level of education attained and hypertension in some studies has been observed. After adjusting for other factors, a higher level of education attained was associated with lower BP levels, however, this was a study that sought to explore the

relationship in the older community-living adults, not adolescents (57). Another inverse association of higher educational level and hypertension was observed only among women in an Austrian population which showed a decrease in the risk of hypertension and diabetes mellitus with a higher educational level after adjusting for other factors (58). Furthermore, education was found to be one of the factors that were associated with hypertension in the study of the burden of hypertension in sub-Saharan Africa (56).

Literature has revealed a coexistence between hypertension and types 2 diabetes in the same individual. The reason for this is the aspects of pathophysiology which are used by both hypertension and type 2 diabetes. This coexistence was observed in a San Antonio Heart Study where 85% of participants with type 2 diabetes had hypertension by the time they reach 50 years of age, while 50% of participants with hypertension had type 2 diabetes (59). Also, Hendricks and colleagues in a study conducted in four countries in sub-Saharan Africa found that there is an association between obesity, diabetes, and hypertension (60).

1.3 Problem statement

Literature accessed for this report suggests that the prevalence estimates of hypertension in children and adolescents vary but can be as high as 22 % (25). Correlates of the prevalence estimates include nutrition and diet as important contributors along with other factors that may have greater contributions such as socioeconomic status, growth rate, pubertal stage, racial and ethnic predispositions, cultural influences, metabolic and genetic factors, overweight and obesity (16). Moreover, early life hypertension and undiagnosed childhood hypertension may lead to adult hypertension and organ damage, so an understanding of correlates of adolescent hypertension might help understand the trajectory of the condition (8,61). Many of the studies in literature described the relationship of hypertension and correlates using simple regression models, thus, a complete image of pathways (direct and indirect) through which SES leads to hypertension needs to be established.

The DHS 2016 is a nationally representative survey of SA and data from there presents an opportunity to estimate the prevalence and correlates of hypertension in adolescents in a nationally representative sample. Data on the pattern of association between SES and hypertension is scarce and the influence of SES on hypertension is not clear especially in South Africa (32). To our knowledge, there is limited data on hypertension and limited studies to explore the relationship between hypertension and SES in adolescents.

1.4 Justification

Several factors contribute to the onset of hypertension including obesity, overweight stress, and SES (12). To our knowledge, no study in SA has examined the indirect pathways in which socioeconomic status follows leading to hypertension in adolescents in a nationwide sample. Examining these pathways may provide more knowledge on adolescent hypertension and inform on ways that may be helpful to the goal of reducing hypertension and essentially cardiovascular disease (CVD) risk in later life. Given the prevalence of hypertension in South Africa, it is a necessity to conduct this study to understand the prevalence and correlates of adolescent hypertension and the trajectory of hypertension from pathways between adolescence, adulthood, and possibly future generations. This will assist the National Department of Health (NDoH) in creating health policies, and devising strategies that are necessary for reducing the prevalence of childhood and adolescent hypertension.

Also, adolescence is a critical time in human development where children are at high risk of adverse behavioural patterns and psychosocial stressors, which could lead to the onset of non-communicable diseases (62).

1.5 Research Question

What is the relationship between socioeconomic status and hypertension in South African adolescents?

1.6 Aim

To investigate direct and indirect pathways through which socioeconomic status leads to hypertension in the South African adolescent population.

1.7 Specific objectives

1. To describe sex-specific prevalence of hypertension in South African adolescents from 15 to 19 years.
2. To describe the relationship between socioeconomic status and hypertension in adolescents from 15 to 19 years.
3. To describe the pathways showing the relationship between socioeconomic status and hypertension adolescents from 15 to 19 years using Generalised Structural equation modelling approach.

2.0 Methodology

This chapter provides information on the methodologies that were employed in this study. The chapter presents details of the study design followed, study setting, and study population. It also provides detail on the measured outcome variable, exposure variables, and the data analysis techniques employed in this study.

2.1 Primary Study

The primary data accessed for this thesis is the South African Demographic and Health Survey of 2016 (SADHS 2016), which is a national survey carried out under the auspices of the National Department of Health. The sample of the survey was patterned to provide updated estimates of demographic and health indicators for the whole country with nine provinces with urban and non-urban areas (63).

2.2 Study design

This study undertakes a secondary analysis of cross-sectional data from the SADHS 2016 to estimate the prevalence of hypertension and investigate the association between SES hypertension in adolescence.

2.3 Study setting

SADHS 2016 had a nationally representative sample of 8514 women aged 15- 49, and 3618 men aged 15-59. The interviews were conducted with women and men from all the selected households. (63).

2.4 Study population and sampling

The study consisted of 2227 South African adolescents aged 15-19yrs. The respondents were from all of the nine provinces of SA (Eastern Cape, Western Cape, Northern Cape, Free State, KwaZulu-Natal, North West, Gauteng, Mpumalanga, and Limpopo). 1090 of those adolescents did not have results on blood pressure, thus we were left with 1137 participants. From the 1137 participants, those who had a history of hypertension (n = 59), and women who were pregnant (n = 16) at the time of the survey were excluded. Therefore, the total number of participants used in this study 1062. Figure 1 presents the flow chart which shows how the final sample was obtained.

Inclusion criteria

Males and females aged between 15 and 19 years who participated in the survey in the year 2016. The status of hypertension among the participants must be available.

Exclusion criteria

Participants who had missing data on SES and blood pressure were excluded. Participants who had a history of hypertension (already told by the doctor that they have hypertension) and /or were pregnant participants during the study period were also excluded.

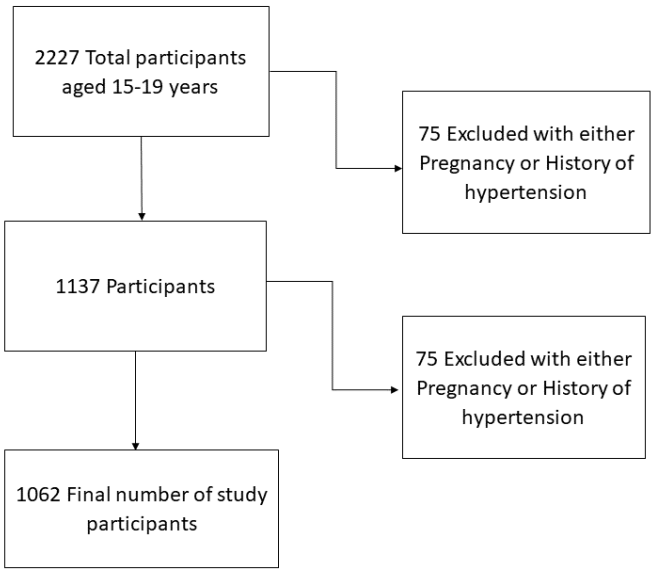


Figure 1: Flow chart presenting the sampling strategy to obtain the final study sample

2.5 Measures

Outcome/dependent variable

The **outcome** variable in this study was hypertension. BP was classified as normal if BP was less than the 90th percentile for age, sex, and height for adolescents less than 18 years. BP was classified as hypertensive if BP was greater than the 95th percentile for age. On those who are 18 and 19 years old, the normotensive status was less than 120/80mmHg for systolic blood pressure (SBP) and diastolic blood pressure (DBP) above 90mmHg, accordingly. Hypertension was classified as SBP above 140 mmHg and/or DBP above 90mmHg (2).

Exposure/predictor variables

Socioeconomic status was the main exposure variable in this study.

Socio-demographic variables: Type of residence, marital status, level of education, cigarette use, household density, age, sex, BMI, and race/ethnicity.

Comorbid condition: Diabetes

2.5.1 Description of variables in this study

Age

Age was a continuous variable ranging from 15 years to 19 years.

Sex

This was a categorical variable with two categories for male and female

SES

SES was kept as it was in the primary study (SADHS 2016), a categorical variable named "wealth index" consisting of five quintiles. These quintiles are derived from a score given to each household based on their household possessions. These ranged from television, car, bicycle, and others are housing characteristics such as toilet facility used, source of drinking water, and flooring material. The five quintiles were; poorest, poorer, middle, richer, and richest.

Race

There were four categories for this variable in the primary study for racial classification, typical of the race categories used in South Africa and were; Black/African, White, Coloured, Asian/Indian.

Type of residence

Participants in the primary study were grouped into two types of residence according to the classification of the enumeration area of each participant and were; Rural and Urban

Marital status

This variable had three categories of marital status where participants reported their current marital status. The categories were; Never married, Married, Divorced/Separated.

Education

This variable consisted of the level of education each participant attained and were grouped into five categories. However, during analysis (logistic regression) the categories with too few numbers of participants ("no education, pre-school" "don't know" and "higher") presented as outliers, and estimates could not be obtained. Thus, the variable was collapsed into two categories namely; "Primary" and "Secondary" to obtain estimates. The five categories of the primary study were; No education, pre-school, Primary, Secondary, Higher, Don't know.

BMI

The variable was initially a continuous variable with the record of each participant's BMI. The following four categories were generated using the WHO classification of weight status and were used in descriptive statistics only (64). In logistic regression and GSEM, BMI was used as a continuous variable. The categories for classification were; <18.5 (kg/m^2) - Underweight, 18.5 - 24.9 (kg/m^2) - Normal weight, 25.0 - 29.9 (kg/m^2) – Overweight, >29.9 (kg/m^2) – Obese.

Household density

This was a continuous variable with the record of household members in each household and was ranging from 1 to 24 members in a household.

Use of cigarettes

This variable was initially a continuous variable consisting of the number of cigarettes each participant used within the last 24 hours before the survey and was used as such in the logistic regression. Those with the record of 0 (zero) and "doesn't smoke" were then categorized as "No" meaning they are not smokers while those with the record of 1 and above were categorized as "Yes" meaning that they do smoke. The variable was categorized as follows for analysis were; Yes (smoker), No (non-smoker).

Diabetes status

The variable was a continuous variable which was a measure of glucose in the blood (HbA1c) in each participant. The following categories were generated according to the American Diabetes Association (ADA 2010), and were; Values from 4.8% to 5.6% - Normal, Values between 5.7% to 6.4% - Pre-diabetic, Values from 6.5% and above – Diabetic.

Hypertension status

The primary study had three records for each of the blood pressures (SBP and DBP). Average blood pressure was obtained using a mean of the three measures. Subsequently, the two variables (average SBP and average DBP) were used to generate the outcome variable "hypertension status" according to the definition of hypertension as described in the previous chapters above. The variable was categorized as; Yes (Hypertensive), No (Non-hypertensive).

The list of variables and their codes are presented in appendix 6 .

2.6 Statistical Analysis

2.6.1 Data management

Data were obtained from the Health and Demographic Survey site in Stata format. Data cleaning involved extracting the study age-group (15 -19 years) from the dataset and dropping all variables not needed in this study. Some of the variables had to be generated and others were re-coded for analysis.

2.6.2 Descriptive statistics

Data cleaning and exploratory analyses were conducted including testing for underlying assumptions of the tests used. Continuous variables were tested for normality using the histogram test and when data did not follow a normal distribution, they were presented as median and interquartile range. To investigate the differences between the excluded and the included sample Pearson Chi-squared test was performed. This was done in order to ensure external validity and to control for bias. The difference between the two samples was addressed and reported in a table in appendix 5.

Sex-specific prevalence of hypertension.

Characteristics for continuous variables were reported as a median and interquartile range while for categorical variables they were reported as frequencies. Pearson Chi-squared test was performed to test for association between different categorical variables and the outcome variable. Tables and graphs were used to present the results.

2.6.3 Inferential statistics

To account for the study design of the DHS 2016 survey, survey-weighted data analysis was used in all models discussed below

Relationship between SES and hypertension

The relationship between SES and hypertension was explored using logistic regression. Univariable logistic regression was performed on all explanatory variables listed in section 2.5 against the outcome variable of hypertension. Following the univariable analysis, stepwise regression was used for the selection of variables using forward selection with a probability of entry of p-value = 0.2. Age, sex, and SES were locked in the selection step along with the level of education which was marginally significant in the univariable model. A multivariable logistic regression model was fitted as the final model using the selected variables and the results are presented and subsequently discussed.

Pathways between SES and hypertension

The pathways linking SES and hypertension were analysed using GSEM. The proposed conceptual framework that guided the GSEM diagram is presented in figure 2. The direct and indirect effects of SES on hypertension were explored using both the continuous and categorical variables that were used on the final multivariable logistic regression. As the outcome variable is binary, it was specified as binomial logit and the explanatory variables were treated as normal observed variables. Ordinal logistic regression was conducted to assist in generating variables for the GSEM because the outcome variable is categorical and some of the independent variables were also categorical. The first model fitted was the model with only direct pathways of all variables on the outcome variable hypertension.

The direct pathways between the outcome variable and explanatory variables were drawn in the GSEM builder interface from each variable to the outcome. The exponentiated OR's for the direct effects were obtained from the coefficients produced. The second model fitted was the model with both direct and indirect pathways to the outcome. Taken from the proposed conceptual framework, the direct pathways between the outcome variable and explanatory variables were drawn in the GSEM interface from each variable to the outcome and from one variable to the other variable(s) where possible. The total effects for each variable were generated by adding the direct effects and the indirect effects.

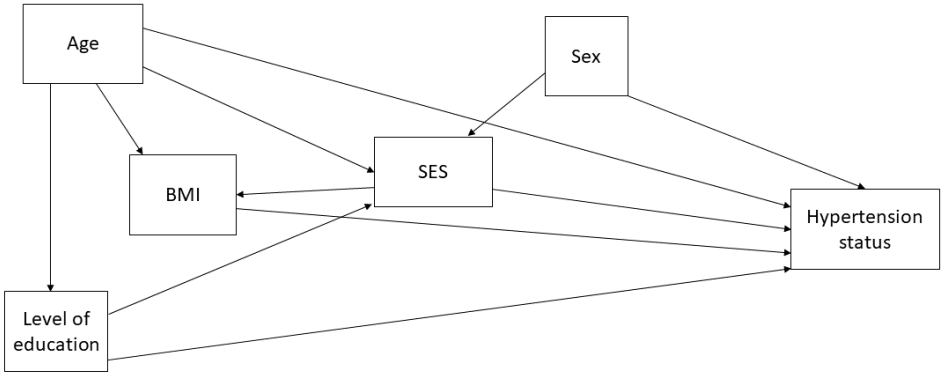


Figure 2: A proposed analytical framework for the GSEM

All analyses were carried out using Stata version 16.0 at alpha 0.05 significance level.

2.7 Ethical considerations

Ethical approval for the study was obtained from the Human Research Ethics Committee (HREC) of the University of the Witwatersrand and the ethics waiver certificate is presented in appendices as appendix 3 with protocol number W-NN-200106-01.

3.0 Results

This chapter presents the results of the study. It contains tables, graphs, and GSEM diagrams, all of which present study participants' characteristics and the prevalence of hypertension along with the association that exists between the explanatory variables and the outcome variable. The results in this chapter are presented in the order that follows the study objectives.

3.1 Sex-specific prevalence of hypertension.

Table 1 presents the socio-demographic and anthropometric measures of the study participants. 61.8% of the participants were aged below 18 years and 50.7% were males. The majority (91.5%) of the study participants were Black/African while the minority (0.6%) were of Asian/Indian descent. The majority of the study participants had normal range BMI (59.0%), while 11.1% and 5.7% were pre-obese and obese respectively. At least 80% of the study participants had a secondary level of education and 47.2% were in the lowest two SES quintiles.

The overall prevalence of hypertension in the study was 26% and was higher in males (28.3%) compared to females (24.6%). SBP and DBP medians were higher among the hypertensives at 132 mmHg and 85 mmHg respectively compared to corresponding measures among the non-hypertensives at 116 mmHg and 73 mmHg respectively. A total of 69 (6.5%) study participants were cigarette smokers of which 14 (29.3%) were hypertensive. In addition, 85 (8.0%) study participants were diabetic of which 27.1% were hypertensive. Statistical significance was observed on the effect of age (p-value <0.001), level of education (p-value = 0.01), BMI (p-value = 0.05), and glucose homeostasis (p-value=0.012) on the hypertensive status of the study participants.

Table 1: Socio-demographic and anthropometric measures of the study participants

Variable	Hypertensive	Non-Hypertensive	Total	P-value
	n = 281	**n = 781**	**n = 1062**	
Age in years				$\chi^2 = 39.9$
15 n (%)	141 (66.20)	72 (33.80)	213 (20.06)	<0.001
16 n (%)	157 (65.42)	83 (34.58)	240 (22.60)	
17 n (%)	142 (69.61)	62 (30.39)	204 (19.21)	
18 n (%)	181 (85.78)	30 (14.22)	211 (19.87)	
19 n (%)	160 (82.47)	34 (17.53)	194 (18.27)	
Sex/Gender				$\chi^2 = 1.8$
Male n (%)	152 (28.25)	386 (71.75)	538 (50.66)	0.179
Female n (%)	129 (24.62)	395 (75.38)	524 (49.34)	
Race				
Black/African n (%)	253 (26.03)	719 (73.97)	972 (91.53)	0.425*
White n (%)	3 (18.75)	13 (81.25)	16 (1.51)	
Coloured n (%)	23 (33.82)	45 (66.18)	68 (6.40)	
Indian/Asian n (%)	2 (33.33)	4 (66.67)	6 (0.56)	
SES				$\chi^2 = 4.4$
Poorest n (%)	79 (28.83)	195 (71.17)	274 (25.8)	0.350
Poorer n (%)	63 (27.75)	164 (72.25)	227 (21.37)	
Middle n (%)	58 (22.48)	200 (77.52)	258 (24.29)	
Richer n (%)	61 (28.64)	152 (71.36)	213 (20.06)	
Richest n (%)	20 (22.22)	70 (77.78)	90 (8.47)	
Type of residence				$\chi^2 = 0.8$
Urban n (%)	132 (27.85)	342 (72.15)	474 (44. 63)	0.357
Rural n (%)	149 (25.34)	439 (74.66)	588 (55.37)	
Marital status				
Never married n (%)	279 (26.57)	771 (73.43)	1050 (98.87)	0.807*
Married n (%)	2 (18.18)	9 (81.82)	11 (1.04)	
Divorced/Separated n (%)	0 (0)	1 (100.0)	1 (0.09)	
Education				$\chi^2 = 5.5$
Primary n (%)	69 (32.86)	141 (67.14)	210 (19.77)	0.019
Secondary n (%)	212 (24.88)	640 (75.12)	852 (80.23)	
BMI (kg/m^2)				
<18.5 n (%)	57 (27.98)	191 (77.02)	248 (23.35)	0.053*
18.5-24.9 n (%)	166 (26.48)	461 (73.52)	627 (59.04)	
25.0 - 29.9 n (%)	33 (27.97)	85 (72.03)	118 (11.11)	
> 29.9 n (%)	19 (31.67)	42 (68.33)	60 (5.65)	
Missing n (%)	6 (66.67)	3 (33.33)	9 (0.85)	
Average Blood Pressure				
SBP median (IQR)	132 (17)	116 (14)	119 (17)	
DBP median IQR)	85 (10)	73 (10)	76 (12)	
Household density median (IQR)	5 (3)	5 (3)	5 (3)	

Smoke cigarette				$\chi^2 = 1.4$
Yes n (%)	14 (20.29)	55 (79.71)	69 (6.50)	0.230
No n (%)	267 (26.89)	726 (73.11)	993 (93.50)	
Diabetes status				$\chi^2 = 11.0$
Normal n (%)	27 (20.15)	107 (79.85)	134 (12.62)	0.012
Prediabetic n (%)	174 (25.36)	512 (74.64)	686 (64.6)	
Diabetic n (%)	23 (27.06)	62 (72.94)	85 (8.0)	
Missing n (%)	57 (36.31)	100 (63.69)	157 (14.78)	

Note: (The symbol $\chi 2$ denotes Chi-squared). * means that Fisher's exact was used in that category.

Figure 3 presents a sex-specific crude prevalence of hypertension across the five different age groups. In males, hypertension prevalence was highest at age 16 (40.0%) and was lowest at age 18 (18.8%). In females, the prevalence of hypertension was highest at age 17 (35.2%) and as it was with males, was lowest at age 18 (9.90%).

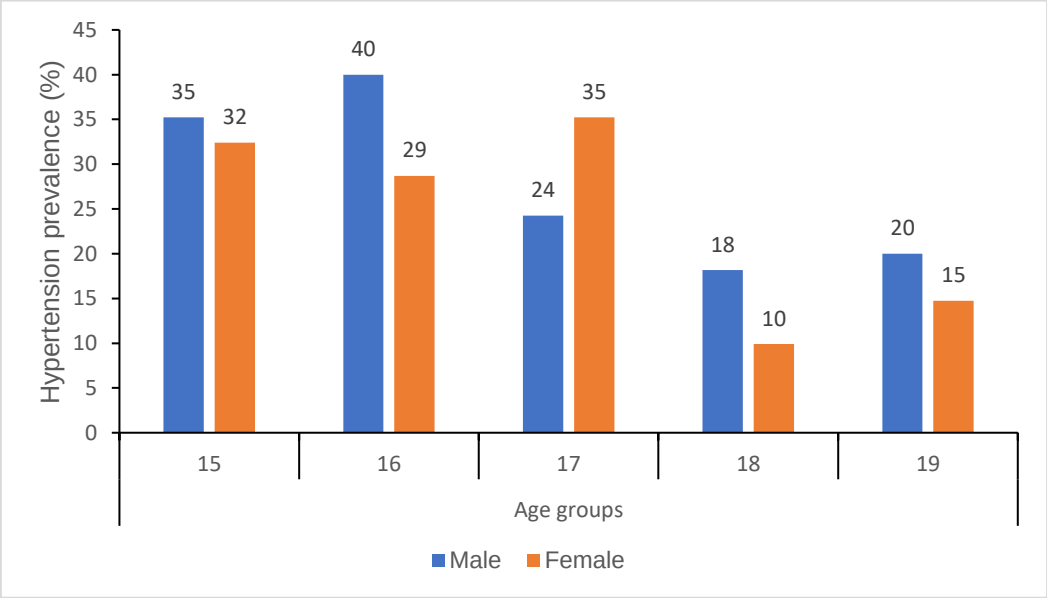


Figure 3: Sex-specific crude prevalence of hypertension in South African adolescents from 15 to 19 years 2016

Figure 4 presents the sex-specific prevalence of hypertension across the five different quintiles of SES. The highest prevalence of hypertension among males was found in the poorer quintile (35.5%) while for females the highest prevalence of hypertension (32.1%) was found in the poorest quintile. Prevalence fluctuated across gender and SES, neither the upper two nor the lowest two quintiles bore the highest burden.

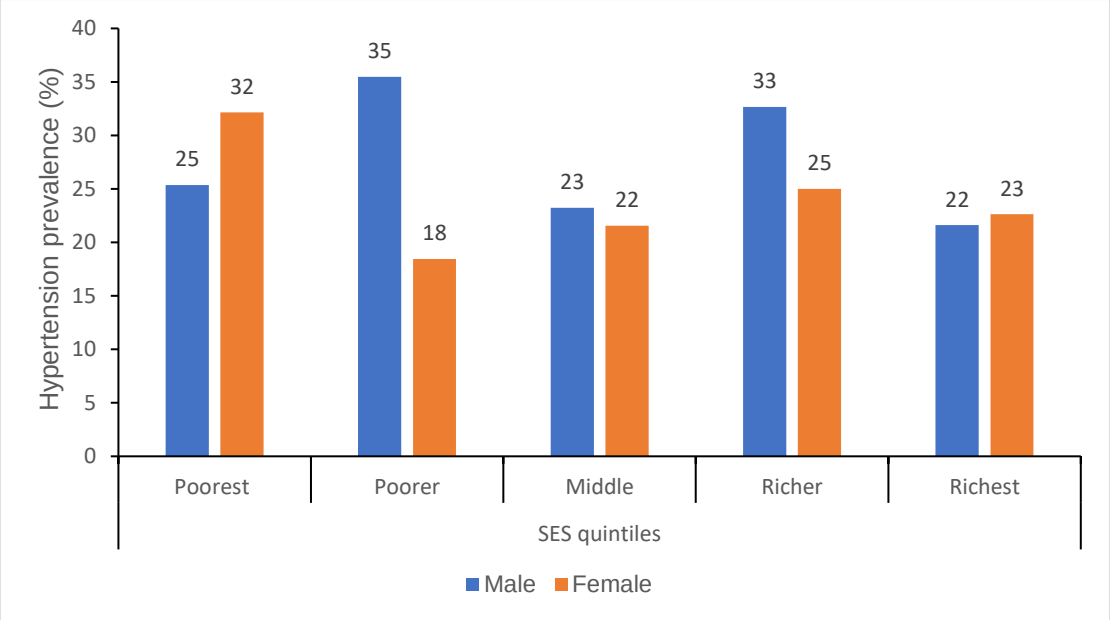


Figure 4: Sex-specific prevalence of hypertension in South African adolescents across SES quintiles 2016

3.2 The relationship between SES and hypertension.

Table 2 presents the unadjusted (univariable) and adjusted (multivariable) ORs and 95% confidence intervals (CIs) obtained from logistic regression. Results from univariable models suggest that age (OR = 0.72, 95% CI: 0.64 - 0.81, p-value <0.001) and BMI (OR = 1.05, 95% CI: 1.01 - 1.08, p-value = 0.013) were the only two explanatory variable that were independently associated with hypertension status of the adolescents. An increase in age by years decreased the likelihood of developing hypertension by 25%, while an increase in BMI by a kilogram per meter squared increased the likelihood of developing hypertension by 5%. The level of education had a marginally significant effect on hypertension status with OR = 0.70, p-value = 0.067. All five SES quintiles did not present statistical significance in their effect on hypertension status.

After adjusting for age, sex, BMI, and level of education, SES remained with no insignificant effect on hypertension status. The marginal significance of the level of education on hypertension status was lost in the multivariate analysis. Age and BMI remained the only two factors that were associated with hypertension status with OR = 0.71, p-value <0.001, and OR = 1.06, p-value = 0.007 respectively.

Table 2: Results from the univariable and multivariable logistic regression of hypertension and factors examined

Variable	Univariable		Multivariable	
	OR (95 % CI)	P-value	OR (95 % CI)	P-value
Age	0.72 (0.64 - 0.81)	<0.001	0.71 (0.63 - 0.81)	<0.001
Sex				
Female	0.96 (0.67 - 1.37)	0.818	0.82 (0.54 - 1.23)	0.328
Male	Ref			
Race				
Black/African	Ref			
White	0.50 (0.08 - 3.24)	0.465		
Coloured	1.40 (0.67 - 2.94)	0.375		
Indian/Asian	1.33 (0.15 - 12.11	0.801		
SES				
Poorest	1.20 (0.74 - 1.95)	0.453	1.20 (0.72 - 1.98)	0.486
Poorer	1.25 (0.77 - 2.03)	0.364	1.18 (0.71 - 1.96)	0.518
Middle	Ref		Ref	
Richer	1.02 (0.61 - 1.72)	0.930	1.00 (0.58 - 1.73)	0.999
Richest	0.72 (0.32 - 1.64)	0.439	0.68 (0.30 - 1.55)	0.358
Type of residence				
Urban	Ref			
Rural	1.13 (0.79 - 1.60)	0.508		
Marital status				
Never married	Ref			
Married	0.64 (0.12 - 3.44)	0.601		
Divorced/Separated	Empty*			
Education				
Primary	Ref		Ref	
Secondary	0.70 (0.47 - 1.03)	0.067	0.98 (0.65 - 1.47)	0.907
BMI (kg/m^2)	1.05 (1.01 - 1.08)	0.013	1.06 (1.02 - 1.11)	0.007
Use of tobacco	1.03 (0.99 - 1.08)	0.133		
Household density	1.00 (0.94 - 1.06)	0.901		
Diabetes status				
Normal	Ref			
Prediabetic	1.28 (0.75 - 2.18)	0.370		
Diabetic	1.25 (0.60 - 2.58)	0.547		

Note: Blank spaces under the multivariate column denote that the variable was not selected in the stepwise forward selection and was not included in the final model. Empty* denotes that no participant in that group was observed as hypertensive thus, no estimations were found.

3.3 Pathways between SES and hypertension

Figure 5 presents results from the direct path analysis. The variables used are the same as those that were used in the multivariable logistic regression model and the observed ORs in the direct

path analysis were consistent with those from the multivariable regression. Age, sex, and education had negative direct effects on hypertension with OR = 0.71 (95% CI: 0.63 - 0.81), OR = 0.82 (95% CI: 0.54 - 1.23), and OR = 0.98 (95% CI: 0.65 - 1.47) respectively. Between the five SES, the richest quintile had a negative effect on hypertension although no statistical significance was observed. Table 3 presents corresponding results from the direct path analysis, the exponentiated odds ratios were generated for comparison with those observed in the multivariable logistic regression.

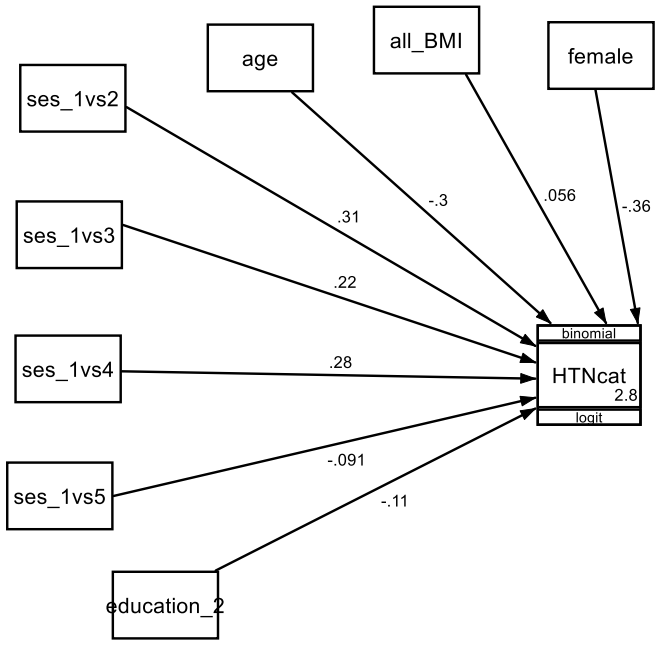


Figure 5: The direct path analysis of the different factors on hypertension displayed using GSEM.

Variables: Female (female), BMI (all_BMI), Age (age), SES poorest quintile (ses_1vs), SES poorer quintile (ses_1vs3), SES richer quintile (ses_1vs4), SES richest quintile (ses_1vs5), Secondary education (education_2)

Table 3: GSEM results showing the beta coefficients (β) and the exponentiated OR's from the direct path analysis.

Variable	Coefficient (95% CI)	Exponentiated Odds ratio (95% CI)	P-value
Age	-0.34 (-0.47, -0.21)	0.71 (0.63,0.81)	<0.001
Sex (Female)	-0.20 (-0.61, -0.20)	0.82 (0.54,1.23)	0.328
SES			
Poorest	0.18 (-0.33,0.68)	1.20 (0.72,1.98)	0.486
Poorer	0.17 (-0.34,0.67)	1.18 (0.72,1.95)	0.518
Middle	Ref	Ref	
Richer	0.001 (-0.55,0.55)	1.00 (0.58,1.73)	0.999
Richest	-0.38 (-1.21,0.44)	0.68 (0.30,1.55)	0.358
BMI (kg/m^2)	0.06 (0.02,0.11)	1.06 (1.02,1.11)	0.007
Education (secondary)	-0.02 (-0.43 - 0.38)	0.98 (0.65, 1.47)	0.907

Figure 6 presents the indirect path analysis that seeks to explore the influential factor of SES on the other factors related to hypertension. The indirect effect of age did not show statistical significance, but, the total effect remained statistically significant suggesting that with age, there is a 28% decreased risk of hypertension. The total effects of sex suggested that being female was associated with 2% odds of being hypertensive even though statistical significance was not reached. The total effects of all the SES quintiles also remained insignificant

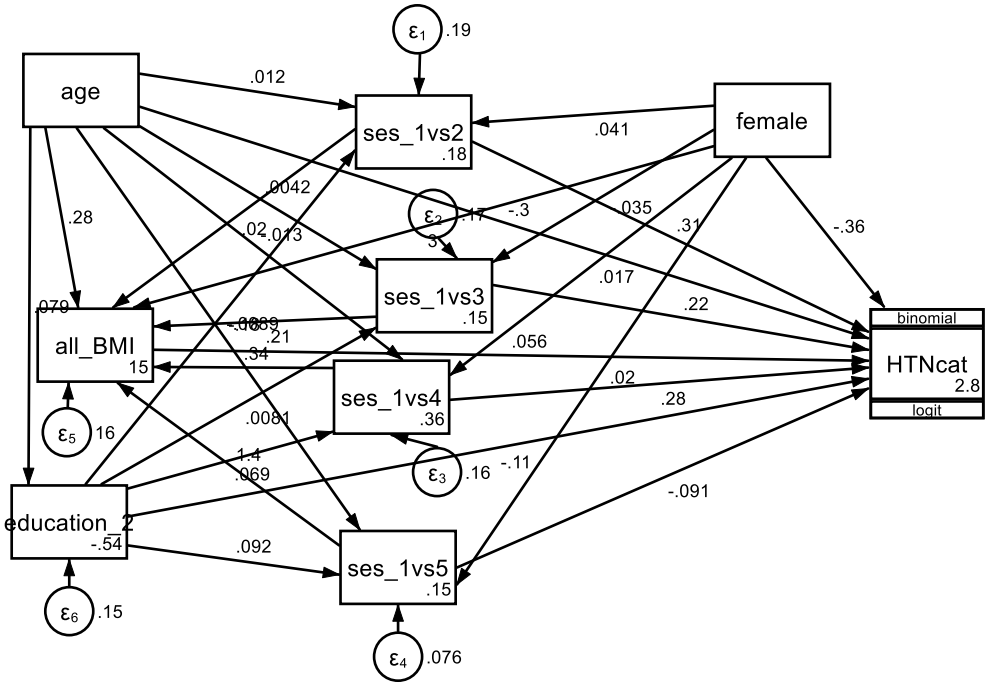


Figure 6:The indirect path analysis diagram of the different factors on hypertension displayed using GSEM

Variables: Female (female), BMI (all_BMI), Age (age), SES poorest quintile (ses_1vs), SES poorer quintile (ses_1vs3), SES richer quintile (ses_1vs4), SES richest quintile (ses_1vs5), Secondary education (education_2).

Table 4: GSEM results showing the OR's for indirect effects, direct effects, and total effects from the indirect path analysis

Variable	**Indirect Effects Odds ratio (95%CI)**	**Direct Effects Odds ratio (95%CI)**	**Total Effects Odds ratio (95%CI)**
Age	1.01 (0.98 - 1.05)	0.71 (0.6 - 0.81)	0.72 (0.63 - 0.82)
Sex (Female)	1.18 (1.03 - 1.36)	0.82 (0.54 - 1.23)	0.98 (0.67 - 1.39)
SES			
Poorest	0.98 (0.92 - 1.03)	1.20 (0.72 - 1.98)	1.17 (0.70 - 1.95)
Poorer	1.00 (0.94 - 1.06)	1.18 (0.72 - 1.95)	1.18 (0.71 - 1.97)
Middle	Ref	Ref	Ref
Richer	1.00 (0.94 - 1.07)	1.00 (0.58 - 1.73)	1.00 (0.58 - 1.73)
Richest	1.04 (0.94 - 1.16)	0.92 (0.49 - 1.74)	0.71 (0.31 - 1.65)
BMI (kg/m^2)	None	1.06 (1.02 - 1.11)	1.06 (1.02 - 1.11)
Education (secondary)	0.92 (0.82 - 1.04)	0.98 (0.65 -1.47)	0.90 (0.60 - 1.35)

4.0 Discussion

This chapter presents the discussion of the main findings of the study, strengths, and limitations of the study, conclusion, and recommendations.

The main findings of this study were that the prevalence of adolescent hypertension is high and there was no obvious relationship between SES and hypertension.

4.1 The relationship between SES and hypertension.

The main objective of this study was to describe the relationship between SES and hypertension in this study of SA adolescents. There was no relationship between SES and hypertension. The findings of no association between SES and hypertension were consistent with previous studies. Kagura and colleagues from their study in SA where they investigated the effect of SES change from low to high between childhood and adolescence. They observed no association between SES mobility from low to high between childhood and adolescence (34). Similarly, Hallal and colleagues in their study in Brazil also found no association between SES change between childhood adolescence (35).

One of the possible explanations for our findings is that SES in the primary study was derived from a score that was given to each household and was based on the household's assets. This may not have been the best or the most sensitive indicator for SES which subsequently failed to describe the relationship. A meta-analysis by Leng and colleagues used education as a measure of SES (13), this is not possible in adolescents as most are still in school. This may be the reason for the inconsistent findings.

It is noteworthy that studies that explored the relationship between household income and hypertension also had complex but similar findings. Since 47.2% of our study participants was found in the lowest two SES while 28.5% were in the highest 2 quintiles, the prevalence of hypertension was high. These results are similar to previous findings where adolescents from low-income settings had higher BP levels despite having a normal weight. As it was with our study, the prevalence of hypertension was high and many were undiagnosed, this finding is similar to the study that was conducted on paediatric patients in North Carolina (42).

Again, these emphasize the intricacy of the cause of elevated blood pressure and how different factors need to be explored together with SES disparities.

4.2 Pathways between SES and hypertension

The third objective of this study was to describe the pathways through which SES influences the development of hypertension in South African adolescents. This was to explore the combination of direct and indirect ways in which SES may lead to hypertension in this population group. From the analysis of total effects, statistical significance was not observed in all five SES quintiles and the effect size was not drastically changed when comparing the results from the logistic regression and the GSEM. The decreased risk of hypertension with increased SES remained, but this was not significant. Thus, the influence of SES on BMI did not yield significant changes in its association with hypertension.

The total effects were not different and revealed a reduced risk of hypertension with an increase in age by a year. This result is in contrast with the meta-analysis conducted by Leng and colleagues which found that an increase in age results in an increased risk of hypertension (13). Literature has revealed that the prevalence of hypertension has been higher among adults than in adolescents, e.g. one study conducted in SA in 2017 suggested a high prevalence (42-54%) of hypertension (7). Another study in SA conducted on teenagers in their final year of high school suggested a prevalence of 13.7% (8).

The contrasting finding in our study could be explained by the disproportions in the number of participants among the different ages of our study participants. It was observed earlier in the study that 61.8% of our study participants were below the age of 18 which may have distorted the age factor.

4.3 Sex-specific prevalence of hypertension

The first objective of the study was to investigate sex disparities in the prevalence of hypertension in adolescents. In this study, a prevalence of 26% of hypertension was reported. This is higher than the prevalence found in previous studies of less than 10% prevalence in children and adolescents aged 6 to 19 years (7,8,24). Also, this is higher than the reported prevalence in SADHS 2016 of 17% among women and 20% among men aged 15-24 years (63). Nevertheless, the prevalence of 26 % found in this study is closer to the findings from a systematic review which was conducted in SA and reported a prevalence range of 7.5 % to 22% in childhood hypertension (25).

There was an observed difference in the prevalence of hypertension between males and females but was not tested for statistical significance. This was expected as it has been found previously on South Asian children in Birmingham where males had higher blood pressure and were more likely to have hypertension compared to females (28). Additionally, sex differences in blood pressure during adolescence show that boys usually have a higher blood pressure than girls, a study on white adolescents in Canada by Syme and colleagues showed similar results. This difference in blood pressures was been attributed to the difference in body composition between males and females (29). Even though there was no statistical significance in the association between sex and hypertension in this study, the results suggested that being female reduced the risk of hypertension by 4% in the bivariate analysis and by 18% in the multivariate analysis.

Ibekwe and colleagues in their study in Nigeria established that BMI is one of the factors that contribute to an increased prevalence of hypertension (12). In our study, while the majority of participants remained in the normal range of BMI (59.0%) while the prevalence of obesity and overweight was 11.1% and 5.7%, there was an observed relationship between BMI and hypertension status. When the association between BMI and hypertension was examined, there was a 6% increase in the risk of hypertension with every kilogram per meter squared increase in BMI.

4.4 Strengths and limitations of the study

The strength of this study is that was a national survey and the sample size was a large sample. Another strength was that the blood pressure records which made up the outcome variable were measured three times which limits the risk of misdiagnosed hypertension status. The first limitation of the study is that as a cross sectional study, the attempts to infer causality are tentative. The limitations of the study are that more confounding factors are associated with hypertension which were not measured in the DHS 2016 survey which may lead to biased results. Such factors include salt intake (65), stress (15,66), physical activity, alcohol intake, and domestic violence(50,51,67).

4.5 Conclusion

This study has shown that the prevalence of hypertension in South African adolescents is high but falls within the range of previous studies. The findings on hypertension prevalence in this study were higher than expected but consistent with some findings made in prior literature.

Surprisingly, there was no association between SES and hypertension, this indicates that wealth index may not be a sensitive indicator of SES in this age group which warrants further studies to be conducted using other proxies for SES. The high prevalence of hypertension in his study demonstrates how important it is to develop strategies that will address this high rise in the number of hypertensive adolescents and further decrease the chances of having more hypertensive adults in the future.

4.6 Recommendations

There is need for efforts in addressing the high prevalence of hypertension among children and adolescents. There is a need to work on this burden from the community level by engaging parents and children on the risks associated with hypertension. This study also supports the suggestion that there is a requirement for further studies which will make use of reliable and authentic diagnostic measures and find detail on the factors and risks associated with childhood hypertension.

We recommend cohort studies to follow adolescents through time and investigate pathways to hypertension. Further studies on SES, metabolism, and genetics and their contribution to adolescent hypertension are recommended. Prevention of childhood hypertension may play a big role in decreasing adult hypertension which will further contribute to a decrease in CVD.

5.0 References

1. Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, et al. 2020 International Society of Hypertension Global Hypertension Practice Guidelines. Hypertension. 2020;1334–57.

2. Acosta AA, Samuels JA, Portman RJ, Redwine KM. Prevalence of persistent prehypertension in adolescents. J Pediatr [Internet]. 2012;160(5):757–61.

3. De Venecia T, Lu M, Figueredo VM. Hypertension in young adults. Postgraduate Medicine. 2016.

4. Mills KT, Bundy JD, Kelly TN, Reed JE, Kearney PM, Reynolds K. Global Disparities of Hypertension Prevalence and Control. 2016;441–50.

5. World Health Organization. A global brief on Hyper - tension World Health Day. World Heal Organ. 2013;1–40.

6. Gómez-Olivé FX, Ali SA, Made F, Kyobutungi C, Nonterah E, Micklesfield L, et al. Regional and Sex Differences in the Prevalence and Awareness of Hypertension: An H3Africa AWI-Gen Study Across 6 Sites in Sub-Saharan Africa. Glob Heart. 2017;12(2):81–90.

7. Shisana, O, Rehle, T, Simbayi LC, Zuma, K, Jooste, S, Zungu N, Labadarios, D, Onoya D. South African National HIV Prevalence, Incidence and Behaviour Survey, 2012. HSRC Press. 2014.

8. Bhimma R, Naicker E, Gounden V, Nandlal L, Connolly C, Hariparshad S. Prevalence of Primary Hypertension and Risk Factors in Grade XII Learners in KwaZulu-Natal, South Africa. Int J Hypertens. 2018;2018:1–9.

9. Anchala R, Kannuri NK, Pant H, Khan H, Franco OH, Di Angelantonio E, et al. Hypertension in India: A systematic review and meta-analysis of prevalence, awareness, and control of hypertension. J Hypertens. 2014;32(6):1170–7.

10. Manus MB, Bloomfield GS, Leonard AS, Guidera LN, Samson DR, Nunn CL. High prevalence of hypertension in an agricultural village in Madagascar. PLoS One. 2018;13(8):1–12.

11. Shen Y, Chang C, Zhang J, Jiang Y, Ni B, Wang Y. Prevalence and risk factors associated with hypertension and prehypertension in a working population at high

altitude in China: A cross-sectional study. Environ Health Prev Med. 2017;22(1).

12. Ibekwe R. Modifiable risk factors of hypertension and socio-demographic profile in Oghara, Delta State; prevalence and correlates. Ann Med Health Sci Res. 2015;

13. Leng B, Jin Y, Li G, Chen L, Jin N. Socioeconomic status and hypertension. J Hypertens. 2015

14. Satoh A, Arima H, Ohkubo T, Nishi N, Okuda N, Ae R, et al. Associations of socioeconomic status with prevalence, awareness, treatment, and control of hypertension in a general Japanese population: NIPPON DATA2010. J Hypertens. 2017;35(2):401–8.

15. Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, Zhu S, et al. Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. Circulation. 2004;110(1):74–8.

16. Ewald DR, Haldeman LA. Risk Factors in Adolescent Hypertension. 2016;

17. WHO, UNICEF. Maternal, newborn, child and adolescent health. Global strategy for infant and young child feeding. 2003.

18. Puwar T, Saxena D, Yasobant S, Savaliya S. Noncommunicable diseases among school-going adolescents: A case study on prevalence of risk factors from Sabarkantha District of Gujarat, India. Indian J Community Med. 2018;

19. Noubiap JJ, Essouma M, Bigna JJ, Jingi AM, Aminde LN, Nansseu JR. Prevalence of elevated blood pressure in children and adolescents in Africa: a systematic review and meta-analysis. Lancet Public Heal. 2017;2(8):e375–86.

20. Genovesi S, Antolini L, Gallieni M, Aiello A, Mandal SKB, Doneda A, et al. High prevalence of hypertension in normal and underweight Indian children. J Hypertens. 2011;

21. Sharma V, Coleman S, Nixon J, Sharples L, Hamilton-Shield J, Rutter H, et al. A systematic review and meta-analysis estimating the population prevalence of comorbidities in children and adolescents aged 5 to 18 years. Obes Rev. 2019;20(10):1341–9.

22. Stewart CP, Christian P, Wu LSF, Leclerq SC, Khatry SK, West KP. Prevalence and

risk factors of elevated blood pressure, overweight, and dyslipidemia in adolescent and young adults in rural Nepal. Metab Syndr Relat Disord. 2013;11(5):319–28.

23. Akbari M, Moosazadeh M, Ghahramani S, Tabrizi R, Kolahdooz F, Asemi Z, et al. High prevalence of hypertension among Iranian children and adolescents: a systematic review and meta-analysis. 2017;1155–63.

24. Song P, Zhang Y, Yu J, Zha M, Zhu Y, Rahimi K, et al. Global Prevalence of Hypertension in Children: A Systematic Review and Meta-analysis. JAMA Pediatr. 2019;

25. Monyeki KD, Kemper HCG. The risk factors for elevated blood pressure and how to address cardiovascular risk factors: A review in paediatric populations. Journal of Human Hypertension. 2008.

26. Musinguzi G, Nuwaha F. Prevalence, Awareness and Control of Hypertension in Uganda. PLoS One. 2013;8(4).

27. Doulougou B, Kouanda S, Rossier C, Soura A, Zunzunegui MV. Differences in hypertension between informal and formal areas of Ouagadougou, a sub-Saharan African city. BMC Public Health. 2014;14(1):1–9.

28. Wolf-Maier K, Cooper RS, Banegas JR, Giampaoli S, Hense HW, Joffres M, et al. Hypertension Prevalence and Blood Pressure Levels in 6 European Countries, Canada, and the United States. J Am Med Assoc. 2003;289(18):2363–9.

29. Syme C, Abrahamowicz M, Leonard GT, Perron M, Richer L, Veillette S, et al. Sex differences in blood pressure and its relationship to body composition and metabolism in adolescence. Arch Pediatr Adolesc Med. 2009;163(9):818–25.

30. Bruce MA, Beech BM, Norris KC, Griffith DM, Sims M, Thorpe RJ. Sex, Obesity, and Blood Pressure Among African American Adolescents: The Jackson Heart KIDS Pilot Study. Am J Hypertens. 2017;30(9):892–8.

31. Grotto I, Huerta M, Sharabi Y. Hypertension and socioeconomic status. Current Opinion in Cardiology. 2008.

32. Cois A, Ehrlich R. Analysing the socioeconomic determinants of hypertension in South Africa: A structural equation modelling approach. BMC Public Health. 2014;14(1).

33. Cuschieri S, Vassallo J, Calleja N, Pace N, Mamo J. The Effects of Socioeconomic Determinants on Hypertension in a Cardiometabolic At-Risk European Country. Int J Hypertens. 2017;2017.

34. Kagura J, Adair LS, Pisa PT, Griffiths PL, Pettifor JM, Norris SA. Association of socioeconomic status change between infancy and adolescence, and blood pressure, in South African young adults: Birth to Twenty cohort. BMJ Open. 2016;6(3).

35. Hallal PC, Clark VL, Assunção MC, Araújo CLP, Gonçalves H, Menezes AMB, et al. Socioeconomic trajectories from birth to adolescence and risk factors for noncommunicable disease: Prospective analyses. J Adolesc Heal. 2012;51(SUPPL. 6):32–7.

36. Huang H, Wan Mohamed Radzi CWJ bt, Jenatabadi HS. Family environment and childhood obesity: A new framework with structural equation modeling. Int J Environ Res Public Health. 2017;14(2).

37. Fujiwara T, Ito J, Kawachi I. Income inequality, parental socioeconomic status, and birth outcomes in Japan. Am J Epidemiol. 2013;177(10):1042–52.

38. Adler NE, Newman K. Socioeconomic disparities in health: Pathways and policies. Health Aff. 2002;21(2):60–76.

39. Kawachi I, Adler NE, Dow WH. Money, schooling, and health: Mechanisms and causal evidence. Ann N Y Acad Sci. 2010;1186:56–68.

40. Kwok MK, Schooling CM, Subramanian S V., Leung GM, Kawachi I. Pathways from parental educational attainment to adolescent blood pressure. J Hypertens. 2016;34(9):1787–95.

41. Kaczmarek M, Stawińska-Witoszyńska B, Krzyżaniak A, Krzywińska-Wiewiorowska M, Siwińska A. Who is at higher risk of hypertension? Socioeconomic status differences in blood pressure among Polish adolescents: a population-based ADOPOLNOR study. Eur J Pediatr. 2015;174(11):1461–73.

42. Ewald DR, Bond SH, Haldeman LA. Hypertension in Low-Income Adolescents. Glob Pediatr Heal. 2017;4:2333794X1774181.

43. Jackson SL, Yang EC, Zhang Z. Income disparities and cardiovascular risk factors among adolescents. Pediatrics. 2018;142(5).

44. Wilson DK, Kliewer W, Plybon L, Sica DA. Healthy Black Adolescents. 2015;496–501.

45. Chorin E, Hassidim A, Hartal M, Havakuk O, Flint N, Ziv-Baran T, et al. Trends in Adolescents Obesity and the Association between BMI and Blood Pressure: A Cross-Sectional Study in 714,922 Healthy Teenagers. Am J Hypertens. 2015;28(9):1157–63.

46. Goel M, Pal P, Agrawal A, Ashok C. Relationship of body mass index and other life style factors with hypertension in adolescents. Ann Pediatr Cardiol. 2016;9(1):29–34.

47. Xu RY, Zhou YQ, Zhang XM, Wan YP, Gao X. Body mass index, waist circumference, body fat mass, and risk of developing hypertension in normal-weight children and adolescents. Nutr Metab Cardiovasc Dis. 2018;28(10):1061–6.

48. Gordon B, Shamiss A, Derazne E, Tzur D, Afek A. Sex differences in the association between body mass index and hypertension – a cross-sectional study in 717 812 adolescents. Pediatr Obes. 2016;11(4):317–20.

49. Ahanchi NS, Ramezankhani A, Munthali RJ, Asgari S, Azizi F, Hadaegh F. Body mass index trajectories from adolescent to young adult for incident high blood pressure and high plasma glucose. PLoS One. 2019;14(5):1–15.

50. Leary SD, Ness AR, Smith GD, Mattocks C, Deere K, Blair SN, et al. Physical activity and blood pressure in childhood: Findings from a population-based study. Hypertension. 2008;51(1):92–8.

51. Knowles G, Pallan M, Thomas GN, Ekelund U, Cheng KK, Barrett T, et al. Physical activity and blood pressure in primary school children: A longitudinal study. Hypertension. 2013;61(1):70–5.

52. Brage S, WEDDERKOPP, NIELS EKELUND U, FRANKS PW, WAREHAM NJ, ANDERSEN LB, FROBERG K. Features of the Metabolic Syndrome Are Associated With Objectively Measured Physical Activity and Fitness in Danish Children. Diabetes Care. 2004;27(9):2141–8.

53. Masala G, Bendinelli B, Versari D, Saieva C, Ceroti M, Santagiuliana F, et al. Anthropometric and dietary determinants of blood pressure in over 7000 Mediterranean women: The European Prospective Investigation into Cancer and Nutrition-Florence cohort. J Hypertens. 2008;

54. Li G, Wang H, Wang K, Wang W, Dong F, Qian Y, et al. The association between smoking and blood pressure in men: A cross-sectional study. BMC Public Health. 2017;17(1):1–6.

55. Leone A. Does smoking act as a friend or enemy of blood pressure? Let release Pandora's box. Cardiol Res Pract. 2011;1(1).

56. Guwatudde D, Nankya-Mutyoba J, Kalyesubula R, Laurence C, Adebamowo C, Ajayi IO, et al. The burden of hypertension in sub-Saharan Africa: A four-country cross sectional study. BMC Public Health. 2015;15(1):1–8.

57. Kiely DK, Gross AL, Kim DH, Lipsitz LA. The association of educational attainment and SBP among older community-living adults: The Maintenance of Balance, Independent Living, Intellect and Zest in the Elderly (MOBILIZE) Boston Study. J Hypertens. 2012;

58. Kautzky-willer A, Dorner T, Jensby A, Rieder A. Women show a closer association between educational level and hypertension or diabetes mellitus than males : a secondary analysis from the Austrian HIS. ??? [Internet]. 2012;12(1):1. Available from: ???

59. Hazuda HP, Patterson JK. Risk Factors for Cardiovascular Mortality in Mexican. 1990;131(3).

60. Hendriks ME, Wit FWNM, Roos MTL, Brewster LM, Akande TM, de Beer IH, et al. Hypertension in Sub-Saharan Africa: Cross-sectional surveys in four rural and urban communities. PLoS One. 2012;7(3).

61. Tu W, Eckert GJ, DiMeglio LA, Yu Z, Jung J, Pratt JH. Intensified effect of adiposity on blood pressure in overweight and obese children. Hypertension. 2011;58(5):818–24.

62. Blakemore SJ, Robbins TW. Decision-making in the adolescent brain. Nature Neuroscience. 2012.

63. Statistics South Africa. South Africa demographic and health survey 2016: Key indicator report. Statistics South Africa. 2017.

64. Zierle-Ghosh A, Jan A. Physiology, Body Mass Index (BMI). StatPearls. 2018.

65. Yang Q, Zhang Z, Kuklina E V., Fang J, Ayala C, Hong Y, et al. Sodium intake and blood pressure among US children and adolescents. Pediatrics. 2012;130(4):611–9.

66. Pickering T. Cardiovascular pathways: Socioeconomic status and stress effects on hypertension and cardiovascular function. Ann N Y Acad Sci. 1999;896(Cvd):262–77.

67. Gooding HC, Milliren CE, Austin SB, Sheridan MA, McLaughlin KA. Child Abuse, Resting Blood Pressure, and Blood Pressure Reactivity to Psychosocial Stress. J Pediatr Psychol. 2016;41(1):5–14.

6.0 Appendices

Appendix 1: Plagiarism declaration

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

FACULTY OF HEALTH SCIENCES

PLAGIARISM DECLARATION TO BE SIGNED BY ALL HIGHER DEGREE STUDENTS

SENATE PLAGIARISM POLICY: APPENDIX ONE

I Zisiwe Mahlati (Student number: 1475261) am a student registered for the degree of MSc Epidemiology in the academic year 2019.

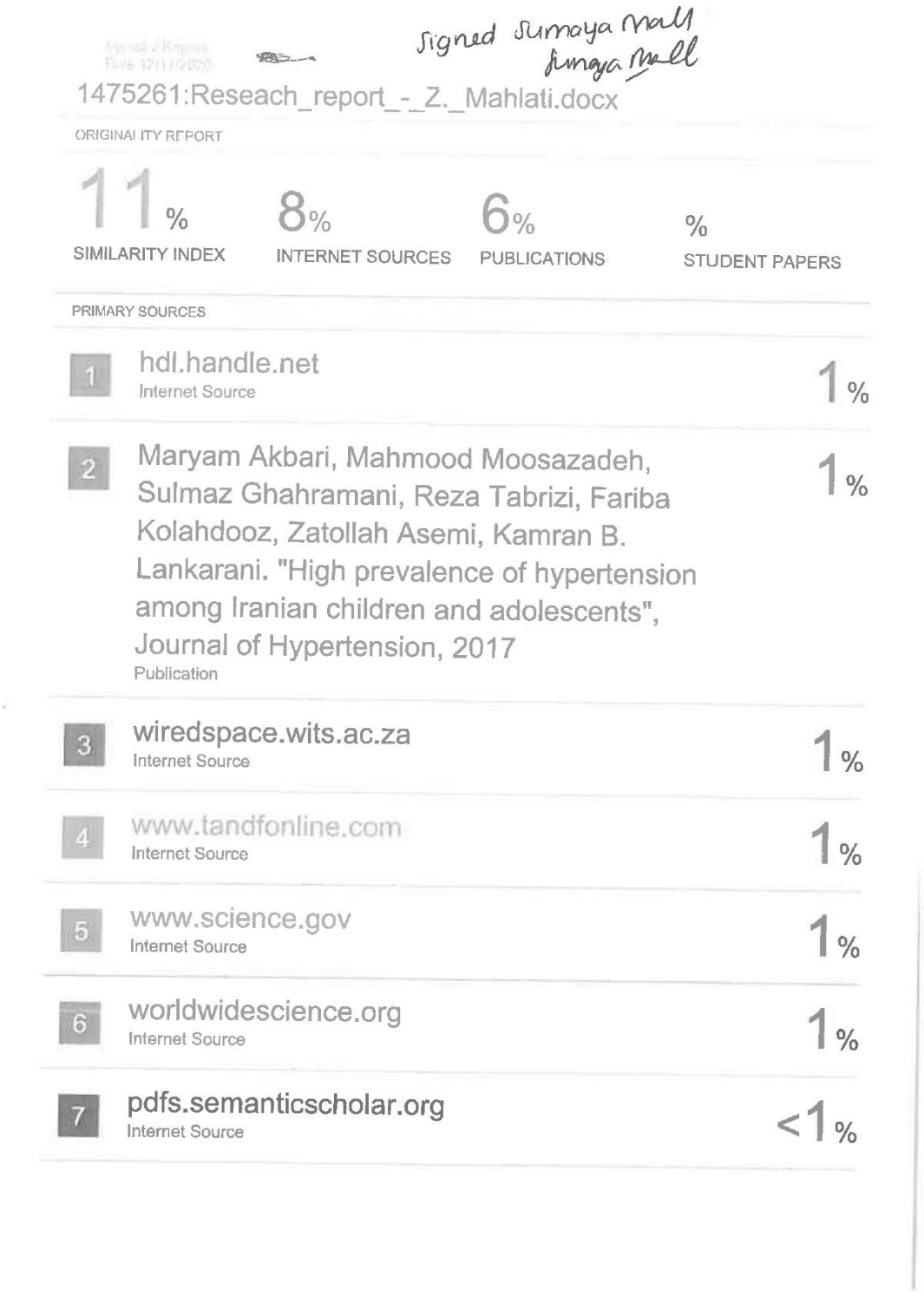
I hereby declare the following:

- I am aware that plagiarism (the use of someone else's work without their permission and/or without acknowledging the original source) is wrong.
- I confirm that the work submitted for assessment for the above degree is my own unaided work except where I have explicitly indicated otherwise.
- I have followed the required conventions in referencing the thoughts and ideas of others.
- I understand that the University of the Witwatersrand may take disciplinary action against me if there is a belief that this is not my own unaided work or that I have failed to acknowledge the source of the ideas or words in my writing.
- I have included as an appendix a report from "Turnitin" (or other approved plagiarism detection) software indicating the level of plagiarism in my research document.

Signature:  Date: 12/11/2020

1

Appendix 2: Turnitin report signed by supervisor



Signed Sumaya Nall
Sumaya Nall

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PRIMARY SOURCES

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2	Maryam Akbari, Mahmood Moosazadeh, Sulmaz Ghahramani, Reza Tabrizi, Fariba Kolahdooz, Zatollah Asemi, Kamran B. Lankarani. "High prevalence of hypertension among Iranian children and adolescents", Journal of Hypertension, 2017 Publication	1%
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Appendix 3: Ethics waiver certificate from the Human Research Ethics Committee of the University of the Witwatersrand.

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

06/01/2020

Ref: W-NN-200106-01

TO WHOM IT MAY CONCERN:

Waiver: This certifies that the following research does not require clearance from the Human Research Ethics Committee (Medical).

Investigator: Ms Z Mahlati
Student No. (if appropriate): 1475261
Staff No. (if appropriate):

Supervisor: Dr J Kagura

School: Public Health
Department: Epidemiology and Biostatistics
Medical School
University

Project title: *The relationship between socioeconomic status, stress and hypertension in South African adolescents*

Reason: Review of information in the public domain.
No human participants will be involved in the study.

Dr N Naran
Co-Chairperson: Human Research Ethics Committee (Medical)

Research Office Secretariat:
Physical address: Phillip Tobias Building, 3rd Floor, Office 302, Corner York Road and Princess of Wales Terrace, Parktown, Johannesburg 2193
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Office E-mail: HREC-Medical.ResearchOffice@wits.ac.za
Website:http://www.wits.ac.za/research/about-our-research/ethics-and-research-integrity/

Appendix 4: Confirmation of change of title

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Private Bag 3 Wits, 2050

Tel: 02711 7172076

Reference: Mrs Sandra Benn
E-mail: sandra.benn@wits.ac.za

10 September 2020
Person No: 1475261
TAA

Miss Z Mahlati
P O Box 12488
Amalinda
5252
South Africa

Dear Miss Zisiwe Mahlati

Master of Science in Epidemiology: Change of title of research

I am pleased to inform you that the following change in the title of your Research Report for the degree of **Master of Science in Epidemiology** has been approved:

From:	**The relationship between socioeconomic status, stress, and hypertension in South African adolescents.**
To:	**The Relationship Between Socioeconomic Status And Hypertension In South African Adolescents**

Yours sincerely

Mrs Sandra Benn
Faculty Registrar
Faculty of Health Sciences

Appendix 5: Table of comparison

Table 5: Comparison of differences between the study sample and the excluded sample.

Variable	Excluded sample	Included sample	Total	P-value
	n = 1165	**n = 1062**	**n = 2227**	
Age in years				
15 n (%)	196 (47.92)	213 (52.08)	409 (18.37)	0.164
16 n (%)	258 (51.81)	240 (48.19)	498 (22.36)	
17 n (%)	245 (54.57)	204 (45.43)	449 (20.16)	
18 n (%)	221 (51.16)	211 (48.84)	432 (19.40)	
19 n (%)	245 (55.81)	194 (44.19)	439 (19.71)	
Sex/Gender				
Male n (%)	172 (24.23)	538 (75.77)	710 (31.88)	<0.001
Female n (%)	933 (65.46)	524 (34.54)	1517 (68.12)	
Race				
African n (%)	1014 (51.06)	972 (48.94)	1986 (89.18)	0.005
White n (%)	19 (54.29)	16 (45.71)	35 (1.57)	
Coloured n (%)	123 (64.40)	68 (35.60)	191 (8.58)	
Indian/Asian n (%)	9 (60.0)	6 (40.0)	15 (0.67)	
SES				
Poorest n (%)	264 (49.07)	274 (50.93)	538 (24.16)	<0.001
Poorer n (%)	282 (55.40)	227 (44.60)	509 (22.86)	
Middle n (%)	231 (47.24)	258 (52.76)	489 (21.96)	
Richer n (%)	223 (51.15)	213 (48.85)	436 (19.58)	
Richest n (%)	165 (64.71)	90 (35.29)	255 (11.45)	
Type of residence				
Urban n (%)	613 (52.62)	474 (44.63)	1087 (48.81)	<0.001
Rural n (%)	552 (47.38)	588 (55.37)	1140 (51.19)	
Marital status				
Never married n (%)	1141 (52.08)	1050 (47.92)	2191 (98.38)	0.050
Married n (%)	24 (68.57)	11 (31.43)	35 (1.57)	
Divorced/Separated n (%)	0 (0)	1 (100.0)	1 (0.04)	
Education				
Primary n (%)	146 (41.01)	210 (58.99)	356 (15.99)	<0.001
Secondary n (%)	1019 (54.46)	852 (45.54)	1871 (84.01)	
BMI (kg/m^2)				
<18.5 n (%)	22 (8.15)	248 (91.85)	270 (12.12)	<0.001
18.5-24.9 n (%)	55 (8.06)	627 (91.94)	682 (30.62)	
25.0 - 29.9 n (%)	16 (11.94)	118 (88.06)	134 (6.02)	
> 29.9 n (%)	11 (15.49)	60 (84.51)	71 (3.19)	
Missing n (%)	1061 (99.16)	9 (0.84)	1070 (48.05)	
Average Blood Pressure				
SBP median (IQR)	122 (20)	119 (17)	119 (17)	
DBP median (IQR)	80 (15)	76 (12)	76 (12)	
Household density median (IQR)	5 (3)	5 (3)	5 (3)	
Use of tobacco				
Yes n (%)	348 (25.95)	993 (74.05)	1341 (60.22)	<0.001
No n (%)	37 (34.91)	69 (65.09)	106 (4.76)	

Missing n (%)	780 (100.0)	0 (0)	780 (35.02)	
Glucose Homeostasis				
Normal n (%)	12 (8.22)	134 (91.78)	146 (6.56)	<0.001
Prediabetic n (%)	47 (6.41)	686 (93.59)	733 (32.91)	
Diabetic n (%)	8 (8.60)	85 (91.40)	93 (4.18)	
Missing n (%)	1098 (87.49)	157 (12.51)	1255 (56.35)	

Appendix 6: List of variables and sources table

Table 6: List of variables used in the study and their sources on the DHS datasets

Variable	Variable categories and codes	Analysis categories and codes	Variable source and (name)
Age	Continuous variable	Continuous variable	PR* (hv105)
Sex	Male - 1 Female - 2	Male - 1 Female - 2	PR* (hv104)
Race	Black/African - 1 White - 2 Coloured - 3 Indian/Asian - 4	Black/African - 1 White - 2 Coloured - 3 Indian/Asian - 4	MR* and IR* (mv131 and v131)
SES	Poorest - 1 Poorer - 2 Middle - 3 Richer - 4 Richest - 5	Poorest - 1 Poorer - 2 Middle - 3 Richer - 4 Richest - 5	PR* (hv270)
Type of residence	Urban - 1 Rural - 2	Urban - 1 Rural - 2	PR* (hv025)
Marital status	Never married - 0 Married - 1 Divorced/Separated - 4	Never married - 0 Married - 1 Divorced/Separated - 4	PR* (hv115)
Education	No education, preschool - 0 Primary - 1 Secondary - 2 Higher - 3 Don't know - 8	Primary - 1 Secondary - 2	PR* (hv106)
BMI (kg/m^2)	Initially a continuous variable	<18.5 - 1 18.5-24.9 - 2 25.0 - 29.9 - 3 > 29.9 - 4	PR* (ha40 (female)) (hb40 (male))
Household density	Continuous variable	Continuous variable	PR* (hv009)

Use of tobacco	Initially a continuous variable	Yes - 1 No - 0	PR* (ha35 (female)) (hb35 (male))
Glucose Homeostasis	Initially a continuous variable	Normal - 0 Prediabetic - 1 Diabetic - 2	MR* (shmhba1c) IR* (shwhba1c)
		Outcome variable	
Hypertension	Initially a continuous variable	Yes - 1 No - 0	PR* (sh221a, sh221b, sh228a, sh228b, sh232a sh323b, sh321a, sh321b, sh328a, sh328b, sh332a, sh332b)

*PR denotes the Personal recode dataset from DHS. *MR denotes Men's recode dataset from DHS. *IR denotes Individual recode dataset which is the women recode.