THE ASSOCIATION BETWEEN EXPERIENCING VIOLENCE AND HYPERTENSION IN URBAN SOUTH AFRICAN ADOLESCENTS

UNIVERSITY OF THE
WITWATERSRAND,
JOHANNESBURG

By

Adatia Chivafa

(Student no: 1839407)

Supervisor 1: Dr Juliana Kagura

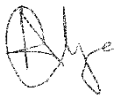
Supervisor 2: Dr Innocent Maposa

This research report is submitted to the Faculty of Health Sciences, the University of the Witwatersrand in part fulfilment of the requirements for the degree of Master of Science in Epidemiology in the field of Biostatistics

September 2020

DECLARATION

I, Adatia Chivafa, declare that this report: **The association between experiencing violence and hypertension in urban South African adolescents**, is my own original work and that all sources that I have used have been indicated by complete references. This report is being submitted for the degree of Master of Science in Epidemiology in the field of Biostatistics at the University of the Witwatersrand, Johannesburg, South Africa. It has not been submitted before for any degree at this or any other University.

Signature: 

Date: 28-09-2020

Dedication

To the wind beneath my wings:

Sister Gee and Mom, your unwavering support and faith in me is appreciated, this one is for you.

Abstract

Introduction: Experiencing violence in childhood has been shown in adult studies to be linked with the development of hypertension and other cardiovascular disease (CVD). Understanding this relationship in earlier years and especially in a country burdened by violence like South Africa (SA) could improve hypertension screening and support in adolescents who experience violence. This study, therefore, aims to determine the pathways through which experiences of violence affects blood pressure (BP) in 18-year-old adolescents in the Soweto-Johannesburg Metropolis

Materials and Methods: This study utilised data collected at a follow-up visit (at age 18) from an on-going longitudinal study on child and adolescent health and development, the Birth-to-Twenty plus cohort (BT20+). This study included all non-pregnant 18-year-olds with BP assessments at the 18th year follow-up visit. BP and anthropometric measures were collected by experienced assistants using standardised tools and other data collected through interviewer-administered face-to-face and self-administered questionnaires. Continuous variables were described using the mean and standard deviation (SD) or the median and interquartile range (IQR). Categorical variables were described using frequencies and percentages and they were all categorised by hypertension status. Structural equation modelling (SEM) and generalised structural equation modelling (GSEM) were used to determine the pathways through which violence affects BP. All estimates were evaluated at 5% level of significance.

Results: After exclusions 1630 participants remained, 44% were female, 87% were black, and 22% percent used alcohol. Fifteen percent of the participants were hypertensive, 54% male and 46% female. Body mass index (BMI), waist-to-hip ratio (WHR), and parity were significantly higher ($p<0.001$, $p<0.001$, and $p=0.006$ respectively) among hypertensives. Income, however, was R2400 lower ($p=0.044$) among hypertensives when compared to normotensives.

From the GSEM we found that experiences of violence had no statistically significant association with hypertension though the odds indicated an upward trend [OR: 1.01; CI: 0.97-1.06]. A unit increase in BMI was associated with an increase in odds [OR: 1.04; CI: 1.01-1.09] of hypertension. Sex mediated the association between experiences of violence and hypertension [OR: 0.61; CI: 0.53-0.69] also BMI and hypertension [OR: 15.09; CI: 8.99-25.31]. Alcohol use, in turn, mediated the association between experiences of violence and hypertension [OR: 0.87; CI: 0.78-.97].

The SEM models also did not show any statistically significant associations between experiences of violence and SBP [β=-0.22; CI: -1.57-1.12] or DBP [β=0.66; CI: -0.509-1.828]. An increase in BMI was associated with an increase in SBP [β=0.729; CI: 0.55-0.90] and DBP [β=0.32; CI: 0.17-0.47], females had significantly lower SBP [β=-7.24; CI: -8.86 to -5.62] than males. Sex mediated the association between violence with SBP [β=-0.51; CI: -0.64 to -0.37] and DBP and the association between BMI with SBP [β=2.69; CI: 2.15-3.23] and DBP inversely. Adolescents born to mothers with more children also had an increased likelihood of having high SBP [β=0.86; CI: 0.09-1.64], while those born to single [β=-1.94; CI:-3.39 to -0.49], older [β=-0.16; CI: -0.17 to -0.02], and mothers with higher education [β=-3.02; CI: -5.77 to -0.27] had a reduced likelihood of having high DBP.

Conclusions: Though there were no significant direct effects between experiencing violence and SBP, DBP, and hypertension among 18-year-old urban adolescents, study findings were indicative of an associated increase in hypertension and DBP with increasing experiences of violence. The mediation of sex between experiences of violence, BMI and the BP variables suggests two risk factors requiring intervention during adolescence. Interventions among males towards positive coping strategies and females towards weight control and body positivity early in life could see a significant decrease in the risk of developing hypertension.

Acknowledgements

I am particularly grateful for the advice, time, encouragement, and patience from my two supervisors; Dr Juliana Kagura and Dr Innocent Maposa that have been invaluable in the compiling of this report.

My appreciation goes to the Sub-Saharan Africa Consortium for Advanced Biostatistics (SSACAB) for funding that made undertaking this master's training possible.

Assistance, advice, and support offered by Prof. Jonathan Levin and Dr Jude Igumbor during the compilation of this report is greatly appreciated.

I am grateful to the DSI-NRF Centre of Excellence in Human Development for allowing me access to the BT20 data.

My sincere gratitude goes to my fellow Biostatistics track classmates who made this journey memorable, enjoyable, and bearable. I also would like to extend special thanks to Aphiwe Dinga, Zvifadzo Matsena-Zingoni, Chenai Mlandu, Victor Williams and staff in the division of Epidemiology.

I wish to acknowledge my family and friends for their unwavering support and forbearance throughout this journey.

Table of Contents

List of Figures

List of Tables

CHAPTER ONE

INTRODUCTION

This chapter gives insight on the size of the problem under consideration, reviews literature in context of the problem and discusses the strengths and gaps of previous studies on the problem. It also clearly outlines the aim and objectives of the current study, states the problem statement and justifies why it is worth investigating.

1.1. Background

Hypertension is considered a leading and preventable risk factor for premature disability and death; it is a syndrome that affects the cardiovascular system gradually and is usually detected by the presence of elevated blood pressure (BP) which is defined as "systolic and/or diastolic blood pressure equal to or above 140/90 mmHg". Hypertension can be classified into two i.e. primary and secondary hypertension. Secondary hypertension is as a result of underlying disease or conditions like adrenal gland malfunctions, renal failure, renovascular disease, among others. Primary hypertension, however, is responsible for more than 95% of all hypertension cases and more variable in its causes. These could include alcohol intake, high salt consumption, lack of exercise, obesity, low potassium consumption, among others (1–4).

It remains a major health problem affecting over 1.3 billion people worldwide (5) and is one of the key modifiable risk factors for cardiovascular disease (CVD) accounting for more than 7 million annual deaths worldwide. The past four decades have seen a shift in the burden of hypertension from high income (HIC) to low-to-medium-income countries (LMIC). Reducing the upward trend of hypertension is predicted to significantly reduce premature death due to CVD (6) hence understanding the aetiology of hypertension is essential.

1.1.1. Global Prevalence of Hypertension

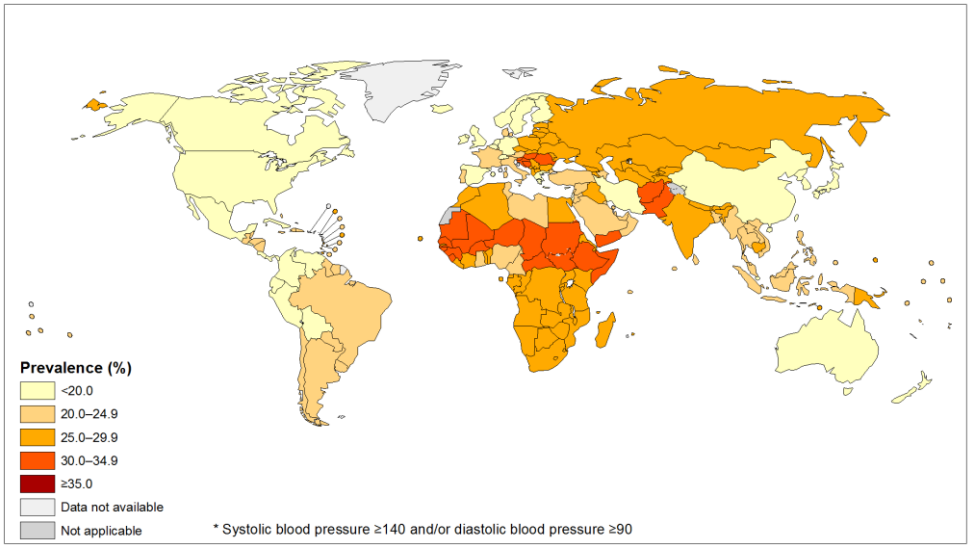


Figure 1: Global raised blood pressure prevalence by WHO region

Source: WHO Global Health Observatory (GHO) maps (Available at http://gamapserver.who.int/mapLibrary/Files/Maps/Global_BloodPressurePrevalence_2015_BothSexes.png)

In the past forty years, the absolute burden of hypertension is reported to have increased by 90% globally (7). The 2017 global burden of disease (GBD) estimates that high blood pressure is responsible for 10.4 million (9.39-11.5 million) deaths worldwide and 218 million (198-237 million) disability-adjusted years (DALYs)(3). The World Health Organisation (WHO) reports the highest hypertension burden to be in Africa with an overall prevalence of 30% in all adults(8,9). The WHO however only uses crude estimates from published and unpublished literature of studies that take random samples of the general population and have clearly indicated survey methods and risk factor definitions. This leads to an underestimation in areas where such surveys are in short supply and comprehensive hypertension data is limited, for example in sub-Saharan Africa (SSA), some reviews report having an average of 0.83 data sources per country indicating countries without any data (7,10).

Previously hypertension has been believed to be a condition more prevalent in the rich but recent trends show significantly higher prevalence in lower-income brackets. The disparities in the prevalence of hypertension between LMIC and high-income countries are continuously increasing with almost 75% of hypertensive people residing in LMIC regions (11).

Between 2000 and 2010 the highest increase in the absolute burden of disease was observed in East Asia & Pacific, South Asia and SSA with reports of more than 7% increase in the prevalence of hypertension (11). Robust information on hypertension in SSA is not as readily available as in higher-income regions but the last 10 years have seen an increase in studies focusing on hypertension but still not enough large studies exploring this major health problem. This has led to insufficient treatment and awareness and very limited understanding of the extensiveness of the problem of hypertension in SSA(12,13).

A 2017 study attempted to bridge the information gap and quantify issues of control, awareness and prevalence in SSA using data on 40- to 60-year-olds based in 6 sites of 4 countries in 3 SSA regions; West, East and Southern Africa. The study reported an overall prevalence of 33.3% (35% in females and 31% in males) but large differences were observable between sites with one site having a prevalence as low as 15% and another as high as 54.1% (12).

1.1.2. Hypertension in South Africa

The prevalence of hypertension in South Africa (SA) had a rather sudden and sharp increase in the past twenty years and is reported to have increased by more than 25% between 1997 and 2007 (14). Ever since then hypertension has become a growing problem that has not been resolved with the quadruple burden of disease (communicable disease, perinatal and maternal mortality, non-communicable diseases (NCDs) and, injury-related disorders) and the epidemiological transition that SA is currently experiencing from communicable to NCDs (15); see Figure 2.

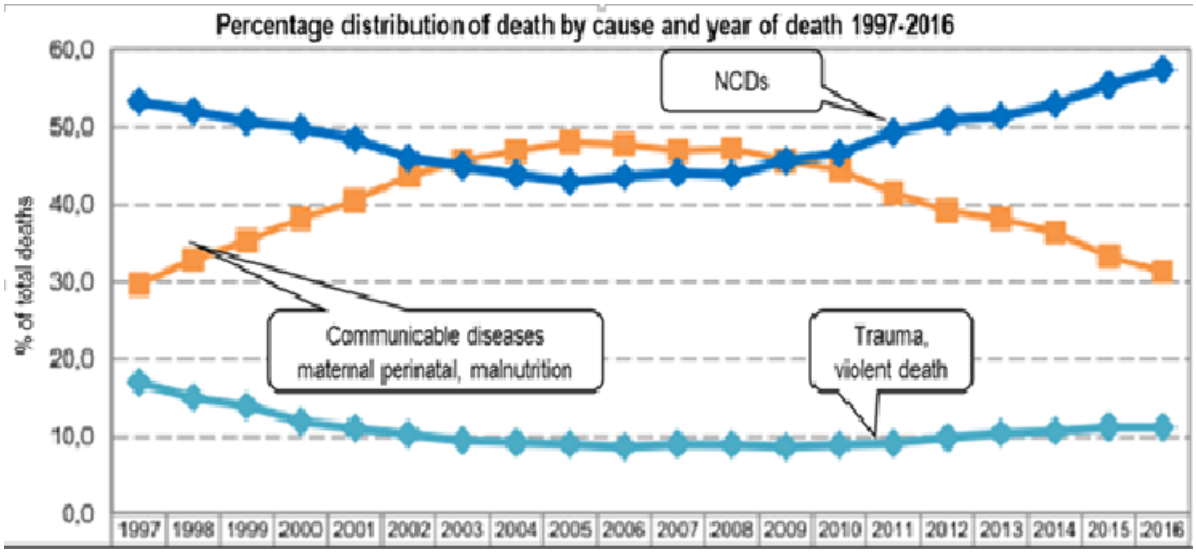


Figure 2: The Epidemiological shift in South Africa (1997 to 2016)

Source: Statistics South Africa (Available at https://www.sancda.org.za/ncds-increasingly-the-main-cause-of-death-in-south-africa-time-for-action/)

Most studies on hypertension in SA have not simultaneously evaluated the state of hypertension across all age groups and hence do not provide a representative picture of the nation. Interpretability and reliability of burden of disease studies have been in question for some years because of the incompleteness of the death registry though great effort and progress has been made in bettering the completeness in recent years making for better estimates that still require careful analysis (16,17). The initial burden of disease study in SA reported hypertensive disease as the 6th leading cause of death in the year 2000. NCDs accounted for 24.2% of the years of life lost (YLL); 21.5% in males and 22.8% in females (17). Fifteen years later hypertensive heart disease still ranks in the top 10 leading causes of death and disability in SA. A number of studies have ranked SA to have the highest burden of hypertension; with a significant portion of the population unaware that they have hypertension, the prevalence is estimated to be around 77% (12,18).

1.1.3. Paediatric hypertension-An emerging problem

A matter that has received little attention in the past that is emerging as an important public health concern is elevated blood pressure in children and adolescents (19). Adolescents and children with primary hypertension are likely to grow into hypertensive adults (20) therefore an exploration of issues relating to the development of hypertension in children is important. Being exposed to and /or experiencing violence in childhood has been shown to increase the odds of hypertension in adults (6). Violence in SA is particularly high; injuries due to violence are the second leading cause of death with South Africans experiencing at least one violence-related traumatic event in their lifetime (7). The relationship between violence and hypertension could be more pronounced during early life where their growth and development including physiological and metabolic processes are highly susceptible to environmental cues, according to several hypotheses(21–23).

1.2. Literature

This section reviews the existing studies on paediatric and adolescent hypertension, risk factors for hypertension, takes stock of the situation in SA and assesses links between violence, social stressors and ill health.

1.2.1. Childhood/Adolescent Hypertension

Previously children and adolescents have been thought to only have secondary hypertension, but recent studies have shown that children and adolescents are also experiencing primary hypertension (24–27). Global estimates of hypertension in children are not known due to classification differences and hypertension being considered mostly an adulthood condition hence hypertensive adolescents are underdiagnosed(28). Outcome data for hypertensives like cardiovascular disease and mortality are also not readily available for children and adolescents.

It is estimated that 75% of hypertensive children and adolescents remain undiagnosed(29) only to be diagnosed in later adulthood when the condition is more advanced and potent. Reviews of prevalence estimates are unfortunately hampered by differing measurement methods, some do not align to current guidelines and others are one-centre studies that are not representative of the population, this makes it difficult to determine the demographics of hypertension(26,30).

A cross-sectional Spanish study in Valencia among 652 children aged 6 to 9 years old from a longitudinal cohort aimed at measuring the prevalence of arterial hypertension estimated the prevalence of hypertension to be 8%. The study associated age-controlled hypertension with diet, anthropometric measurements (BMI, waist circumference, percentage fatness, and set of folds), and sex. The possible covariates were however treated independently and not adjusted for each other in Analysis of Covariance (ANCOVA) analyses. This possibly biased the findings of the study because it did not take into account any other confounding apart from age and no mediation(31). Fan et. al. (2019) estimated the prevalence of prehypertension in Chinese children aged 6 to 17 over 20 years from a national survey. The study found a prevalence of 6% over the survey years and used logistic regression to identify risk factors which were among others age, BMI, and WHR (32). A study in Central India of 11312 children aged between 5 to 15 found a prevalence of 6.8% among males and 7.0% among females and this difference was not statistically significant. Risk factors were identified using linear regression on SBP and DBP only but not on hypertension (33). Hypertension and its correlates among Australian pre-adolescents were estimated recently using a cross-section of 7139 children aged 10 to 12-years from a longitudinal survey of Australian children called Growing up in Australia. The estimated prevalence of hypertension was 5.4% and multiple linear regression was used on blood pressure to identify risk factors in this population which included BMI, Socioeconomic status (SES), hypertension in pregnancy (mother's), birth weight, and sports participation. The regression analysis was conducted on blood pressure adjusted for age, height, and sex.

In Africa, studies on childhood hypertension are still few and robust information on this phenomenon is scarce. The estimates of hypertension among children are quite varied ranging between 0.2% and 24.8%. To bridge this information gap and provide reliable estimates for Africa, Noubiap et al. (2017) reviewed studies in Africa on childhood and adolescent hypertension from 1996 to 2017 using high and medium quality studies. The review only involved 51 studies with almost 20% of the papers having an "unacceptable" definition of hypertension and only 25 for the pooled analysis. The studies unfortunately only captured data from 13 countries which only goes to show the dearth of information in the African region. They estimated the prevalence of elevated BP in Africa to be 5.5%, that of slightly elevated BP to be 12.7% though it rose to alarming proportions amongst obese children (30.8%) (34). More recent studies in SSA have estimated higher prevalence. A 2018 study in Dar es salaam, Tanzania aimed at estimating prevalence and risk factors of elevated BP among adolescents and children aged 6 to 17 found a prevalence of 10.8% from 446 participants. The study utilised log-binomial regression models to identify risk factors which included BMI, height, age, sex, and overweight or obesity (35). Prevalence of hypertension in a sample of high school age adolescents aged 10 to 19 sampled using multistage sampling was 6.3%. Potential risk factors were adjusted for using multivariable logistic regression on hypertension status (36).

Some studies did not adjust for potential confounding amongst the risk factors and some did, but all did not adjust for any potential mediation of some risk factors. They did not consider any potential indirect effects between the risk factors and hypertension.

1.2.2. Risk factors for childhood/adolescent hypertension

Primary hypertension in children and adolescents is predicted by lifestyle and genetics and its epidemiology and aetiology are still under study. Ewald and Halderman (29) group the factors contributing to childhood hypertension into seven groups; metabolic, diet and sleep, energy imbalance, medical, family history, genetic influences and psychosocial factors. These risk

factors can be classified into two; modifiable (preventable) and non-modifiable (non-preventable).

1.2.2.1. Modifiable risk factors

Modifiable risk factors are usually lifestyle-related; characteristics, exposures and behaviours that can be adjusted by an individual. These include dietary habits, sodium intake, poor sleeping duration and quality, passive smoking, mental health, diabetes, activity levels, central adiposity and being overweight.

Several studies have made an attempt to associate risk factors of hypertension in children and adolescents and the most documented include being overweight and obese with hypertension prevalence being much higher in obese and overweight children (37,38). The 2017 review on the prevalence and incidence of hypertension in children and adolescents spanning 22 years for children aged 2 to 19 estimated hypertension to be six times lower in normal-weight children when compared to obese children (34). Another study on 14-year olds participating in the Physical Activity and Health Longitudinal Study (PAHL study) from the North Western province in SA estimated the prevalence of hypertension to be 2 to 3 times higher in overweight adolescents (39). Wu et. al. (2013) found that among 7 to 10-year olds abdominal adiposity was higher in males than females and significantly associated with increasing blood pressure in children as blood pressure was up to six times higher in boys with abdominal obesity(40).

Lack of physical activity is another well-established modifiable risk factor for hypertension in children and adolescents. Several studies have reported this association across all age groups and genders. In investigating multiple cardiovascular risk factors among adolescents Jardim et. al. found that lack of physical activity was significantly higher in females than males and it increases the chances of having adolescent hypertension (41). A South African study among

14-year old adolescents agreed with this finding as they found that adolescents with low cardiovascular fitness had a higher prevalence of hypertension (39).

1.2.2.2. Non-modifiable factors

Non-modifiable risk factors are inherent characteristics and attributes of an individual that place them at a higher risk of being hypertensive and little to nothing can be done to change these. They include race, socio-economic inequalities, sex, family history, low birth weight, premature birth and hormonal imbalances.

Sex appears to be an important risk factor for hypertension in children and adolescents with several studies citing a significant relationship between these two. Akbari et. al. after reviewing studies with children aged 3 to 18 concluded that the prevalence of hypertension was higher in males (10.9%) than females (9.1%) (42). A review in Brazil, though hampered by differing classification methods also found that males had a higher prevalence of hypertension than females which is consistent with findings in other studies including another more recent study in SA among 12 to 17-year olds (41,43). However, an African review amongst 2 to 19-year olds noted the difference in prevalence amongst males and females with males once again being reported to have a higher prevalence than females though this difference was not statistically significant (34). This may point to the fact that in Africa being male is not a risk factor for hypertension as it is in agreement with a review on adult hypertension which found no association between hypertension and sex (44).

Another important predictor for hypertension in childhood is socioeconomic status, Noubiap et al. (2017) suggest that this risk factor is easier to observe at an individual level than at country levels (34). Family history has also been cited as an important non-modifiable risk factor for the development of hypertension in children and adolescents. Few studies have investigated this association, but the significance is not questionable. A Brazilian cross-sectional study

among 12 to 17-year old adolescents found that adolescents with a family history of CVD were likelier to have hypertension and other CVD risk factors (41).

1.2.3. Childhood/Adolescent Hypertension in South Africa

There has been an increase of interest in childhood hypertension in SA and hence an increase in studies on childhood and adolescent hypertension in recent years. SA is the only country in Southern Africa with a somewhat substantial body of information on childhood hypertension and its aetiology. Nkwana (2019) sampled 1665 children with ages 5 to 15-years in Polokwane private schools from the Ellisras Longitudinal Growth and Health Study to find the association between fat patterning with blood pressure. Children in private schools belong to the upper-middle to upper SES group. Hypertension prevalence increased with age group with the 5 to 7-year olds having the lowest prevalence (3.6 in boys, 1.4 in girls) and the 11 to 15 age group having the highest (21.3 boys, 33.0 girls). Linear regression was used to identify risk factors for age and gender-adjusted SBP and DBP, and logistic regression for hypertension (45). Another recent study among grade 7 learners in KwaZulu Natal (KZN) estimated the prevalence among adolescent with a mean age of 17.8 years to be 13.7%. Risk factors were considered individually in relation to a sex-adjusted hypertension status (46). Another Ellisras study in a rural setting of the Western Cape where the children were mostly drawn from the lower-income bracket estimated hypertension among 1811 participants aged 5 to 16 years. The overall prevalence was 1.3% and higher among underweight children (47). The study did not look at the associated risk factors because it was only interested in the blood pressure status of the children and adolescents.

There are several earlier studies on paediatric hypertension in SA but these three give us a fair picture of hypertension across age, SES status and rural/urban dwelling. There is currently

scarce demographically representative information for all South African children on hypertension. The estimated prevalence of paediatric hypertension is variable across population groups (income, SES, rural/urban, race, etc.); it is reported to be as low as 1.3% and as high as 33% in some population sub-groups.

1.2.4. Prevalence of Violence experience in Children and Adolescents

Globally, 50% of children (aged 2-17 years) are estimated to experience physical, emotional, sexual violence or some kind of neglect in the previous year with Africa exceeding 80% prevalence (48). A review of 13 SSA demographic health survey (DHS) data estimates that at least 10% of females aged 15-17 have experienced at least one form of sexual violence (49). A study in SA estimated the proportion of 15-17 year-olds that face lifetime victimization to be 20% for sexual abuse, 34% for physical abuse, 16% for emotional abuse (50) and 22.2% for school abuse (51). A characterization of violence in the birth-to-twenty cohort, a longitudinal study of children in the Soweto-Johannesburg Metropolis reports 99% of the children have experienced or been exposed to at least one type of violence (52). The leading perpetrators of violence are household members, peers and in adolescents; intimate partners (53) which means that most of the violence is in the home, community and the school.

1.2.5. Predictors for experiencing violence

The family structure appears to be an important predictor for experiencing violence during childhood. Orphans are likelier to experience victimization during childhood; a study using 13 sub-Saharan countries on female adolescents aged 15-17 reports that the odds of experiencing violence increase with paternal orphaning, double orphaning, and paternal absence (49). A Swaziland study identifies experiencing emotional abuse or maternal death before the age of 13 and living with three or more families in childhood as predictors of experiencing childhood violence (54) which is consistent with the findings of the violence against children VACS

studies. A study in six LMIC in Africa and Asia also agrees with other studies and identifies age, longer exposure to violence, paternal absence for females, enrolment into school for females especially as it appears peers and teachers are also perpetrators of violence (55) as predictors of experiencing childhood violence. Other predictors for experiencing different kinds of violence are socio-economic status, sex, education, an adult in the home who is ill continuously for at least 3 months, harmful community practices, stress, peer relations, parents' history of substance abuse, social security and ability to confide in family members (56–59).

1.2.6. Experiences of Violence and its implications on health

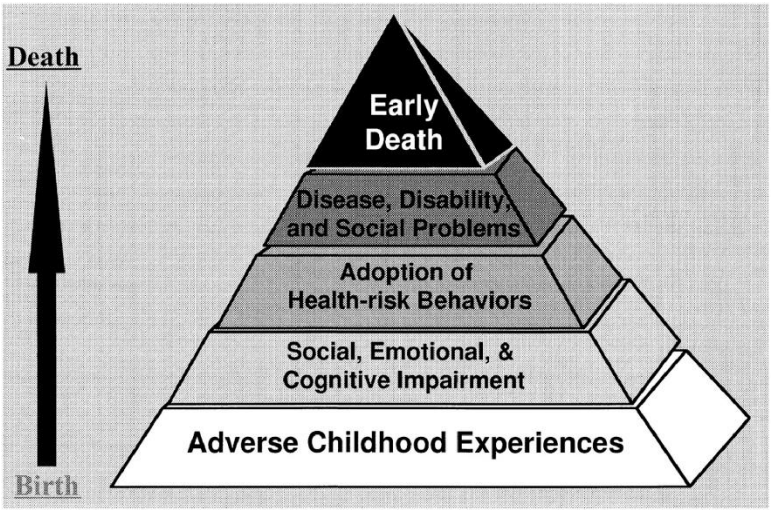


Figure 3: Potential influences throughout the lifespan of adverse childhood experiences (Source: Felitti et.al.(60))

Life-course theory suggests that through exposures that occur during a critical developmental period or accumulation of exposures, experiences and exposures to childhood violence can adversely affect adult health outcomes and consequentially death as shown in *Figure 3.* Neurobiological studies suggest that long-term alteration of the brain structure in the stress-responsive system have enduring effects into adulthood. These affect weight regulation, substance abuse, emotional regulation, somatic signal processing, immune function, inflammatory markers leading to CVD, hypertension, diabetes, dental issues, asthma, liver

cancer, depression and other diseases (61–66). Experiencing child abuse, neglect, family dysfunction and other childhood adversities has been shown to increase the risk of acquiring leading causes of adult morbidity and mortality (67–69).

Felitti et.al. (60) in a large adverse childhood events (ACEs) study involving over 9000 adults who completed a health evaluation found that having experienced multiple ACEs also predicted multiple leading causes of death in adults; these included physical inactivity, severe obesity, ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease. Sonu, Post, and Feinglass in a 2019 study on the early onset of chronic disease in young adulthood found that even though the absolute prevalence of chronic disease is low among young adults (18-34), individuals who had a high burden of ACEs before the age of 18 had a much higher risk of chronic diseases than those with no exposure (70).

A South African study on how childhood trauma and poverty were associated with depression in young adult men (18-30 years) used a sample of 2427 males from two peri-urban areas in Johannesburg and Durban. Childhood trauma was measured on physical, emotional, and sexual abuse before 18, also witnessing intimate partner violence (IPV) in the home. They found that experiencing any form of violence strongly affected the mental health of the individual and predicted probable depression (71). A Kenyan study also agreed with the findings in the other studies in understanding childhood adversities and substance use, finding that mental abuse, physical abuse, neglect and illness in the household during childhood increase odds of having substance abuse in early adulthood (72).

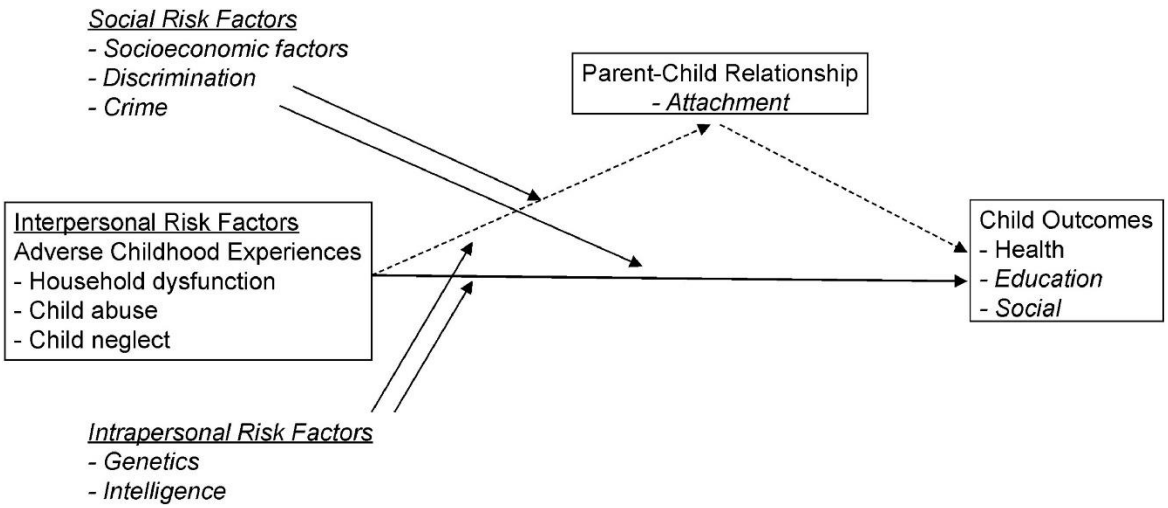


Figure 4:Theoretical framework for child outcomes (source: A Systematic Review of Trials to Improve Child Outcomes Associated With Adverse Childhood Experiences, Marie-Mitchell & Kostolansky (2019) (67)

As much as childhood stressors can be linked to marked traceable effects in adulthood health some studies have documented early detection of multiple adverse emotional, behavioural and physical health effects in children and adolescents. As shown in *Figure 4* child abuse (be it physical, mental, or sexual) mediated by social risk factors (SES, discrimination, and crime (community violence) and intrapersonal risk factors can lead to adverse health, education, and social outcomes (67,73–75). In an investigation of the association between early-life adversity and risk for poor emotional and physical health, Luby et.al. found that ACEs experienced from as early as 3 years of age had a significant association with a reduction in overall health measured in 9 to 15-year olds. This association was still strong after adjusting for age, whole-brain volume, and sex and also the mediation of emotional behaviour and prefrontal sub-region volumes though the sample size was small (119) and health was reported by the parents (76).

Burke and colleagues in a study to determine the impact of ACEs on a paediatric urban population of 701 children aged 0 to 21 (mean age 8.13) used logistic regression to adjust for age, gender and ethnicity. They found that exposure to increased ACEs increased learning and behavioural problem and obesity. This was just measuring association, not causation and learning and behavioural problems were reported by the parents as in many other similar

studies (77). A study on children aged between 18 to 71 months in the US in the welfare system but had not had an out-of-home placement showed that a unit increase in the number of ACEs increases the odds of having a chronic medical condition like diabetes, autism, cystic fibroids, cerebral palsy, blood problems, asthma, attention deficit hyperactivity disorder (ADHD), heart problems, hypertension, and others. This relationship was very clear in older children (36 to 71 months) but still significant in younger children which may suggest that the internal effects of external stress begin almost immediately (78).

Multiple studies on the same subjects at various time points also provide useful insights into the early detection of deleterious effects of experiencing violence and adverse events in childhood. Subjects from LONGSCAN (longitudinal studies of child abuse and neglect) study were examined at cross-sectional points at 14 and 18 years of age. At age 14 they had data for 933 early adolescents and they found that experiencing 2 or 3 adversities especially in recent years (in 2 years) had a significant association with health worries, poor overall health and having health problems (79). At age 18 they had data for 802 adolescents, and at this point, they compared 3 groups experiencing low, early and chronic adversities. The groups with chronic and early adversities had higher health worries and needed or received healthcare more. There was a significant difference in the outcomes between the early and chronic adversities group suggesting that repeatedly suffering or experiencing violence and adversities in childhood causes more harm than occasionally or never suffering them (80). The main weakness of the LONGSCAN was that it was not nationally representative as it was biased towards children with a high likelihood of suffering adversities and health outcomes were either reported by the caregiver before 18 and the participant at 18.

A major issue in the available information on this topic however is that it is mainly from the US and other first world countries yet violence and adversities are more likely to be experienced in LMIC.

1.2.7. Effects of experiencing or exposure to violence on hypertension

Acute stress in response to violent attacks and severe abuse in childhood has been shown to significantly increase the incidence of physician-diagnosed CVD ailments which include hypertension, gradual effects would, however, be more complex to investigate.

Ford and Browning investigated the effects on adult hypertension of exposure to violence with a weapon in 7971 children aged 11 to 17-years in a cohort where they were followed up for hypertension 14 years later. They found that females who experienced violence had higher odds of having hypertension compared to those who did not experience violence whereas males who were exposed to violence had higher odds of hypertension compared to those who were unexposed. This study was hampered by self-reporting of violence exposure and experience by the adolescence but managed to adjust for possible mediation and confounding by CVD risk factors and demographic characteristics (81).

In a review of the relationship between blood pressure mechanisms and exposure to violence where exposure is defined as "experiencing, witnessing, or hearing about violence in the home, school or neighbourhood". Wilson, Kliewer, and Sica conclude that elevated night-time BP in adolescents is associated with exposure to violence and this is a significant marker for the development of hypertension and CVD (82). An earlier study amongst African-American adolescents (11 to 18-year-olds) support this conclusion that violence exposure increased non-dipping (elevated night-time BP) and the relationship was more pronounced among males. The sample size though unfortunately was quite small; 56 participants (83).

Data from the Nurses' Health Study II (NHS II), a prospective cohort of over 60 000 female nurses aged 25 to 44 in the US was used to describe hypertension in adult survivors of child abuse. Child abuse was considered for remembered exposures to physical and sexual abuse between the age of 11 and 17. They found a dose-response relationship between age and race

adjusted risk of incident hypertension, with both sexual and physical violence; the risk increased with increased severity of the abuse in childhood and adolescence. This relationship was not affected by potential confounders except for BMI which left only sever physical abuse and forced sexual activity associated with hypertension risk. The risk also increased further with cumulative abuse exposure (84).

Further another study following up on the same women in the NHS II study for 22 years and used cox proportional hazards models to determine the risk of hypertension after having experienced some traumatic event and showing post-traumatic stress disorder syndrome (PTSD) symptoms. The study also found a dose-response relationship between increasing PTSD symptoms and hypertension, the study was particularly useful in establishing this external stressor-hypertension relationship because they were certain the stress came before hypertension in every case (85) though it was only among women. An earlier cross-sectional study of veterans (mostly male) also confirmed this result to be true among men as it showed that among the 1381 veterans, the ones who showed PTSD symptoms showed a higher likelihood of hypertension. The likelihood was up to 7 times higher among those with PTSD in comparison with those with no mental health issue in the past year (86).

Another study once again on the nurses from the NHS II but this time on IPV investigated how IPV would predict the incidence of hypertension. It included over 50 000 women and concentrated on 3 types of IPV; being physically hurt, forced into sexual activity, or emotionally abused. The study despite other findings showing that physical and sexual abuse are strongly and increasingly associated with hypertension incidence did not manage to establish a significant relationship. However, the study found a very strong relationship with emotional abuse as there is a 24% increase in the hazard of hypertension if one experiences emotional abuse by an intimate partner. The relationship was stronger in severe and non-recent cases (87).

A mental health survey by the WHO using data from 10 countries in Europe, the Americas and Asia and 18630 participants from area clustered representative samples attempted to clear the relationship between early life adversities and hypertension in adulthood. Adversities included sexual abuse, physical abuse, neglect, parental death or divorce, family violence, and so forth and hypertension was self-reported. Cox regressions were used to estimate the hazard ratio of adult-onset hypertension in the face of childhood adversities. Unadjusted hazards increased with increasing exposure to childhood adversities, adjusted hazards do not change that much from the unadjusted thus exhibiting a dose-response relationship (88).

Nomura and Chemtob in a study on the effects of low birth weight (LBW) and abuse in childhood on adaptation and well-being in adolescence and adulthood investigated hypertension among their well-being outcomes. The study followed 1748 children from the John Hopkins collaborative perinatal study. The findings show that after adjusting for demographic confounding that there is no association between hypertension and having experienced some childhood abuse together with LBW (89).

Most of the studies reviewed in this section are focused on adults but give us a fair picture of how hypertension is associated with experiencing violence in childhood. As discussed above CVD outcomes in children and hypertension in early adulthood point towards early alterations of the neurobiological systems in children who experience violence in childhood but more so among those who experience chronic violence. A few studies that also investigated this association could not find a significant association which could be because of myriad reasons like the measurement of hypertension, the time point at which it was measured, sample size, etc. The disagreement between studies necessitates more studies to cement the literature in this field and provide more evidence for early onset of hypertension. Another notable thing is that most of these studies are from developed countries hence making it imperative to contribute to a body of knowledge that is more contextual in LMIC. Some of the studies managed to include

mediators in their analysis but to the researcher's knowledge, none used pathway analysis to adjust for mediation and determine the directionality of the relations.

1.3. Problem Statement

South Africa loses 6560.7 per 100000 disability-adjusted life years (DALYs) due to hypertension (90) which has an estimated prevalence of 21.2% among South African adolescents(91). For long hypertension has been mainly considered a condition of adulthood but it has begun to also emerge in children with the same risk factors observed in adults also turning up in children. Behavioural factors like diet, physical activity, salt intake, and alcohol intake, biological factors like family history and thyroid problems, and psychosocial issues like socio-economic status, stress, violence-related trauma, etc. are influencing the emergence of hypertension in children. With 68.9% of children reporting at least one type of victimisation in their lifetime (92), the relationship between experiencing violence and hypertension is worth understanding.

1.4. Justification

Causes of hypertension in children and adolescents have been receiving steadily increasing attention; in South Africa, some studies have been conducted to understand the effects of BMI, physical activity, growth patterns, and diet. Psychosocial factors have also been receiving some attention within SA, investigating factors like socio-economic status. South Africa is still being plagued by post-apartheid violence due to continued inequalities and the burden of violence is particularly felt by urban children in South Africa (93), yet their coping mechanisms have not yet been clearly understood.

Studies have explored how experiences of violence in childhood related to the development of hypertension later in adulthood but not much has been done investigating the relationship at an earlier stage, that of late adolescence. The progression of hypertension into conditions like

cardiovascular disease, stroke, and organ damage can be controlled better if dealt with earlier in an individual's lifetime. This study, therefore, seeks to understand the associations between experiencing violence and the development of hypertension in adolescents to inform better hypertension screening and support in adolescents who experience violence.

1.5. Research Question

How does experiencing violence influence the development of blood pressure in adolescents?

1.5.1. Aim

To determine the pathways through which experiences of violence affects blood pressure in 18-year-old adolescents in the Soweto-Johannesburg Metropolis

1.5.2. Objectives

1. To describe the sociodemographic characteristics of the 18-year-old adolescents across hypertension status
2. To determine the association between hypertension and the experiences of violence variables without adjusting for other predictors of hypertension amongst the 18-year-olds in the birth-to-twenty cohort.
3. To determine the association between hypertension and experiences of violence adjusting for other predictors of hypertension amongst the 18-year-olds in the birth-to-twenty cohort using Structural Equation Modelling (SEM).

CHAPTER TWO

METHODOLOGY

2. This chapter describes the methods used in this study and gives an overview of the primary study. It provides a background of the primary study site, study design, population, sampling, and data collection. For this study, it provides details on data acquisition, management, selected variables, analysis, and ethical considerations.

2.1. Study Design

This study utilised data collected at a follow-up visit (at age 18) from an on-going longitudinal study on child and adolescent health and development, the Birth-to-Twenty cohort (BT20). This is a longitudinal study following singleton children born within a 7-week enrolment period in the Soweto-Johannesburg Metropolis in 1990. The children were recruited at birth and their families followed up, maintaining contact through the care-givers by phone, field visits, birthday cards and newsletters (94).

2.2. Study Site

The primary study is being conducted in the Soweto-Johannesburg metropolis of SA, one of the largest urban areas in SA. The participants were enrolled from public antenatal clinics in the area during pregnancy.

2.3. Study Population

The secondary study population consisted of all adolescents aged 18 years in 2008 who had anthropometric and BP assessments and were not pregnant at the time of the 18th year follow-up visit. Participants were both male and female and representative of the South African population.

2.4. Sampling

The BT20 study included all children born to women attending local antenatal care (ANC) clinics resident in the study site during the enrolment period and six months thereafter. There were 3273 total participants and the investigators were still in contact with over 70% of the cohort at the time of the follow-up visit used in this study. No sampling was done in the current study, but the exclusion criteria reduced the number of participants obtained from the primary study see Figure 5.

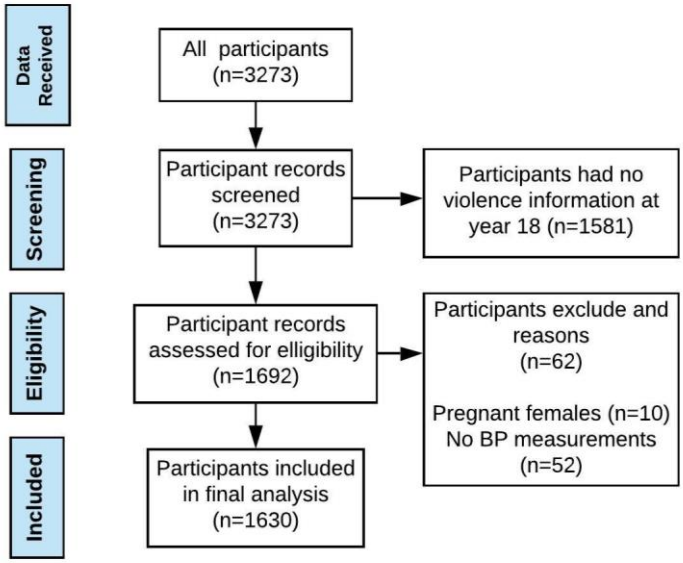


Figure 5: Participant screening for eligibility

2.5. Data Collection

Tools

There were two questionnaires used from the BT20 study used to collect the data for this study. These are the 18th year young adult routine (core) questionnaire and the 18th year adolescent self-complete questionnaire. The self-complete questionnaire was self-administered, and the core questionnaire was administered by trained interviewers who were fluent in the

participant's home language in a face-to-face interview. The self-complete questionnaire collected information that was considered sensitive for adolescents which included substance use and abuse, smoking, addiction, peer influence, sexual risk behaviour, pregnancy status, the experience of and exposure to sexual violence (see Appendix 2 for an extract of the questionnaire). The core questionnaire in addition to demographic information collected data on mood disorders, hope, exposure to and experiences of violence at home, school, community and workplace, physical activity, conflict handling, and others (see Appendix 3 for an extract of the questionnaire). Selected variables from the two 18th year questionnaires were used for this study (95).

2.5.1. Measurement of variables

Outcome Variables

1. *Hypertension*: The main outcome for this study was hypertension. BP was measured during a follow-up visit at age 18 three times on each participant in a sitting position after 5 minutes of rest and 2 minutes between each subsequent measurement using an Omron 6 automated machine (Kyoto, Japan). The first reading was discarded and the second and third averaged and used in the classification. BP status was classified using age, sex, height standardised percentile tables that assign a class for each gender using a CDC-standardized height percentile that classifies a person as either normotensive (<90th percentile), pre-hypertensive (≥90th to < 95th percentile or >120/80 mmHg if <90th percentile) or hypertensive (≥95th percentile) using the fourth report from the National High Blood Pressure Education Program working group on high blood pressure in children and adolescents (NHBPEP) as a guideline(95,96).

2. *Systolic blood pressure (SBP) and Diastolic blood pressure (DBP)*: SBP and DBP were also used as secondary outcomes in this study and were measured as described above. The raw averages for the second and third readings were used as outcomes.

Explanatory variables

These included:

1. *Experiences of Violence*: This was the main exposure variable and it was a latent score constructed using confirmatory factor analysis (CFA). The manifest variables were aggregate scores constructed using yes/no responses from the experiences of violence questions in the core and self-complete questionnaire. They were grouped into one of four categories; sexual, domestic, school, and community/work violence from questions that evaluated whether a participant had experienced physical, sexual, psychological, verbal and emotional violence at home, school or in the community (see lists in Appendix 4).
2. *Socio-demographic factors (adolescent)*: Gender, recorded as male or female was used as is and ethnicity, recorded as Black, Indian, Coloured, and White was recoded to black or other due to the small numbers in some ethnicities.
3. *Anthropometric measurement*: These were collected by trained research assistants. A digital scale to the nearest 0.1kg was used to measure the weight of the participants without shoes and in light clothes. Standing height was measured to the nearest 0.1cm using a wall-mounted stadiometer (Holtain, UK). BMI was calculated using weight and height using the formula $(weight\ (kg)/height\ (m^2))$ (97).
4. *Behavioural factors*: Alcohol use was coded yes or no depending upon the responses of the adolescent to the self-complete questions on alcohol consumption in the previous 30 days
5. *Maternal factors*: Maternal factors like income to gauge socio-economic status (SES) (measured in thousands of Rands), parity, education, age and marital status were also included in the models. Education was recoded to no education/primary from no formal

education to standard 5, high school for standard 6-10, and higher education for post-school training. Marital status which was coded as married (any), living together, separated/divorced/widowed, and single was recategorized to married/living together and single/divorced/widowed.

2.6. Data Analysis

The secondary analysis was conducted on all male and female respondent who met the inclusion criteria from the primary study. All data management and analysis were done in Stata version 15 (98).

2.6.1. Data Management

The data has been used in other studies (95,97) and hence cleaned intensively. For the secondary study, the data was examined for errors and missing values. Most variables had a small (less than 5%) proportion of missing data.

2.6.2. Descriptive Analysis

The socio-demographic characteristics of the adolescents were described by hypertension status. Summaries for categorical variables were done using frequencies and proportions whilst continuous variables like age and income were summarised using means and standard deviation and the other continuous variables using medians and the inter-quartile range (IQR) using Stata version 15.1.

2.6.3. Inferential Analysis

To test associations between hypertension, SBP, DBP and each explanatory variable, bivariate analyses were done using Chi-square tests for categorical-categorical variables, t-tests, analysis of variance (ANOVA), and Mann Whitney tests for categorical-continuous combinations, and

Pearson's correlation coefficient for continuous-continuous combinations. Multivariable analyses were conducted using structural equation modelling (SEM) and generalised structural equation modelling (GSEM) on SBP, DBP, and hypertension. A summary of the data analysis activities can be found in the Appendices.

2.6.3.1. Structural Equation Modelling

The conceptual framework (see Chapter 1) was used to show the hypothesised associations among the variables in the model. SEM and GSEM were then used to investigate associations between hypertension, experiences of violence, socio-demographic and behavioural characteristics, maternal factors, and anthropometric measurements. SEM models allow not only for adjusting for direct effects of exploratory variables but also indirect effects and simultaneous evaluation of multiple exposures (experiences in this case) in one model. The following steps adapted from Mueller and Hancock (99) and Beran and Violato (100) were used in the application of SEM to the research problem:

- Model Specification
- Model Estimation
- Model Evaluation
- Model Modification

Model Specification

An understanding of the problem using expert knowledge, literature, and plausibility is important in hypothesising relationships among variables and this should be done before any data collection or analysis, in this case, is done. This model conceptualisation is done in one of 3 forms:

- structural model, which investigates relationships among measured variables only
- confirmatory factor analysis (CFA), otherwise known as the measurement component/model, which investigates relationships between latent (unmeasured) components and their measured indicators
- a combination model, otherwise known as the latent variable path analysis model which contains aspects of the structural and measurement models (the focus of this research).

The conceptualisation for the model in this research was done using literature, data availability and plausibility and is shown in the conceptual framework in Chapter 1.

Model Estimation

The model's parameters can be estimated using various statistical techniques including maximum likelihood (ML), quasimaximum likelihood (QML), maximum likelihood with missing values (MLMV), and the asymptotic distribution-free (ADF). ADF is based on a kurtosis correction to the regular likelihood ratio test statistic yielding fit tests that are asymptotically insensitive to the observation's distribution. This produces more reliable standard errors and fit statistics in the presence of observed variables that do not fit the multivariate normality assumption (98,101). This study utilised the ADF technique, this is advisable when the observed variables not only deviate from normality but if the model additionally contains categorical variables.

The ADF minimises the following fit function (102)

$$F_{ADF} = [\boldsymbol{s} - \boldsymbol{\sigma}(\theta)]' \boldsymbol{W^{-1}} [\boldsymbol{s} - \boldsymbol{\sigma}(\theta)]$$

Where,

s – vector of non-redundant elements in the empirical covariance matrix,

$\boldsymbol{\sigma}(\theta)$ – the vector of nonredundant elements in the model-implied covariance matrix,

θ – the $t \times 1$ vector of parameters,

$\boldsymbol{W^{-1}}$ – the $k \times k$ positive definite weight matrix with $k = p(p+1)/2$, and

p – the number of manifest variables.

Model Evaluation

SEM estimation methods seek to minimise the discrepancy between the model fit and the data i.e. the discrepancy between the model's covariance matrix and that of the true population. Multiple fit indices can be used to determine the fit of the model to data. Traditionally the chi-square which is however thought to be too strict in the evaluation of a deviation has been used in the evaluation. The chi-square is evaluated using the following formula

$$\chi^2(df) = (N-1)F[\boldsymbol{S}, \boldsymbol{\Sigma}(\hat{\theta})]$$

Where,

df – are the degrees of freedom $s - t$,

s – the number of nonredundant elements in $\boldsymbol{S}$,

t – the number of parameters to be estimated,

N – the sample size,

$\boldsymbol{S}$ – the empirical covariance matrix, and

$\boldsymbol{\Sigma}(\hat{\theta})$ – the model-implies covariance matrix.

More desirable indices include absolute indices like the standardised root mean square residual (SRMR) and the goodness-of-fit index (GFI), parsimonious indices like the root mean square error of approximation (RMSEA) and its associated confidence interval (CI), and the Akaike

information criterion (AIC), and incremental indices like the comparative fit index (CFI) and the normed fit index (NFI). The RMSEA is a "closeness of fit" test that assesses prediction induced discrepancy by estimating the square root of the discrepancy per degree of freedom; this estimate is zero bound. The SRMR assesses the badness of the fit by standardising residuals from the empirical and model-implied covariance matrices, squaring the fitted standardised residuals, summing, and calculating the square root of the sum. The CFI is a model-comparison index that compares subsequent models against a "baseline" model and increasing values indicate model improvement. The CFI is computed as

$$CFI = 1 - \frac{\max\left[(\chi_t^2 - df_t), 0\right]}{\max\left[(\chi_t^2 - df_t), (\chi_i^2 - df_i), 0\right]}$$

Where,

χ_t^2 – the chi-square of the target model,

χ_i^2 - the chi-square of the baseline model, and

df – the number of degrees of freedom.

An inspection of standard errors is also helpful in determining whether a model is not underidentified. The acceptability of the fit is determined by a joint criterion of fit indices $\text{CFI} \geq 0.96, \text{SRMR} \leq 0.09, \text{and RMSEA} \leq 0.06$.

Model Modification

If the hypothesised model does not meet the fit indices criterion then it can be modified to make the fit better. This can be remedied in one of two ways i.e. updating the theory by re-specifying the model or dealing with the model internally by removing irrelevant paths and adding relevant ones. The internal modification can be done using modification indices which are exploratory in nature and still require knowledge of the subject matter. Modification indices

were used in this study to add correlations and some mediation paths to improve initial fit. This process can be used iteratively with the previous step until a reasonably satisfactory model is achieved.

2.6.3.2. Generalised Structural Equation Modelling (GSEM)

This an extension of SEM that allows continuous, binary, count, categorical, ordered and survival time measurements, and models can be logit, probit, ordinal logit, multinomial logit, and many others (103). GSEM is however limited in its provision of modification indices, summary statistics, and standardisation of coefficients mainly because it does not have as many distribution-based assumptions as SEM. Its ability to handle binary outcomes was particularly useful in this study in the modelling of hypertension status as an outcome.

2.7. Ethical Considerations

The primary study obtained ethics clearance from the Human Ethics Research Committee (HREC) of the University of the Witwatersrand before commencement of the study at each data collection time point. Participants below the age of 18 had consent provided by their parents/caregivers and those over the age of 18 provided informed consent. Ethics approval for the secondary study was also sought from the HREC of the University of the Witwatersrand (M181007) before data was accessed. The data from this study can be requested from the Developmental Pathways for Health Research Unit data management department.

CHAPTER THREE

RESULTS

3.1. Introduction

This chapter presents results including a description of the population, bivariate analyses between the outcome(s) and covariates, and results from the SEM and GSEM analyses.

3.2. Descriptive statistics

3.2.1. Characteristics of the study population

Descriptive statistics are shown in Table 1 below.

After excluding pregnant female adolescents, there remained 1630 participants; 249 (15%) who were hypertensive and 1381 (85%) who were normotensive. Most of the participants were black, born to single mothers, and mothers with at least a high school education and report to have experienced at least one type of violence in the past year. Hypertensive adolescents are born to older mothers, with less income per month and with more children when compared with the averages in mothers of normotensive adolescents. Hypertension appears to be lower in adolescents born to mothers who are either married or cohabiting and have a higher education level.

The least commonly reported experience of violence is sexual, then followed by community, home and school violence which is the most common. BMI, WHR, income, and maternal parity differ significantly between hypertensive and normotensive adolescents.

Table 1: Study sample characteristics by hypertension status

Characteristic	Level	Normotensive (n=1381) n (%)	Hypertensive (n=249) n (%)	All (n=1630) n (%)	p-value
Individual Factors					
Body Mass Index (BMI)*	-	21.2 (3.7)	23.1 (4.8)	21.5 (4.0)	**<0.001**
Waist-to-hip Ratio*	-	0.3(0.1)	0.4(0.1)	0.3(0.1)	**<0.001**
Ethnicity	Non-Black	186(88.2)	25(11.8)	211(100.0)	0.138
	Black	1,195(84.2)	224(15.8)	1,419(100.0)	

Sex	Male	663 (84.8)	119 (15.2)	782 (100.0)	0.950
	Female	515 (84.7)	102 (15.3)	617 (100.0)	
Alcohol Abuse	Yes	332 (86.9)	50 (13.1)	382 (100.0)	0.190
	No	1041 (84.2)	196 (15.8)	1237 (100.0)	
Systolic Blood Pressure (SBP)*	-	115.5(12.5)	132.5(9.5)	117.0(15.0)	
Diastolic Blood Pressure (DBP)*	-	69.5(9.5)	83.0(13.5)	71.0(11.0)	
Experiences of Violence					
Home	Yes	298 (25.8)	60 (27.3)	358 (26.0)	0.771
	No	859 (74.2)	160 (72.7)	1,019 (74.0)	
School	Yes	393 (34.9)	80 (37.2)	473 (35.3)	0.457
	No	732 (65.1)	135 (62.8)	867 (64.7)	
Community	Yes	302 (26.1)	54 (24.5)	356 (25.9)	0.805
	No	855 (73.9)	166 (75.5)	1,021 (74.1)	
Sexual	Yes	40 (3.6)	6 (2.9)	46 (3.5)	0.179
	No	1,077 (96.4)	201 (97.1)	1,278 (96.5)	
Overall	Yes	592 (51.17)	115 (52.27)	707 (51.34)	0.776
	No	565 (48.83)	105 (47.72)	670 (48.66)	
Maternal Factors					
Income (thousands of Rands) *	-	11.0(10.1)	8.6(6.7)	10.6(9.7)	**0.044**
Parity*	-	2.2(1.4)	2.5(1.5)	2.2(1.4)	**0.006**
Marital Status	Married	488.0(35.6)	102.0(41.1)	590.0(36.5)	0.097
	Single	882.0(64.4)	146.0(58.9)	1,028.0(63.5)	
Education	No Education/ Primary	151.0(11.9)	37.0(16.2)	188.0(12.5)	0.084
	High School	1,004.0(79.0)	177.0(77.6)	1,181.0(78.8)	
	Higher Education	116.0(9.1)	14.0(6.1)	130.0(8.7)	
Age*	-	25.8(6.2)	26.1(6.3)	25.8(6.2)	0.365

*** indicates variables described using mean and standard deviation in the form: mean (sd)/median (IQR)**

3.2.2. Bivariate analyses between hypertension, SBP, DBP, and other covariates

Results for bivariate analysis are shown in Table 2 below.

BMI, parity, and WHR are significantly higher amongst hypertensive adolescents as compared to those who are not, maternal income, however, is significantly lower. Males have a significantly higher SBP whilst females have a significantly higher DBP though the actual difference is slight. SBP is significantly lower among non-black and drinking adolescents whilst BMI, parity, and WHR are positively correlated with SBP and DBP. Participants who experienced violence at school had a higher SBP than those who did not, as did those born to

mothers who were either married or cohabiting. SBP and DBP significantly decreased with increasing mother's education and DBP was positively though slightly correlated with mother's age.

Table 2: Bivariate Analyses between Hypertension, SBP, DBP, and the covariates

Characteristic	Hypertension Unadjusted OR (SE)	SBP coefficient (SE)	DBP coefficient (SE)
Individual Factors			
Ethnicity	1.39 (0.31)	**2.04 (0.8)**	1.07 (0.64)
Sex	1.01 (0.14)	**-6.33 (0.52)**	**0.91 (0.43)**
Alcohol Use	0.80 (0.14)	**-1.48 (0.64)**	-0.58 (0.51)
Body Mass Index (BMI)	**1.10 (0.02)**	**0.43 (0.06)**	**0.31 (0.05)**
Experiences of Violence			
School	1.12 (0.16)	**1.15 (0.58)**	-0.43 (0.47)
Home	1.05 (0.16)	-0.3 (0.63)	-0.54 (0.5)
Community	0.96 (0.16)	0.9 (0.64)	-0.19 (0.51)
Sexual	0.66 (0.21)	-1.49 (1.07)	-0.93 (0.85)
Overall	1.04 (0.14)	0.66 (0.54)	-0.63 (0.44)
Maternal Factors			
Marital Status	0.79 (0.11)	**-1.44 (0.56)**	-0.84 (0.45)
Education			
High School	0.72 (0.14)	**-2.15 (0.85)**	**-1.46 (0.69)**
Higher Education	0.49 (0.17)	**-2.85 (1.24)**	**-2.39 (1.00)**
Age*	1.01 (0.01)	0.12 (0.04)	**0.04 (0.03)**
Income (thousands of Rands)	0.97 (0.01)	-0.08 (0.04)	-0.01 (0.03)
Parity	1.14 (0.05)	**0.9 (0.19)**	0.28 (0.16)

Estimates in bold indicate p<0.05. Reference levels for ethnicity, sex, education, alcohol abuse, and marital status are non-black, male, primary/no education, yes and married respectively

3.3. Associations between experiencing violence and hypertension, SBP, and DBP using GSEM and SEM

Associations between experiencing violence and hypertension, SBP, and DBP were investigated using SEM models based on the framework proposed in Chapter 1. The results of the models are shown in the following tables. The measurement portion of the model was first investigated for fit (see Appendices). The structural models and the results displayed in 6, 7, and Figure 8

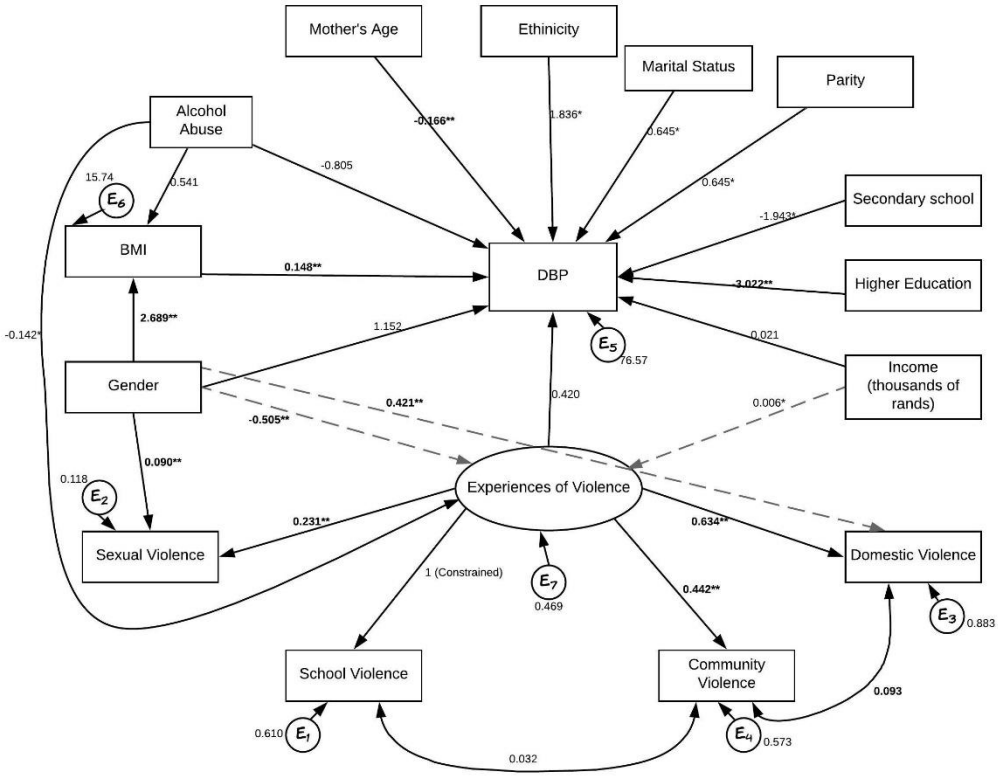


Figure 7: The association of experiences of violence with DBP adjusting for other factors using the SEM approach

3.3.1. The association of experiences of violence on SBP using SEM approach

Figure 66 shows the SEM model investigating the association between SBP and experiences of violence adjusting for other covariates.

Being female significantly decreases SBP by 5.17 mmHg when compared with males; directly it reduces SBP by an even larger margin of 7.24 mmHg and indirectly when mediated by BMI increases SBP by 2.07 (See Appendix 5 for indirect effects). This means females, when compared with males of an equal BMI, have a higher SBP. A unit increase in both BMI and Parity significantly increase SBP by 0.73 mmHg and 0.86 mmHg respectively. Females also have a lower SBP by 0.51 mmHg when compared with males of a comparable experience of violence score and levels of other factors. Black adolescents had a marginally higher SBP than

non-black adolescents and children born to single, divorced, or widowed mothers had a marginally lower SBP when compared to children born to married or cohabiting mothers whilst adolescents who did not abuse alcohol had a marginally lower SBP when compared to those who do. Adolescents from higher-income homes having the same experience of violence score also had a marginally slighter SBP as an increase in a thousand rands income sees a corresponding 0.01 mmHg decrease in SBP through experiences of violence (See Appendix 5 for indirect effects). A unit increase in the experiences of violence score decreases SBP by 0.222 mmHg but not significantly. The other covariates i.e. mother's education, age, and income had no significant effect on SBP.

See Appendix 5 for detailed indirect and direct effects, confidence intervals and model diagnostics.

Estimates in bold indicate (**) p<0.05. Reference levels for ethnicity, sex, education, alcohol abuse, and marital status are non-black, male, primary/no education, yes and married respectively

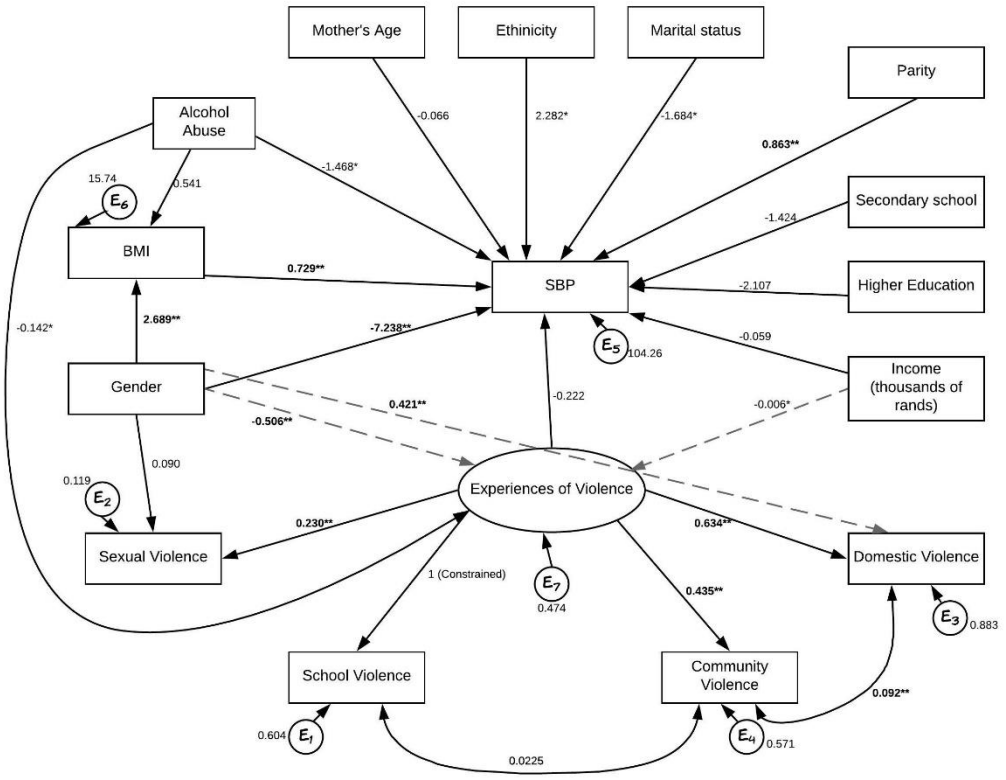


Figure 6: The association of experiences of violence with SBP adjusting for other factors

3.3.2. The association of experiences of violence on DBP using the SEM approach

Figure 77 shows the SEM model investigating the association between DBP and experiences of violence adjusting for other covariates.

Females have a significantly higher DBP than males by 1.67 mmHg when compared with males. A unit increase in BMI significantly increases DBP by 0.32 mmHg while children born to older mothers' DBP significantly decreased by 0.17mmHg for each 1-year increase in age of the mother. Being born to single, divorced or widowed mothers had a significantly lowering effect on DBP by 1.94 when compared with being born to married or cohabiting mothers. Increasing mother's education had a lowering effect on DBP in adolescents the highest lowering effect was in children born to mothers who had some tertiary education; 3.02 mmHg. Black adolescents had a marginally higher DBP than non-black children and children from larger families had a marginally higher DBP. The other covariates i.e. experience of violence, alcohol abuse and income had no significant effect on DBP.

Estimates in bold indicate (**) p<0.05. Reference levels for ethnicity, sex, education, alcohol abuse, and marital status are non-black, male, primary/no education, yes and married respectively

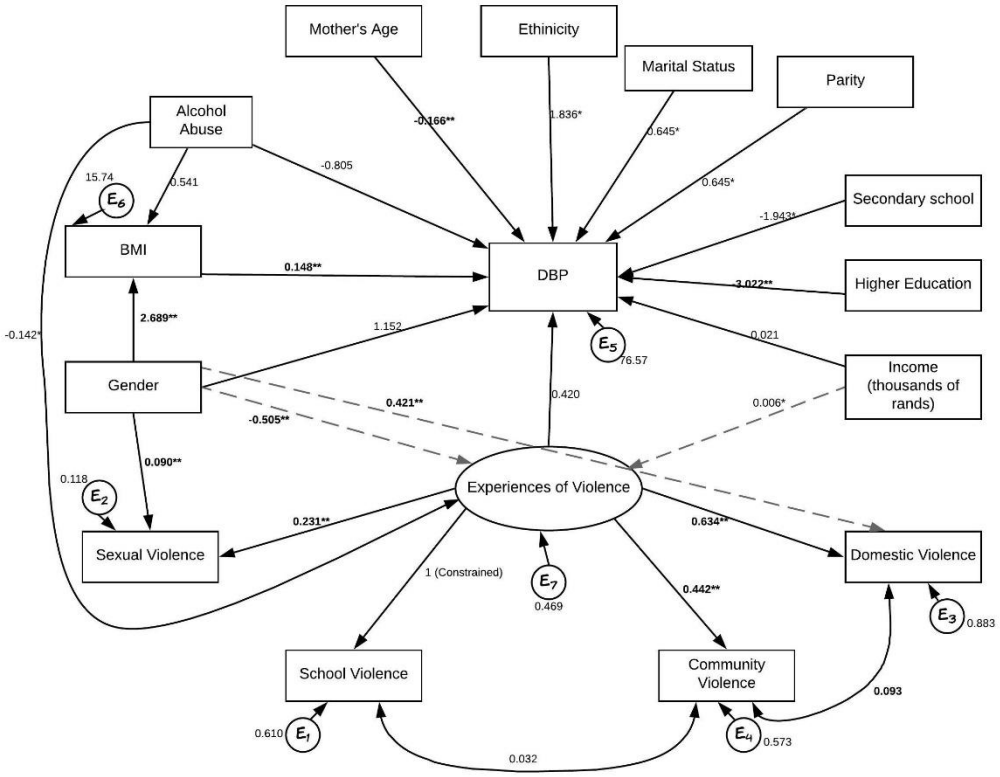


Figure 7: The association of experiences of violence with DBP adjusting for other factors using the SEM approach

3.3.3. The association between experiencing violence and hypertension using the GSEM approach

Figure 8Figure 7 shows the GSEM model investigating the association between hypertension and experiences of violence adjusting for other covariates.

A unit increase in BMI corresponds to a significant increase in odds of having hypertension by 2%, whilst females, when compared with males of an equal BMI, have 15 times increased odds of having hypertension. Females, after adjusting for other factors, when compared with males experiencing the same level of violence are 39% less likely to have hypertension. Adolescents born to married or cohabiting mothers are 7% less likely to have hypertension. Adolescents who do not use alcohol when compared to those who do and have experienced a similar level of violence and other factors are 13% less likely to have hypertension (see Table 3 for indirect effects). Those from higher-income families also when compared to others who have

comparable factor levels and experience of violence score a decrease of 1% in likelihood of having hypertension with every increase of a thousand rand in household income (see Table 3 for indirect effects). Other explanatory variables like experience of violence, ethnicity, sex, alcohol use, and maternal factors have no significant direct effects on hypertension.

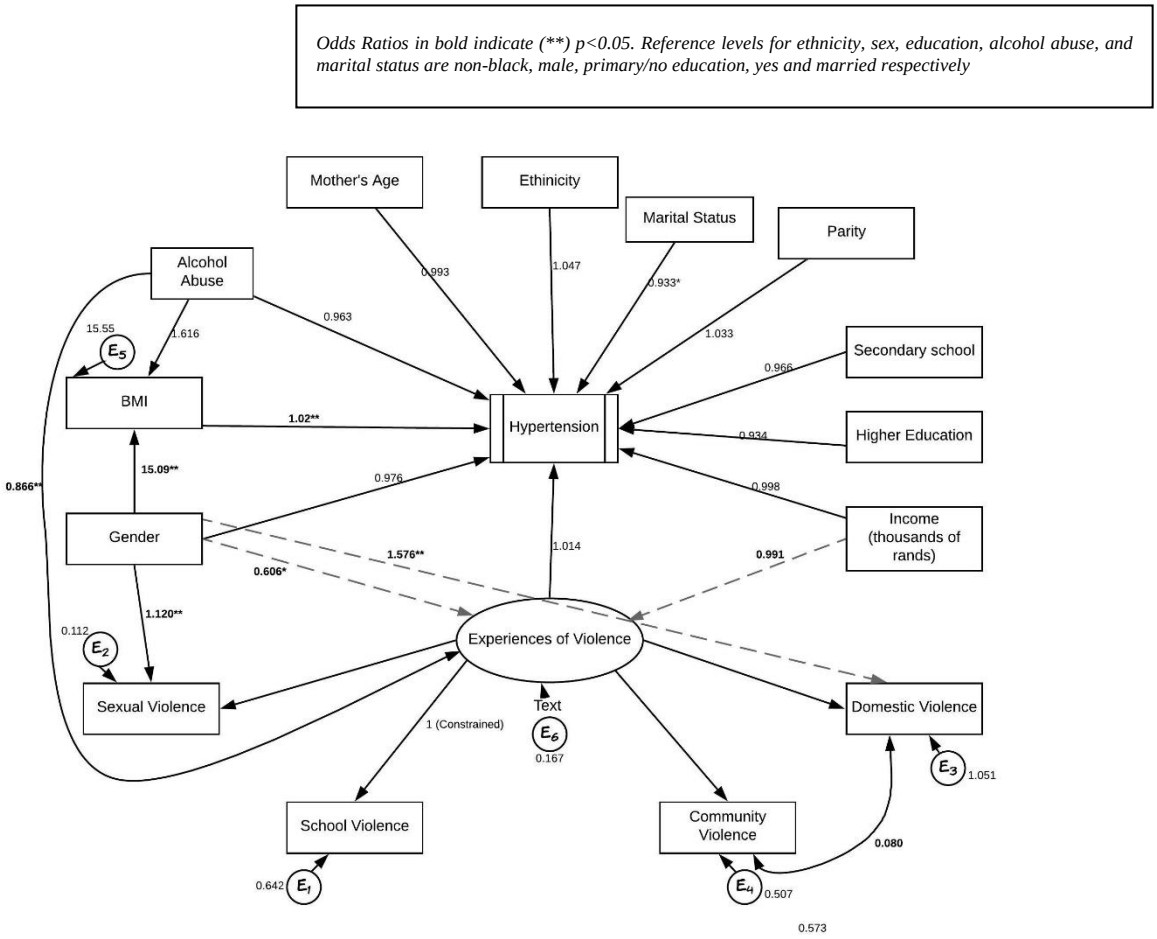


Figure 8:The association of experiences of violence with hypertension adjusting for other factors using the GSEM approach

Table 3 shows detailed indirect and direct effects, confidence intervals and model diagnostics.

Table 3: The effects of experiences of violence on hypertension adjusting for other factors using the GSEM approach

Endogenous Variable	Exogenous Variable	Direct Effects Adj OR (95% CI)	Indirect Effects Adj OR (95% CI)	Total Effects Adj OR (95% CI)	p-value
Hypertension (binary)	Experiences of Violence	1.01 (0.97:1.06)		1.01 (0.97:1.06)	0.548
	Ethnicity	1.05 (0.96:1.15)		1.05 (0.96:1.15)	0.323
	Sex	0.98 (0.91:1.05)	**1.05 (1.01:1.09)**	0.97 (0.93:1.02)	0.513
	Alcohol Use	0.96 (0.90:1.03)	1.01 (0.99:1.02)	0.97 (0.91:1.03)	0.236
	Body Mass Index (BMI)	**1.02 (1.01:1.03)**		**1.02 (1.01:1.03)**	<0.001
	Mother's Marital Status	0.93 (0.86:1.01)		0.93 (0.86:1.01)	0.096

	Education	0.97 (0.84:1.11)		0.97 (0.84:1.11)	0.616
		0.93 (0.78:1.12)		0.93 (0.78:1.12)	0.453
	Mother's Age	0.99 (0.98:1.01)		0.99 (0.98:1.01)	0.249
	Income (thousands of Rands)	1.00 (0.99:1.00)	1.00 (0.99:1.00)	1 (0.99:1.00)	0.211
	Parity	1.03 (0.98:1.09)		1.03 (0.98:1.09)	0.246
BMI	Sex	**15.09 (8.99:25.31)**		**15.09 (8.99:25.31)**	<0.001
	Alcohol Abuse	1.62 (0.78:3.36)		1.62 (0.78:3.36)	0.200
Experiences of Violence	Income (thousands of Rands)	**0.99 (0.99:0.995)**		**0.99 (0.99:0.995)**	<0.001
	Sex	**0.61 (0.53:0.69)**		**0.61 (0.53:0.69)**	<0.001
	Alcohol Abuse	**0.87 (0.78:0.97)**		**0.87 (0.78:0.97)**	0.01

Estimates in bold indicate p<0.05, * indicates p<0.1, Reference levels for ethnicity, sex, education, alcohol abuse, and marital status are non-black, male, primary/no education, yes and married respectively

1

2

CHAPTER FOUR

DISCUSSION AND CONCLUSIONS

4.1.Chapter Introduction

This chapter highlights the findings from the current study in light of existing literature. It also evaluates the strengths and limitations of the current study and its implications for future research and policy. This study investigated the association between experiencing violence and SBP, DBP and hypertension, in adolescents adjusting for demographic, behavioural and socio-economic factors in adolescents.

4.2.Main Findings

The results from the GSEM and SEM show a trend of increasing odds of hypertension, increased DBP and lowered SBP with increasing experience of violence score though none of the associations were statistically significant.

A bulk of studies on the effect of experiencing childhood focus on the emergence of health issues in early adulthood and older age groups (104,105). Studies that investigate the effect of violence on early life health outcomes suggest that recent exposure or experience of adversity have a stronger effect of negative health outcomes(79,80,106); this study, however, finds no significant associations between experiencing violence and the health outcomes though there is an indication of increasing DBP and odds of hypertension with increasing experiences. A study on 13 to 17-year-olds in Boston only found a significant difference in DBP between adolescents who experienced child abuse and those who did not (107). The Boston study unlike the current study has a broader definition of experiences of violence looking at a “have you ever” situation and classifying by the number of yeses. Su et al investigated the long term-effect of experiencing adversities in childhood on trajectories of BP from childhood to young

adulthood and did not find a significant main effect only observing a faster increase in BP after 30 among participants who had experienced adversities in childhood (104). Some studies have rationalised that the effects of violence on health outcomes are easier to observe in adulthood as risk factors like hypertension are more common in adulthood than in childhood/adolescence (106). This study assessed association in adolescents hence the effects of experiencing violence in adolescence may not be easily observable.

4.3.Other Findings

In this study, we found that BMI had significant direct effects of increasing the odds of having hypertension, levels of DBP, and SBP which is consistent with a number of studies that evaluated the outcomes together or separately. Dekkers et al found that increasing BMI was associated with increasing SBP in both males and females in a growth trajectories study (35,108–111). An Indonesian study also found that the odds of hypertension were increased among adolescents with a high BMI, another study found that decreasing BMI significantly reduced the odds of hypertension in adolescents (109,112). BMI further mediates the relationship between gender and hypertension and SBP, females when compared with males of the same BMI have a higher SBP and their odds of having hypertension are higher. This result is contrary to other studies that investigated the effect of gender for same BMI levels on health outcomes and did not find an interaction or found the odds higher in boys when compared with girls (108,112,113).

This study did not find a significant direct effect of gender on hypertension but rather on increasing DBP and decreasing SBP among females. Muhihi et al did not find an association between hypertension and gender, as did Maximova et al in investigating the association to SBP and other studies (35,110,114). However other studies among adolescents and young adults found a significant association between being male and increased odds of hypertension (115). Another study found that in younger children hypertension is higher in girls than boys,

but this was for a much younger population (6-year-olds) than the one under consideration in this study (116). This relationship is clearly documented in adults but in childhood and adolescents is more variable with the population under consideration. This could be attributed to the ethnicity prevalent in the group for example for this study we have 87% black adolescents and a number of studies and reviews among African or afro-descendant children and adolescents show that gender is not a significant association (34,92,114).

Being female had protective effects despite experiencing comparable levels of violence with males on increasing SBP, DBP, and odds of hypertension. This is inconsistent with most studies which found instead that women generally had worse physical health outcomes than men who experienced childhood violence (both in adult and paediatric populations) (117,118). Other studies however did not find any significant differences between health outcomes for males and females by increasing violence exposure(119,120).

The current study found a significant direct association between decreased DBP and increasing mother's age and education, and among children to single mothers. which concurs with other studies. Marie-Pierre et al reported that DBP trajectories of adolescents decreased among male children from single-parent homes and those with mothers who have a tertiary qualification (121). On the contrary Dekkers et al and Gupta-Malhotra et al found that children from single-parent homes had increased DBP and those from 2-parent homes had decreased odds of hypertension respectively. Gupta-Malhotra et al further reported that family status effects were more clearly observed in adulthood than in childhood-onset hypertension (108,122).

Other explanatory variables like ethnicity, income, and alcohol abuse had no significant direct or indirect effect on the outcomes. This is contrary to other studies which have found significant adjusted effects of these factors (46,108,121)

4.4.Strengths and Limitations

4.4.1. Study design

This study utilised data reported and measured from a birth cohort study at age 18 and incorporating maternal characteristics collected at birth. All measurements data were collected by trained data collectors which would point towards good high-quality data. However, the information on sensitive issues which include the main explanatory variable was not only self-reported but self-administered which may result in underreporting due to recall or social desirability biases. Self-reporting however is preferable to caregiver reporting and the "in the past year" period also minimises recall errors though not entirely. One main limitation of secondary data analyses is that inasmuch as the data was collected well and in an organised manner it was not collected to answer the current research question. Other data that would have been useful in modelling like sedentary behaviour, family history of hypertension, and dietary information could not be accessed as this is a secondary analysis and could only make use of available data. The cohort study is advantageous in that one can establish temporality, some of that advantage was lost to some extent in this study, though inference is used, it concentrated on one time point. This study is also entirely focused on urban adolescents and hence cannot be generalised to peri-urban and rural adolescents. In addition, this sample may differ with urban adolescents who are not part of the cohort due to the knowledge that they are being followed up and their mothers opted for hospital delivery. Generalising the findings to any group of adolescents then should be done with caution (123,124).

4.4.2. Analysis and study sample

SEM is flexible and allows complex models using various types of data and a comparison of multiple models that represent a variety of hypotheses (125). Its flexibility allows for variables to be both dependent and independent in the same model allowing for some causal inference within an inherently observational study. The path analysis technique is also good for investigating mediation as the pathway technique allows for the easier specification of

temporality and causality even of the same variable. SEM, through modification indices, helps improve the specified model to come up with the "best" model (99). SEM also allows for easy estimation of latent variables whilst evaluating a more complex model simultaneously. GSEM extends SEM as explained in the Methods section but does not have goodness of fit indices within Stata to determine if any improvements to the model are necessary. The main exposure was limited to exposure in the "past 12 months", this may have been too restrictive in terms of defining experiences of violence in childhood. The sample also was sufficiently representative with a ratio of almost 1:1 between males and females and slightly over 80% black adolescents which is population representative despite making inference a little unclear.

4.5.Conclusion

Even though there was no significant association between experiencing violence and hypertension among 18-year-old urban adolescents, findings from this study were indicative of an associated increase in hypertension and DBP with increasing experiences of violence. This indication requires more exploration. Males were at a higher risk than females of comparable violence experiences of having hypertension and higher SBP and DBP which possibly shows poor male coping strategies. Alcohol use and higher BMI also put adolescents at higher risk of having hypertension and general elevated blood pressure, which may be an indicator of the need for lifestyle change interventions.

Maternal factors like higher education and a higher income were protective against the risk of hypertension and increased SBP and DBP. This finding points towards long-term economic and educational increase goals that would see an overall improvement of the quality of life.

Interventions encouraging violence awareness and reporting, male-engagement, drinking awareness, body positivity, weight control, and other risk modification should be engaged early in children's lives to encourage positive health outcomes.

4.6.Recommendations for policy and further research

This study indicated that an increase in experiences of violence could lead to an increase in hypertension and DBP. This is of importance in volatile environments like South Africa where violence is still a major cause of death and disability and NCDs are also on the rise. Family, community and national interventions on violence awareness, reporting, and care-seeking could go a long way in interrupting the effects of psychosocial stressors on later health. This though requires more research.

For LMIC undergoing an epidemiological, urbanisation, and nutritional shift like South Africa; malnutrition is an important factor that drives NCDs. Changes in the nutritional pattern, overnutrition, body image, cultural views of weight, low-income, and increased urbanisation rates all work against the maintenance of healthy bodies (126,127). Findings from this study show that an increase in BMI corresponds with increased hypertension, SBP, and DBP. Interventions to help the population deal with weight gain and nutrition are necessary at the individual, community and national levels.

The repeated occurrence of gender as a mediator between experiences of violence and hypertension, SBP, and DBP with males being at a greater risk despite having experienced comparable levels of violence with female indicates the need for male-focused intervention. This may point towards more internalisation, poor coping strategies, a disconnect with the help-seeking structures, and a burden of masculinity (128,129) among males. This implies the need for gender-specific intervention and research towards better engaging males and ensuring better health outcomes for them. Alcohol use also placed adolescents with comparable experiences of violence at greater risk of having hypertension. Alcohol abuse, responsible use, and engagement interventions could be useful in engaging adolescents and helping them to make better, more responsible decisions for their health

More research towards the role of life events and in this case, adverse, in the development of disease in adolescence would help establish clearer patterns. Looking into more exposure and experiences time points, a more representative sample, and/or including other risk factors could be instrumental in the provision of evidence to life course theory.

REFERENCES

1. Giles TD, Materson BJ, Cohn JN, Kostis JB. Definition and Classification of Hypertension: An Update. J Clin Hypertens. 2009;11(11):611–4.

2. Global status report on noncommunicable diseases 2014 [Internet]. [cited 2019 Jul 26]. Available from: https://apps.who.int/iris/handle/10665/148114

3. Stanaway JD, Afshin A, Gakidou E, Lim SS, Abate D, Abate KH, et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet. 2018 Nov 10;392(10159):1923–94.

4. Carretero OA, Oparil S. Essential hypertension. Part I: definition and etiology. Circulation. 2000 Jan 25;101(3):329–35.

5. The 2017 update, Global Health Workforce Statistics, World Health Organization, Geneva [Internet]. WHO. 2017 [cited 2018 Jul 3]. Available from: http://www.who.int/gho/ncd/risk_factors/blood_pressure_prevalence/en/

6. Roth Gregory A., Nguyen Grant, Forouzanfar Mohammad H., Mokdad Ali H., Naghavi Mohsen, Murray Christopher J.L. Estimates of Global and Regional Premature Cardiovascular Mortality in 2025. Circulation. 2015 Sep 29;132(13):1270–82.

7. Zhou B, Bentham J, Di Cesare M, Bixby H, Danaei G, Cowan MJ, et al. Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19·1 million participants. The Lancet. 2017 Jan 7;389(10064):37–55.

8. World Health Organization. Global Health Observatory (GHO) Data [Internet]. 2015 [cited 2019 Apr 25]. Available from: https://www.who.int/gho/ncd/risk_factors/blood_pressure_prevalence_text/en/

9. World Health Organization. Global status report on noncommunicable diseases 2014 [Internet]. 2014 [cited 2019 May 26]. Report No.: WHO/NMH/NVI/15.1. Available from: https://apps.who.int/iris/handle/10665/148114

10. Hendriks ME, Wit FWNM, Roos MTL, Brewster LM, Akande TM, Beer IH de, et al. Hypertension in Sub-Saharan Africa: Cross-Sectional Surveys in Four Rural and Urban Communities. PLOS ONE. 2012 Mar 12;7(3):e32638.

11. Mills KT, Bundy JD, Kelly TN, Reed JE, Kearney PM, Reynolds K, et al. Global Disparities of Hypertension Prevalence and Control: A Systematic Analysis of Population-based Studies from 90 Countries. Circulation. 2016 Aug 9;134(6):441–50.

12. 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.

13. Poulter NR, Prabhakaran D, Caulfield M. Hypertension. The Lancet. 2015 Aug 22;386(9995):801–12.

14. Mayosi B. A statement of intent on the formation of the NCRP on cardiovascular and metabolic disease: a new initiative to fight heart disease, stroke, diabetes and obesity in South Africa. Cardiovasc J South Afr Off J South Afr Card Soc South Afr Soc Card Pract. 2007 Feb;18(1):4–6.

15. Bradshaw D, Groenewald P, Laubscher R, Nannan N, Nojilana B, Norman R, et al. Initial burden of disease estimates for South Africa, 2000. South Afr Med J Suid-Afr Tydskr Vir Geneeskd. 2003 Sep;93(9):682–8.

16. Dorrington RE, Bradshaw D. GBD 2016 estimates problematic for South Africa. The Lancet. 2018 Sep 1;392(10149):735–6.

17. Norman R, Bradshaw D, Schneider M, Pieterse D, Groenewald P. Revised Burden of Disease Estimates for the Comparative Risk Factor Assessment, South Africa 2000. Methodological Note. Cape Town: South African Medical Research Council, 2006.;

18. Lloyd-Sherlock P, Beard J, Minicuci N, Ebrahim S, Chatterji S. Hypertension among older adults in low- and middle-income countries: prevalence, awareness and control. Int J Epidemiol. 2014 Feb;43(1):116–28.

19. Goon D, Amusa L, Mhlongo D, Khoza L, Anyanwu F. Elevated Blood Pressure among Rural South African Children in Thohoyandou, South Africa. Iran J Public Health. 2013;42(5):489–96.

20. Zhang Y-X, Sun G-Z, Zhao J-S, Lin M, Chu Z-H. Monitoring of blood pressure among children and adolescents in a coastal province in China: results of a 2010 survey. Asia Pac J Public Health. 2015 Mar;27(2):NP1529-1536.

21. Barker DJP. The origins of the developmental origins theory. J Intern Med. 2007 May;261(5):412–7.

22. Barker DJ, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. Lancet Lond Engl. 1986 May 10;1(8489):1077–81.

23. Wadhwa PD, Buss C, Entringer S, Swanson JM. Developmental Origins of Health and Disease: Brief History of the Approach and Current Focus on Epigenetic Mechanisms. Semin Reprod Med. 2009 Sep;27(5):358–68.

24. Croix B, Feig DI. Childhood hypertension is not a silent disease. Pediatr Nephrol. 2006 Apr 1;21(4):527–32.

25. Gomes RS, Quirino IG, Pereira RM, Vitor BM, Leite AF, Oliveira EA, et al. Primary versus secondary hypertension in children followed up at an outpatient tertiary unit. Pediatr Nephrol. 2011 Mar 1;26(3):441–7.

26. Flynn Joseph, Zhang Ying, Solar-Yohay Susan, Shi Victor. Clinical and Demographic Characteristics of Children With Hypertension. Hypertension. 2012 Oct 1;60(4):1047–54.

27. Kapur G, Ahmed M, Pan C, Mitsnefes M, Chiang M, Mattoo TK. Secondary Hypertension in Overweight and Stage 1 Hypertensive Children: A Midwest Pediatric Nephrology Consortium Report. J Clin Hypertens. 2010;12(1):34–9.

28. Weaver DJ. Hypertension in Children and Adolescents. Pediatr Rev. 2017 Aug;38(8):369–82.

29. Ewald DR, Haldeman LA. Risk Factors in Adolescent Hypertension. Glob Pediatr Health. 2016 Jan 1;3:2333794X15625159.

30. Rao G. Diagnosis, Epidemiology, and Management of Hypertension in Children. Pediatrics. 2016;138(2).

31. Morales Suárez-Varela M, Mohino Chocano Mª C, Soler Quiles C, Llopis-Morales A, Peraita-Costa I, Llopis-González A. [Prevalence of arterial hypertension and its association with anthropometry and diet in children (6 to 9 years old): ANIVA study]. Nutr Hosp. 2019 Mar 7;36(1):133–41.

32. Fan Z, Liao Z, Zong X, Zhang S. Differences in prevalence of prehypertension and hypertension in children and adolescents in the eastern, central and western regions of China from 1991-2011 and the associated risk factors. PloS One. 2019;14(1):e0210591.

33. Patel A, Bharani A, Sharma M, Bhagwat A, Ganguli N, Chouhan DS. Prevalence of hypertension and prehypertension in schoolchildren from Central India. Ann Pediatr Cardiol. 2019 Aug;12(2):90–6.

34. 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 Health. 2017;2(8):e375–86.

35. Muhihi AJ, Njelekela MA, Mpembeni RNM, Muhihi BG, Anaeli A, Chillo O, et al. Elevated blood pressure among primary school children in Dar es salaam, Tanzania: prevalence and risk factors. BMC Pediatr [Internet]. 2018 Feb 13 [cited 2018 Jul 20];18. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5809963/

36. Ezeudu CE, Chukwuka JO, Ebenebe JC, Igwe WC, Egbuonu I. Hypertension and prehypertension among adolescents attending secondary schools in urban area of South-East, Nigeria. Pan Afr Med J. 2018;31:145.

37. Lo JC, Sinaiko A, Chandra M, Daley MF, Greenspan LC, Parker ED, et al. Prehypertension and Hypertension in Community-Based Pediatric Practice. Pediatrics. 2013 Feb 1;131(2):e415–24.

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

39. Awotidebe A, Monyeki MA, Moss SJ, Strydom GL, Amstrong M, Kemper HCG. Relationship of adiposity and cardiorespiratory fitness with resting blood pressure of South African adolescents: the PAHL Study. J Hum Hypertens. 2016 Apr;30(4):245–51.

40. Wu L, Ma J, Fu L, Wang H, Li X, Dong B, et al. [Association between abdominal obesity and blood pressure among 7 to 10 years old Chinese children]. Zhonghua Yu Fang Yi Xue Za Zhi. 2013 Aug;47(8):689–94.

41. Jardim TV, Gaziano TA, Nascente FM, Carneiro C de S, Morais P, Roriz V, et al. Multiple cardiovascular risk factors in adolescents from a middle-income country: Prevalence and associated factors. PLOS ONE. 2018 Jul 5;13(7):e0200075.

42. 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. J Hypertens. 2017;35(6):1155–63.

43. Magliano ES, Guedes LG, Coutinho ESF, Bloch KV. Prevalence of arterial hypertension among Brazilian adolescents: systematic review and meta-analysis. BMC Public Health. 2013 Sep 11;13:833.

44. Ataklte F, Erqou S, Kaptoge S, Taye B, Echouffo-Tcheugui JB, Kengne AP. Burden of undiagnosed hypertension in sub-saharan Africa: a systematic review and meta-analysis. Hypertens Dallas Tex 1979. 2015 Feb;65(2):291–8.

45. Nkwana MR, Monyeki KD, Monyeki SM, Makata TT, Monyeki JM. Ellisras Longitudinal Study 2017: the association of fat patterning with blood pressure in Polokwane private school children aged five to 15 years (ELS 22). Cardiovasc J Afr. 2019 Jun 23;30(3):142–5.

46. 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 [Internet]. International Journal of Hypertension. 2018 [cited 2018 Jul 19]. Available from: https://www.hindawi.com/journals/ijhy/2018/3848591/abs/

47. Mphekgwana PM, Makgopa HM, Monyeki KD, Malatji JM, Mabila TE. Ellisras Longitudinal Study 2017: Childhood underweight and blood pressure status in a rural black population of South Africa (ELS 26). Cardiovasc J Afr. 2019 Jun 23;30(3):146–50.

48. Hillis S, Mercy J, Amobi A, Kress H. Global Prevalence of Past-year Violence Against Children: A Systematic Review and Minimum Estimates. Pediatrics. 2016 Mar;137(3):e20154079.

49. Kidman R, Palermo T. The relationship between parental presence and child sexual violence: Evidence from thirteen countries in sub-Saharan Africa. Child Abuse Negl. 2016 Jan 1;51:172–80.

50. Burton P, Ward CL, Artz L. The Optimus Study on Child Abuse, Violence and Neglect in South Africa [Internet]. Centre for Justice and Crime Prevention. [cited 2018 Jul 23]. Available from: http://www.cjcp.org.za/uploads/2/7/8/4/27845461/cjcp_ubs_proof_18.pdf

51. Burton P, Leoschut L. School Violence in South Africa, Results of the 2012 National School Violence Study [Internet]. Centre for Justice and Crime Prevention; 2013 [cited 2018 Jul 23]. Available from: https://www.saferspaces.org.za/uploads/files/Monograph12-School-violence-in-South_Africa.pdf

52. Richter LM, Mathews S, Kagura J, Nonterah E. A longitudinal perspective on violence in the lives of South African children from the Birth to Twenty Plus cohort study in Johannesburg-Soweto. SAMJ South Afr Med J. 2018 Mar;108(3):181–6.

53. Devries K, Knight L, Petzold M, Merrill KG, Maxwell L, Williams A, et al. Who perpetrates violence against children? A systematic analysis of age-specific and sex-specific data. BMJ Paediatr Open [Internet]. 2018 Feb 7 [cited 2018 Jul 23];2(1). Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5842994/

54. Breiding MJ, Mercy JA, Gulaid J, Reza A, Hleta-Nkambule N. A national survey of childhood physical abuse among females in Swaziland. J Epidemiol Glob Health. 2013;3(2):73–81.

55. Palermo T, Pereira A, Neijhoft N, Bello G, Buluma R, Diem P, et al. Risk factors for childhood violence and polyvictimization: A cross-country analysis from three regions. Child Abuse Negl. 2019 Feb 1;88:348–61.

56. Barth J, Bermetz L, Heim E, Trelle S, Tonia T. The current prevalence of child sexual abuse worldwide: a systematic review and meta-analysis. Int J Public Health. 2013 Jun;58(3):469–83.

57. Chigiji H, Fry D, Mwadiwa TE, Elizalde A, Izumi N, Baago-Rasmussen L, et al. Risk factors and health consequences of physical and emotional violence against children in Zimbabwe: a nationally representative survey. BMJ Glob Health. 2018 Jun 1;3(3):e000533.

58. Welcome to CDC stacks | Connecting the dots : an overview of the links among multiple forms of violence - 31552 | Stephen B. Thacker CDC Library collection [Internet]. [cited 2019 Oct 3]. Available from: https://stacks.cdc.gov/view/cdc/31552

59. Kumar AKS, Stern V, Subrahmanian R, Sherr L, Burton P, Guerra N, et al. Ending violence in childhood: a global imperative. Psychol Health Med. 2017 Mar 6;22(sup1):1–16.

60. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, et al. Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study. Am J Prev Med. 2019 Jun 1;56(6):774–86.

61. Anda RF, Felitti VJ, Bremner JD, Walker JD, Whitfield Ch, Perry BD, et al. The enduring effects of abuse and related adverse experiences in childhood. Eur Arch Psychiatry Clin Neurosci. 2006 Apr 1;256(3):174–86.

62. Shonkoff JP, Garner AS, The Committee on Psychosocial Aspects of Child and Family Health C on EC, Siegel BS, Dobbins MI, Earls MF, et al. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. Pediatrics. 2012 Jan 1;129(1):e232–46.

63. Copeland WE, Shanahan L, Hinesley J, Chan RF, Aberg KA, Fairbank JA, et al. Association of Childhood Trauma Exposure With Adult Psychiatric Disorders and Functional Outcomes. JAMA Netw Open. 2018 Nov 2;1(7):e184493–e184493.

64. Teicher MH, Samson JA. Annual Research Review: Enduring neurobiological effects of childhood abuse and neglect. J Child Psychol Psychiatry. 2016 Mar 1;57(3):241–66.

65. Shin SH, McDonald SE, Conley D. Patterns of adverse childhood experiences and substance use among young adults: A latent class analysis. Addict Behav. 2018;78:187–92.

66. Hillis SD, Mercy JA, Saul JR. The enduring impact of violence against children. Psychol Health Med. 2017 Apr 21;22(4):393–405.

67. Marie-Mitchell A, Kostolansky R. A Systematic Review of Trials to Improve Child Outcomes Associated With Adverse Childhood Experiences. Am J Prev Med. 2019 May 1;56(5):756–64.

68. Gilbert LK, Breiding MJ, Merrick MT, Thompson WW, Ford DC, Dhingra SS, et al. Childhood Adversity and Adult Chronic Disease: An Update from Ten States and the District of Columbia, 2010. Am J Prev Med. 2015 Mar 1;48(3):345–9.

69. Chang X, Jiang X, Mkandarwire T, Shen M. Associations between adverse childhood experiences and health outcomes in adults aged 18–59 years. PLOS ONE. 2019 Feb 7;14(2):e0211850.

70. Sonu S, Post S, Feinglass J. Adverse childhood experiences and the onset of chronic disease in young adulthood. Prev Med. 2019 Jun 1;123:163–70.

71. Hatcher AM, Gibbs A, Jewkes R, McBride R-S, Peacock D, Christofides N. Effect of Childhood Poverty and Trauma on Adult Depressive Symptoms Among Young Men in Peri-Urban South African Settlements. J Adolesc Health. 2019 Jan 1;64(1):79–85.

72. Kiburi SK, Molebatsi K, Obondo A, Kuria MW. Adverse childhood experiences among patients with substance use disorders at a referral psychiatric hospital in Kenya. BMC Psychiatry. 2018 18;18(1):197.

73. Brown NM, Brown SN, Briggs RD, Germán M, Belamarich PF, Oyeku SO. Associations Between Adverse Childhood Experiences and ADHD Diagnosis and Severity. Acad Pediatr. 2017 Jun;17(4):349–55.

74. Bright MA, Knapp C, Hinojosa MS, Alford S, Bonner B. The Comorbidity of Physical, Mental, and Developmental Conditions Associated with Childhood Adversity: A Population Based Study. Matern Child Health J. 2016 Apr 1;20(4):843–53.

75. Ramratnam SK, Han Y-Y, Rosas-Salazar C, Forno E, Brehm JM, Rosser F, et al. Exposure to gun violence and asthma among children in Puerto Rico. Respir Med. 2015 Aug 1;109(8):975–81.

76. Luby JL, Barch D, Whalen D, Tillman R, Belden A. Association Between Early Life Adversity and Risk for Poor Emotional and Physical Health in Adolescence: A Putative Mechanistic Neurodevelopmental Pathway. JAMA Pediatr. 2017 Dec 1;171(12):1168–75.

77. Burke NJ, Hellman JL, Scott BG, Weems CF, Carrion VG. The impact of adverse childhood experiences on an urban pediatric population. Child Abuse Negl. 2011 Jun 1;35(6):408–13.

78. Kerker BD, Zhang J, Nadeem E, Stein REK, Hurlburt MS, Heneghan A, et al. Adverse Childhood Experiences and Mental Health, Chronic Medical Conditions, and Development in Young Children. Acad Pediatr. 2015 Sep 1;15(5):510–7.

79. Flaherty EG, Thompson R, Dubowitz H, Harvey EM, English DJ, Proctor LJ, et al. Adverse Childhood Experiences and Child Health in Early Adolescence. JAMA Pediatr. 2013 Jul 1;167(7):622–9.

80. Thompson R, Flaherty EG, English DJ, Litrownik AJ, Dubowitz H, Kotch JB, et al. Trajectories of Adverse Childhood Experiences and Self-Reported Health at Age 18. Acad Pediatr. 2015 Sep 1;15(5):503–9.

81. Ford JL, Browning CR. Effects of exposure to violence with a weapon during adolescence on adult hypertension. Ann Epidemiol. 2014 Mar;24(3):193–8.

82. Wilson DK, Kliewer W, Sica DA. The relationship between exposure to violence and blood pressure mechanisms. Curr Hypertens Rep. 2004 Aug;6(4):321–6.

83. Wilson DK, Kliewer W, Teasley N, Plybon L, Sica DA. Violence exposure, catecholamine excretion, and blood pressure nondipping status in African American male versus female adolescents. Psychosom Med. 2002;64(6):906–15.

84. Riley EH, Wright RJ, Jun HJ, Hibert EN, Rich-Edwards JW. Hypertension in adult survivors of child abuse: observations from the Nurses' Health Study II. J Epidemiol Community Health. 2010 May 1;64(5):413–8.

85. Sumner JA, Kubzansky LD, Roberts AL, Gilsanz P, Chen Q, Winning A, et al. Post-traumatic stress disorder symptoms and risk of hypertension over 22 years in a large cohort of younger and middle-aged women. Psychol Med. 2016;46(15):3105–16.

86. Abouzeid M, Kelsall HL, Forbes AB, Sim MR, Creamer MC. Posttraumatic stress disorder and hypertension in Australian veterans of the 1991 Gulf War. J Psychosom Res. 2012 Jan;72(1):33–8.

87. Mason SM, Wright RJ, Hibert EN, Spiegelman D, Forman JP, Rich-Edwards JW. Intimate partner violence and incidence of hypertension in women. Ann Epidemiol. 2012 Aug;22(8):562–7.

88. Stein DJ, Scott K, Haro Abad JM, Aguilar-Gaxiola S, Alonso J, Angermeyer M, et al. Early childhood adversity and later hypertension: data from the World Mental Health Survey. Ann Clin Psychiatry Off J Am Acad Clin Psychiatr. 2010 Feb;22(1):19–28.

89. Nomura Y, Chemtob CM. Conjoined effects of low birth weight and childhood abuse on adaptation and well-being in adolescence and adulthood. Arch Pediatr Adolesc Med. 2007 Feb;161(2):186–92.

90. Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L, et al. Global Burden of Hypertension and Systolic Blood Pressure of at Least 110 to 115 mm Hg, 1990-2015. JAMA. 2017 10;317(2):165–82.

91. Nkeh-Chungag BN, Sekokotla AM, Sewani-Rusike C, Namugowa A, Iputo JE. Prevalence of hypertension and pre-hypertension in 13-17 year old adolescents living in Mthatha - South Africa: a cross-sectional study. Cent Eur J Public Health. 2015 Mar;23(1):59–64.

92. Meinck F, Cluver LD, Boyes ME, Loening-Voysey H. Physical, emotional and sexual adolescent abuse victimisation in South Africa: prevalence, incidence, perpetrators and locations. J Epidemiol Community Health. 2016 Mar 9;

93. Dawes A, Tredoux C, Feinstein A. Political violence in South Africa: Some effects on children of the violent destruction of their community. Int J Ment Health. 1989;18(2):16–43.

94. Richter L, Norris S, Pettifor J, Yach D, Cameron N. Cohort Profile: Mandela's children: The 1990 birth to twenty study in South Africa. Int J Epidemiol. 2007 Jun;36(3):504–11.

95. Kagura J, Adair LS, Musa MG, Pettifor JM, Norris SA. Blood pressure tracking in urban black South African children: birth to twenty cohort. BMC Pediatr. 2015 Jul 15;15:78.

96. Falkner B, Daniels SR, Flynn JT, Gidding S, Green LA, Ingelfinger JR, et al. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. 2004 Aug;114(2 III):555–76.

97. Munthali RJ, Kagura J, Lombard Z, Norris SA. Childhood adiposity trajectories are associated with late adolescent blood pressure: birth to twenty cohort. BMC Public Health. 2016 29;16:665.

98. StataCorp. Stata Statistical Software: Release 15. [Internet]. College Station, Texas, USA: StataCorp LLC.; 2017. Available from: http://www.stata.com

99. Hancock GR, Mueller RO. Best Practices in Structural Equation Modelling. In: Osborne JW, editor. Best Practices in Quantitative Methods. SAGE; 2008. p. 488–507.

100. Beran TN, Violato C. Structural equation modeling in medical research: a primer. BMC Res Notes. 2010 Oct 22;3:267.

101. Browne MW. Asymptotically distribution-free methods for the analysis of covariance structures. Br J Math Stat Psychol. 1984 May 1;37(1):62–83.

102. Schermelleh-Engel K, Moosbrugger H, Müller H. Evaluating the Fit of Structural Equation Models: Tests of Significance and Descriptive Goodness-of-Fit Measures. Methods Psychol Res. 2003;8(2):23–74.

103. Generalized structural equation modeling | Stata [Internet]. [cited 2018 Jul 24]. Available from: https://www.stata.com/features/overview/generalized-sem/

104. Su S, Wang X, Pollock JS, Treiber FA, Xu X, Snieder H, et al. Adverse childhood experiences and blood pressure trajectories from childhood to young adulthood: the Georgia stress and Heart study. Circulation. 2015 May 12;131(19):1674–81.

105. Gooding HC, Milliren C, McLaughlin KA, Richmond TK, Katz-Wise SL, Rich-Edwards J, et al. Child maltreatment and blood pressure in young adulthood. Child Abuse Negl. 2014 Nov;38(11):1747–54.

106. Flaherty EG, Thompson R, Litrownik AJ, Zolotor AJ, Dubowitz H, Runyan DK, et al. Adverse childhood exposures and reported child health at age 12. Acad Pediatr. 2009 Jun;9(3):150–6.

107. 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 Jan;41(1):5–14.

108. Dekkers JC, Snieder H, Van Den Oord EJCG, Treiber FA. Moderators of blood pressure development from childhood to adulthood: a 10-year longitudinal study. J Pediatr. 2002 Dec;141(6):770–9.

109. Araújo J, Ramos E, Barros H. Decreases in adiposity reduce the risk of hypertension: Results from a prospective cohort of adolescents. Prev Med. 2019 Mar 1;120:1–7.

110. Maximova K, O'Loughlin J, Paradis G, Hanley JA, Lynch J. Changes in anthropometric characteristics and blood pressure during adolescence. Epidemiology. 2010;21(3):324–31.

111. Szabo L, Szabo L, Kormos-Tasi J, Gácsi E, Scheuring N. SYSTOLIC AND DIASTOLIC BLOOD PRESSURE BY HEIGHT, BMI, GENDER AND AGE BETWEEN 14 AND 18 YEARS OF AGE. J Hypertens. 2019 Jul;37:e33.

112. Simbolon D, Yorita E, Talib RA. Consequences of Overweight and Obesity in Adolescence against the Risk of Hypertension in Adulthood. Kesmas Natl Public Health J [Internet]. 2019 Aug 1 [cited 2019 Nov 3];14(1). Available from: http://journal.fkm.ui.ac.id/kesmas/article/view/2723

113. Yang Y, Dong B, Wang S, Dong Y, Zou Z, Fu L, et al. Prevalence of high blood pressure subtypes and its associations with BMI in Chinese children: a national cross-sectional survey. BMC Public Health. 2017 26;17(1):598.

114. Quaresma FRP, da Silva Maciel E, dos Santos Figueiredo FW, Adami F. Factors associated with blood pressure disorders in Afro-descendant children and adolescents. BMC Pediatr [Internet]. 2019 Jul 20 [cited 2019 Nov 14];19. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6642598/

115. 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 Sep 7;163(9):818–25.

116. Barba G, Casullo C, Dello Russo M, Russo P, Nappo A, Lauria F, et al. Gender-Related Differences in the Relationships Between Blood Pressure, Age, and Body Size in Prepubertal Children. Am J Hypertens. 2008 Sep 1;21(9):1007–10.

117. Thompson MP, Kingree JB, Desai S. Gender Differences in Long-Term Health Consequences of Physical Abuse of Children: Data From a Nationally Representative Survey. Am J Public Health. 2004 Apr;94(4):599–604.

118. Séguin-Lemire A, Hébert M, Cossette L, Langevin R. A longitudinal study of emotion regulation among sexually abused preschoolers. Child Abuse Negl. 2017 Jan 1;63:307–16.

119. Dube SR, Anda RF, Whitfield CL, Brown DW, Felitti VJ, Dong M, et al. Long-term consequences of childhood sexual abuse by gender of victim. Am J Prev Med. 2005 Jun;28(5):430–8.

120. Annerbäck E-M, Sahlqvist L, Svedin CG, Wingren G, Gustafsson PA. Child physical abuse and concurrence of other types of child abuse in Sweden—Associations with health and risk behaviors. Child Abuse Negl. 2012 Jul 1;36(7):585–95.

121. Sylvestre M-P, Kâ K, Dugas EN, Zappitelli M, O'Loughlin J. Sex-specific trajectories of systolic and diastolic blood pressure in youth. J Hypertens. 2017;35(7):1416–23.

122. Gupta-Malhotra M, Banker A, Shete S, Hashmi SS, Tyson JE, Barratt MS, et al. Essential hypertension vs. secondary hypertension among children. Am J Hypertens. 2015 Jan;28(1):73–80.

123. Grimes DA, Schulz KF. Cohort studies: marching towards outcomes. The Lancet. 2002 Jan 26;359(9303):341–5.

124. Mann CJ. Observational research methods. Research design II: cohort, cross sectional, and case-control studies. Emerg Med J. 2003 Jan 1;20(1):54–60.

125. Tomarken AJ, Waller NG. Structural equation modeling: strengths, limitations, and misconceptions. Annu Rev Clin Psychol. 2005;1:31–65.

126. Puoane T, Steyn K, Bradshaw D, Laubscher R, Fourie J, Lambert V, et al. Obesity in South Africa: The South African Demographic and Health Survey. Obes Res. 2002;10(10):1038–48.

127. Klingberg S, Sluijs EMF van, Draper CE. "The thing is, kids don't grow the same": Parent perspectives on preschoolers' weight and size in Soweto, South Africa. PLOS ONE. 2020 Apr 6;15(4):e0231094.

128. Rice SM, Purcell R, McGorry PD. Adolescent and Young Adult Male Mental Health: Transforming System Failures Into Proactive Models of Engagement. J Adolesc Health. 2018 Mar 1;62(3, Supplement):S9–17.

129. Lawrence J, Ashford K, Dent P. Gender differences in coping strategies of undergraduate students and their impact on self-esteem and attainment. Act Learn High Educ. 2006 Nov 1;7(3):273–81.

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 Adatia Chivafa (Student number: 1839407) am a student registered for the degree of MSc Epidemiology in the field of Biostatistics in the academic year 2020.

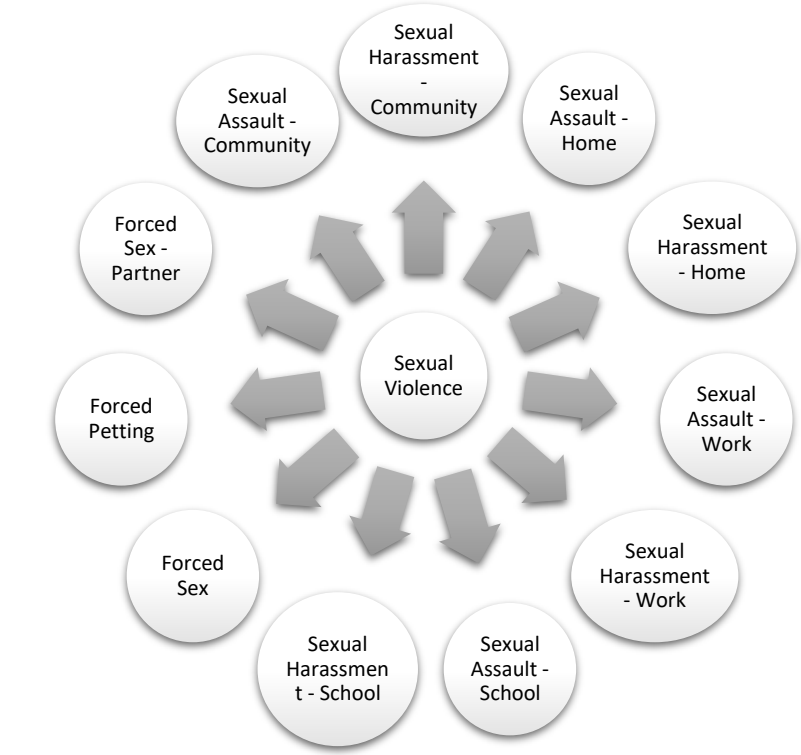
I hereby declare the following:

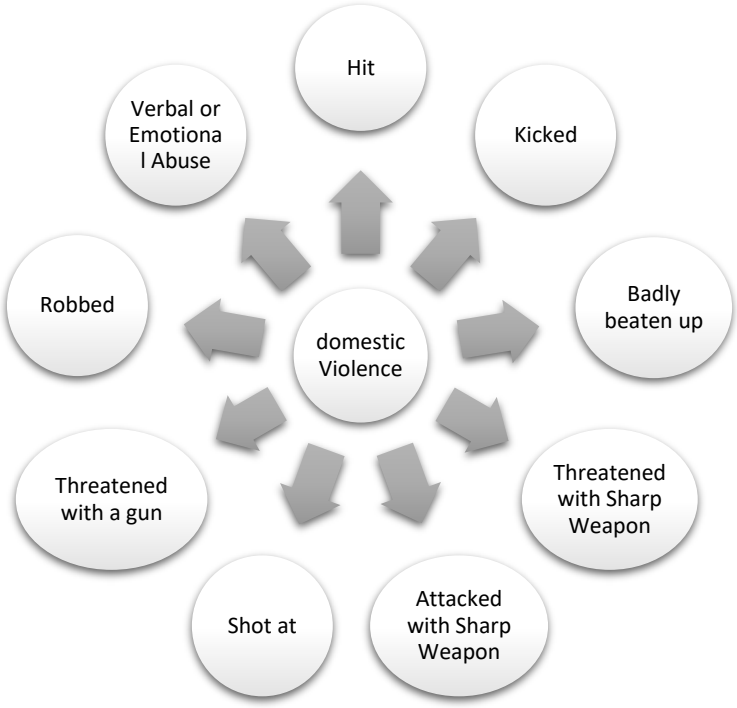
- I am aware that plagiarism (the use of someone else's work without their permission and/or without acknowledging the original source) is wrong.
- I confirm that the work submitted for assessment for the above degree is my own unaided work except where I have explicitly indicated otherwise.
- I have 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.

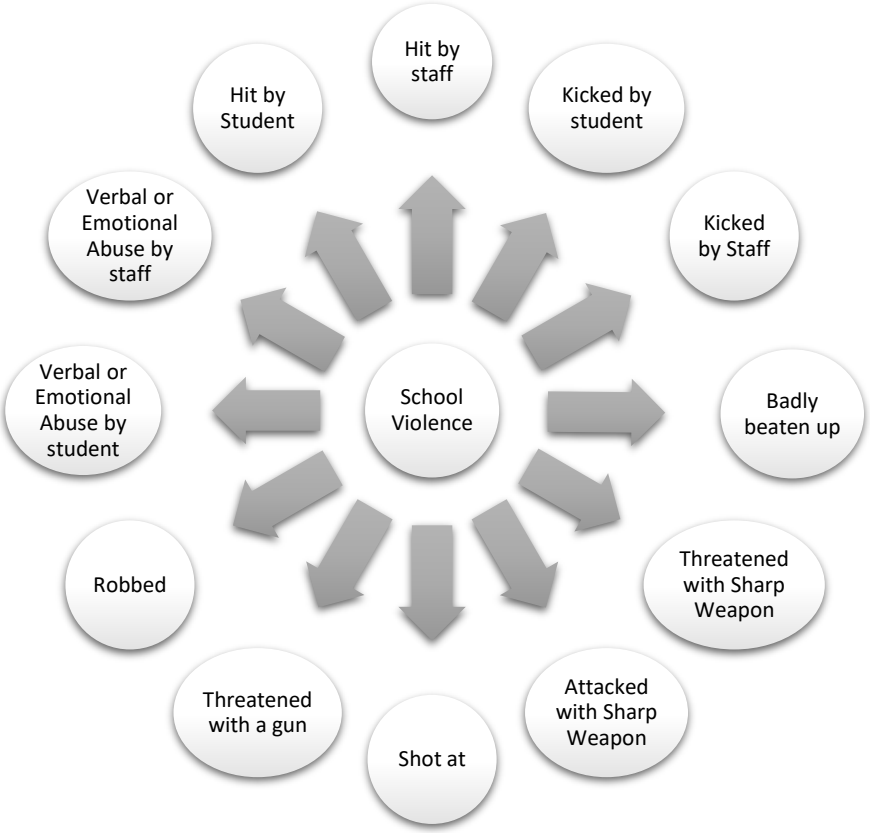
Signature:  Date: 28-09-2020

1

Appendix 2: Constructing experiences of violence variables






School Violence
Hit by staff
Kicked by student
Kicked by Staff
Badly beaten up
Threatened with Sharp Weapon
Attacked with Sharp Weapon
Shot at
Threatened with a gun
Robbed
Verbal or Emotional Abuse by student
Verbal or Emotional Abuse by staff
Hit by Student


Community Violence
Hit - Work or Community
Kicked - Work or Community
Badly beaten up
Threatened with Sharp Weapon
Attacked with Sharp Weapon
Shot at
Threatened with a gun
Robbed
Verbal or Emotional Abuse

Appendix 3: Questionnaires

SECTION E: VIOLENCE IN YOUR COMMUNITY, SCHOOL OR WORK-PLACE

In this section of the questionnaire we are going to talk about VIOLENCE in your community, school or work-place in the last 12 MONTHS (e.g. last Christmas, Easter, School term, Halloween, etc)

How often do the following apply to you (not on TV or in movies)	Never	Once or twice	A few times	Many times
I have heard gun shots				
I have seen somebody arrested				
I have seen drug deals				
I have seen someone being beaten up				
My house has been broken into while I was home				
I have seen somebody get stabbed				
I have seen somebody get shot				
I have seen a gun in my home				
I have seen gangs in my neighbourhood				
I have seen somebody pull a gun on another person				
I have seen someone in my home get shot or stabbed				

At school in the last 12 months, how often have you been:	Never	Once or twice	A few times	Many times
Hit by a student				
Hit by school staff				
Kicked or pushed by a student				
Kicked or pushed by school staff				
Badly beaten up				
Threatened with a knife or sharp weapon				
Attacked with a knife or sharp weapon				
Threatened with a gun				
Verbally or emotionally abused by a student, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Verbally or emotionally abused by school staff				

11

Sexually harassed by a student (unwelcome advances which continue after saying no)				
Sexually harassed by school staff				
Sexually assaulted (attacked)				
Robbed				

At work in the last 12 months , how often have you been:	**Never**	**Once or twice**	**A few times**	**Many times**
Hit by a colleague/co-worker				
Hit by your supervisor				
Kicked or pushed by a colleague/co-worker				
Kicked or pushed by your supervisor				
Badly beaten up				
Threatened with a knife or sharp weapon				
Attacked with a knife or sharp weapon				
Threatened with a gun				
Verbally or emotionally abused by a colleague, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Verbally or emotionally abused by your supervisor				
Sexually harassed by a colleague/coworker (unwelcome advances which continue after saying no)				
Sexually harassed by your supervisor				
Sexually assaulted (attacked)				
Robbed				

In your neighbourhood in the last 12 months, how often have you been:	**Never**	**Once or twice**	**A few times**	**Many times**
Hit				
Kicked				
Pushed or shoved				
Badly beaten up				
Threatened with a knife or sharp weapon				
Attacked with a knife or sharp weapon				
Threatened with a gun				
Verbally or emotionally abused, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Shot at				
Sexually harassed				
Sexually assaulted (attacked)				
Robbed				

At home, in the last 12 months, how often have you been:	**Never**	**Once or twice**	**A few times**	**Many times**
Hit				
Kicked				

Pushed or shoved				
Badly beaten up				
Threatened with a knife or sharp weapon				
Attacked with a knife or sharp weapon				
Threatened with a gun				
Verbally or emotionally abused, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Shot at				
Sexually harassed				
Sexually assaulted (attacked)				
Robbed				

At school in the last 12 months, how often have **YOU** done these things:	**Never**	**Once or twice**	**A few times**	**Many times**
Hit or kicked someone				
Pushed or shoved someone when you were angry				
Badly beaten someone up				
Threatened someone with a knife or sharp weapon				
Attacked someone with a knife or sharp weapon				
Threatened someone with a gun				
Verbally or emotionally abused someone, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Sexually harassed someone				
Robbed someone				
Been suspended from school				
Gotten into a fight after drinking or getting high				

At work in the last 12 months, how often have **YOU** done these things:	**Never**	**Once or twice**	**A few times**	**Many times**
Hit or kicked someone				
Pushed or shoved someone when you were angry				
Badly beaten someone up				
Threatened someone with a knife or sharp weapon				
Attacked someone with a knife or sharp weapon				
Threatened someone with a gun				
Verbally or emotionally abused someone, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Sexually harassed someone				
Robbed someone				
Been suspended from work				
Gotten into a fight after drinking or getting high				

Outside of school OR work in the last 12 months,	**Never**	**Once or**	**A few**	**Many**

how often have **YOU** done these things:		**twice**	**times**	**times**
Hit or kicked someone				
Pushed or shoved someone when you were angry				
Badly beaten someone up				
Threatened someone with a knife or sharp weapon				
Attacked someone with a knife or sharp weapon				
Threatened someone with a gun				
Verbally or emotionally abused someone, that is, being called names or having things said to you that make you feel bad about yourself or afraid				
Sexually harassed someone				
Robbed someone				
Gotten into a fight after drinking or getting high				

Did your caregiver, or an adult in your home, **ever** smack you (that is, hit you once or twice with an empty hand)?		
1. No	2. Yes, it happened just once	3. Yes it happened more than once
Did your caregiver, or an adult in your home, **ever** beat you with a strap, a belt, a stick, or a similar object?		
1. No	2. Yes, it happened just once	3. Yes it happened more than once

Appendix 4: Data Analysis Plan

Objective	Variables	Analysis Plan
3. To describe the demographic characteristics of the 18-year-old adolescents across hypertension status	• Hypertension • Home Violence • School Violence • Community Violence • Sexual Violence • Sex • Alcohol Abuse • Income • BMI • WHR • Maternal Education • Maternal Marital Status • Maternal Age • Parity • SBP • DBP	• Constructed the individual violence variables (see Appendix A for variables that were used in the construction) using aggregate scores. • Summary statistics for each of the variables. • Proportions for categorical variables for example, what proportion of females are hypertensive, pre-hypertensive or normotensive. • Means with standard deviations and medians with inter-quartile ranges (IQR) for continuous variables, for example, the mean BMI of hypertensive and normotensive teenagers.
4. To determine the association between hypertension, SBP, DBP and each of the explanatory variables	• Hypertension • Home Violence • School Violence • Community Violence • Sexual Violence • Sex • Alcohol Abuse • Income • BMI • WHR • Maternal Education • Maternal Marital Status • Maternal Age • Parity • SBP • DBP	• Exploring the relationship of each variable with hypertension, SBP, and DBP • The student's t-test and Mann Whitney tests for continuous variables like BMI • The Chi-square test and ANOVA for categorical variables like each of the experiences of violence variables and maternal education

5. To determine the association between hypertension, SBP, and DBP and experiences of violence adjusting for other predictors of hypertension amongst the 18-year-olds in the birth-to-twenty cohort using SEM.	• Hypertension • Home Violence • School Violence • Community Violence • Sexual Violence • Sex • Alcohol Abuse • Income • BMI • WHR • Maternal Education • Maternal Marital Status • Maternal Age • Parity • SBP • DBP	• Fitted SEM models using the proposed conceptual framework for the SBP and DBP outcomes • Fitted a GSEM, which fits networks of constructs to data; as it allows for continuous, binary, count, categorical, ordered and survival time measurements(103) for the hypertension outcome. • Logistic regression was used for intercept estimation

Appendix 5: SEM and GSEM tables

Measurement portion of the model/CFA model

Observed Variable	Latent Construct	B	β	SE	p-value
School Violence	Experiences of Violence	1.00	0.596		<0.001
Domestic Violence	Experiences of Violence	0.778	0.452	0.032	<0.001
Sexual Violence	Experiences of Violence	0.198	0.355	0.032	<0.001
Community Violence	Experiences of Violence	0.677	0.538	0.032	<0.001

$$\boldsymbol{\chi^2 = 9.67\ (df = 2, p = 0.008), SRMR = 0.019, RMSEA = 0.049\ CI_{90}: (0.021, 0.082), CFI = 0.983}$$

The association of experiences of violence with SBP adjusting for other factors

Endogenous Variable	Exogenous Variable	Direct Effects Coeff (95% CI)	Indirect Effects Coeff (95% CI)	Total Effects Coeff (95% CI)	p-value
SBP	Experiences of Violence	-0.222 (-1.568:1.123)		-0.222 (-1.568:1.123)	0.746
	Ethnicity	2.282 (-0.114:4.679)		2.282 (-0.114:4.679)	0.062
	Sex	**-7.238 (-8.856:-5.621)**	**2.072 (1.158:2.986)**	**-5.167 (-6.614:-3.720)**	<0.001
	Alcohol Abuse	-1.468 (-3.178:0.243)	0.426 (-0.092:0.944)	-1.042 (-2.803:0.72)	0.246
	Body Mass Index (BMI)	**0.729 (0.553-0.904)**		**0.729 (0.553-0.904)**	<0.001
	Mother's Marital Status	-1.684 (-3.374:0.007)		-1.684 (-3.374:0.007)	0.051
	Education	-1.424 (-3.727:0.879)		-1.424 (-3.727:0.879)	0.226
		-2.107 (-5.313:1.100)		-2.107 (-5.313:1.100)	0.198
	Mother's Age	-0.066 (-0.234:0.102)		-0.066 (-0.234:0.102)	0.443
	Income (thousands of Rands)	-0.059 (-0.145:0.026)	0.001 (-0.007:0.010)	-0.058 (-0.144:0.028)	0.184
	Parity	**0.863 (0.091:1.635)**		**0.863 (0.091:1.635)**	0.028
BMI	Sex	**2.689 (2.149:3.229)**		**2.689 (2.149:3.229)**	<0.001
	Alcohol Abuse	0.541 (-0.105:1.188)		0.541 (-0.105:1.188)	0.101
Experiences of Violence	Income (thousands of Rands)	-0.006 (-0.013:0.000)		-0.006 (-0.013:0.000)	0.053
	Sex	**-0.506 (-0.644:-0.368)**		**-0.506 (-0.644:-0.368)**	<0.001
	Alcohol Abuse	-0.142 (-0.295:0.011)		-0.142 (-0.295:0.011)	0.069
Sexual Violence	Income (thousands of Rands)		-0.001 (-0.003:0.000)	-0.001 (-0.003-0.000)	0.075
	Sex	**0.09 (0.022:0.158)**	**-0.116 (-0.17:-0.063)**	-0.026 (-0.078-0.025)	0.315
	Alcohol Abuse		-0.033 (-0.068:0.003)	-0.033 (-0.068-0.003)	0.073

Domestic Violence	Income (thousands of Rands)		-0.004 (-0.008:0.000)	-0.004 (-0.008-0.000)	0.071
	Sex	**0.421 (0.241:0.602)**	**-0.321 (-0.464:-0.177)**	0.1 (-0.04-0.241)	0.161
	Alcohol Abuse		-0.09 (-0.189:0.009)	-0.09 (-0.189-0.009)	0.076
School Violence	Income (thousands of Rands)		-0.006 (-0.013:0.000)	-0.006 (-0.013:0.000)	0.053
	Sex		**-0.506 (-0.644:-0.368)**	**-0.506 (-0.644:-0.368)**	<0.001
	Alcohol Abuse		-0.142 (-0.295:0.011)	-0.142 (-0.295:0.011)	0.069
Community Violence	Income (thousands of Rands)		-0.003 (-0.006-0.000)	-0.003 (-0.006-0.000)	0.058
	Sex		**-0.22 (-0.304:-0.137)**	**-0.22 (-0.304:-0.137)**	<0.001
	Alcohol Abuse		-0.062 (-0.132:0.009)	-0.062 (-0.132:0.009)	0.087

$\boldsymbol{\chi^2 = 30.92\ (df = 45, p = 0.946), SRMR = 0.017, RMSEA = 0.000\ CI_{90}: (0.000, 0.003), CFI = 1.000}$

Estimates in bold indicate p<0.05, Reference levels for ethnicity, sex, education, alcohol abuse, and marital status are non-black, male, primary/no education, yes and married respectively

The association of experiences of violence to DBP adjusting for other factors

Endogenous Variable	Exogenous Variable	Direct Effects (DE)	Indirect Effects (IE)	Total Effects (TE)	p-value
DBP	Experiences of Violence	0.659 (-0.509:1.828)		0.659 (-0.509:1.828)	0.269
	Ethnicity	1.836 (-0.219:3.891)		1.836 (-0.219:3.891)	0.080
	Sex	1.152 (-0.238:2.541)	0.518 (-0.218:1.254)	**1.67 (0.463:2.877)**	0.007
	Alcohol Abuse	-0.805 (-2.273:0.662)	0.077 (-0.215:0.37)	-0.728 (-2.198:0.743)	0.332
	Body Mass Index (BMI)	**0.316 (0.166:0.467)**		**0.316 (0.166:0.467)**	<0.001
	Mother's Marital Status	**-1.937 (-3.386:-0.487)**		**-1.937 (-3.386:-0.487)**	0.009
	Education	-1.943 (-3.918:0.032)		-1.943 (-3.918:0.032)	0.054
		-3.022 (-5.771:-0.272)		**-3.022 (-5.771:-0.272)**	0.031
	Mother's Age	**-0.166 (-0.31:-0.022)**		**-0.166 (-0.31:-0.022)**	0.024
	Income (thousands of Rands)	0.025 (-0.049:0.099)	-0.004 (-0.013:0.004)	0.021 (-0.053:0.094)	0.579
	Parity	0.645 (-0.017:1.307)		0.645 (-0.017:1.307)	0.056
BMI	Sex	**2.689 (2.149:3.229)**		**2.689 (2.149:3.229)**	<0.001

Endogenous Variable	Exogenous Variable	Direct Effects (DE)	Indirect Effects (IE)	Total Effects (TE)	p-value
	Alcohol Abuse	0.541 (-0.105:1.188)		0.541 (-0.105:1.188)	0.101
Experiences of Violence	Income (thousands of Rands)	-0.006 (-0.013:0)		-0.006 (-0.013:0)	0.054
	Sex	**-0.505 (-0.643:-0.367)**		**-0.505 (-0.643:-0.367)**	<0.001
	Alcohol Abuse	-0.142 (-0.295:0.01)		-0.142 (-0.295:0.01)	0.067
Sexual Violence	Income (thousands of Rands)		-0.001 (-0.003:0.000)	-0.001 (-0.003:0.000)	0.075
	Sex	**0.090 (0.022:0.158)**	**-0.117 (-0.170:-0.063)**	-0.026 (-0.078:0.025)	0.316
	Alcohol Abuse		-0.033 (-0.069:0.003)	-0.033 (-0.069:0.003)	0.071
Domestic Violence	Income (thousands of Rands)		-0.004 (-0.008:0.000)	-0.004 (-0.008:0.000)	0.071
	Sex	**0.420 (0.241:0.600)**	**-0.32 (-0.462:-0.179)**	0.100 (-0.04:0.24)	0.163
	Alcohol Abuse		-0.090 (-0.189:0.009)	-0.09 (-0.189:0.009)	0.074
School Violence	Income (thousands of Rands)		-0.006 (-0.013:0.00)	-0.006 (-0.013:0.000)	0.054
	Sex		**-0.505 (-0.643:-0.367)**	**-0.505 (-0.643:-0.367)**	<0.001
	Alcohol Abuse		-0.142 (-0.295:0.01)	-0.142 (-0.295:0.010)	0.067
Community Violence	Income (thousands of Rands)		-0.003 (-0.006:0.00)	-0.003 (-0.006:0)	0.058
	Sex		**-0.223 (-0.307:-0.139)**	**-0.223 (-0.307:-0.139)**	<0.001
	Alcohol Abuse		-0.063 (-0.134:0.009)	-0.063 (-0.134:0.009)	0.085

$\boldsymbol{\chi^2 = 32.37\ (df = 45, p = 0.921), SRMR = 0.018, RMSEA = 0.000\ CI_{90}: (0.000, 0.008), CFI = 1.000}$

Estimates in bold indicate p<0.05, Reference levels for ethnicity, sex, education, alcohol abuse, and marital status are non-black, male, primary/no education, yes and married respectively

Appendix 6: Ethics Certificate

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

R14/49 Miss Adatia Chivafa

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M181007

NAME: (Principal Investigator)	Miss Adatia Chivafa
DEPARTMENT:	School of Public Health
PROJECT TITLE:	The association between Experiencing Violence and Hypertension in Urban South African Adolescents
DATE CONSIDERED:	26/10/2018
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Dr Juliana Kagura
APPROVED BY:	Dr CB Penny, Chairperson, HREC (Medical)
DATE OF APPROVAL:	29/10/2018

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary on the Third Floor, Faculty of Health Sciences, Phillip Tobias Building, 29 Princess of Wales Terrace, Parktown, 2193, University of the Witwatersrand. I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to resubmit the application to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. In this case, the study was initially reviewed in October and will therefore be due in the month of October each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature

Date 02.11.2018

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES