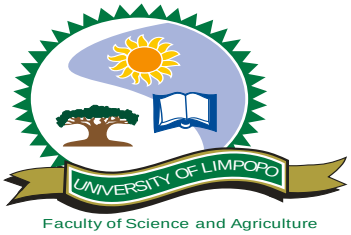

A LONGITUDINAL INVESTIGATION ON THE EFFECTS OF SODIUM AND POTASSIUM INTAKE HAVE ON THE DEVELOPMENT OF HYPERTENSION AND ABDOMINAL OBESITY FROM CHILDHOOD TO YOUNG ADULTHOOD AMONGST ELLISRAS RURAL POPULATION, SOUTH AFRICA

by

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DISSERTATION/THESIS

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SUPERVISOR: Prof KD Monyeki

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DECLARATION

I declare that the research project titled: A longitudinal investigation on the effects of sodium and potassium intake have on the development of hypertension and abdominal obesity from childhood to adulthood amongst Ellisras rural population, South Africa. Hereby submitted to the University of Limpopo, for the degree of Masters of Science in Physiology has not previously been submitted by me for a degree at this or any other university; that it is my work in design and in execution, and that all material contained herein has been duly acknowledged.

Raphadu, TT (Ms) 07 APRIL 2023

Surname, initials (Title) **Date**

ABSTRACT

Background: Hypertension (HT) and obesity have both been on the rise in children. Each is associated with an increase in cardiovascular disease risk and both track into adulthood.

Objectives: Hence, this study aimed to identify the association of sodium intake (Na), potassium (K) intake, and sodium-to-potassium (Na/K) ratio with the development of HT and abdominal obesity amongst the Ellisras rural population over time.

Methods: In this longitudinal study, data on dietary intake of Na and K were collected using a 24-h recall questionnaire from a total of 325 participants tracking them from 1999 (5–12 years), 2001 (7–14 years) and 2015 (18–30 years). Blood pressure (BP) and anthropometric measurements [waist circumference (WC) and height] Parametric (independent t-test) and Chi-square/Fishers' exact tests were conducted to determine the difference between the years for numerical data and categorical variables. A generalized estimating equation (GEE) was conducted to assess the association of Na intake, K intake; and their ratio on BP, WC and WHtR.

Results: Our results indicate a significant positive association between K intake and WHtR [β= 0.019, (95% CL: 0.004, 0.034) *p*-value= 0.012], and even the model was adjusted for age and sex there was still an association with WHtR. Na/K ratio was associated with SBP [β= 4.326, (95% CL: 2.056, 6.595) *p*-value= < 0.001], DBP [β= 2.028, (95% CL: 0.703, 3.353) *p*-value= 0.003], WC [β= 4.191, (95% CL: 2.080, 6.302) *p*-value= < 0.001] and WHtR [β= 0.014, (95% CL: 0.003, 0.026) *p*-value= 0.015], respectively. Furthermore, Na/K was shown to be associated with an increased risk of developing HT [Exp = 1.603, (95% CL: 1.164, 2.207) p-value= 0.004] and abdominal obesity [Exp = 1.797, (95% CL: 1.207, 2.677) p-value= 0.004].

Conclusion: In our study we observed that an increase in Na/K it's a predictor of HT and abdominal obesity over time compared to Na and K alone. However, more studies are required to further prove this.

Keywords: hypertension, abdominal obesity, sodium, potassium, longitudinal study

DEDICATION

I dedicate this dissertation to my parents (Elias and Florina Raphadu), to my two sisters (Portia and Basetsana Raphadu), my little brother (Dimakatso Raphadu) and my nephews (Katlego, Sphamandla and Mogomotsi Mashishi) for their support, love, prayers and care they gave during this process, and especially during the covid pandemic.

ACKNOWLEDGEMENTS

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LIST OF ABBREVIATIONS

ABBREVIATION	DESCRIPTION
BMI	Body Mass Index
BP	Blood pressure
CVDs	Cardiovascular diseases
DASH	Dietary Approaches to Stop Hypertension
DALYs	Disability-adjusted life-years
DBP	Diastolic Blood Pressure
ELS	Ellisras Longitudinal Study
GEE	Generalized estimating equation
GWAS	Geno-wide Association Study
HT	Hypertension
ISAK	International Society for the Advancement of Kinanthropometry
K	Potassium
LMICs	Low- and middle- income countries
mg	milligram
mmol	milli-moles
Na	Sodium
NCDs	Non-communicable diseases

NHLBI	National Heart, Lung and Blood Institute
NaCl	Sodium chloride
Na/K	Sodium-to-potassium ratio
PSSA	Physiology Society of Southern Africa
SAFOODS	South African Food Composition Database System
SBP	Systolic Blood Pressure
SD	Standard Deviation
SPSS	Statistical Package for Social Sciences
TREC	Turfloop Research Ethics
WC	Waist circumference
WHO	World Health Organization
WHR	Waist-to-hip ratio
WHtR	Waist-to-height ratio

CHAPTER 1

PROBLEMS AND AIMS OF THE STUDY

1.1. Problem statement

1.2. Rationale

1.3. Aim and Objectives

1.4. Research questions

1.5. Hypothesis

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1.1. PROBLEM STATEMENT

Hypertension (HT) is becoming more common in children and adolescents (Adrogué and Madias, 2014). Blood pressure (BP) tracking in longitudinal studies beginning in childhood may provide insight into the aetiology of HT in adults, but few studies have addressed the absence of longitudinal data on BP in children and adolescents in the same context (Schutte *et al*., 2003; Monyeki *et al*., 2006; Kemp *et al*., 2011; Goon *et al*., 2013).

Most research has focused on the health implications in adults; however, it has recently been demonstrated that a minor salt reduction in children may result in an immediate drop in blood pressure and, if sustained, may minimise the future rise in blood pressure with age (Huybrechts *et al*., 2011). Although the correlation between sodium (Na) and potassium (K) consumption has primarily been demonstrated concerning BP, there have lately been a few studies demonstrating a link between Na intake and body weight in children and adolescents irrespective of total energy intake (Grimes *et al*., 2013; Woodruff *et al*., 2014; Yoon and Oh, 2013). However, the combined influence of dietary Na and K consumption in the development of other diseases, such as overweight and obesity, is less well understood, particularly in children (O'Donnell *et al*., 2011). According to Wang et al. (2002), children's nutritional consumption habits are likely to persist from childhood into adolescence. As a result, children with high salt diets are more likely to eat large levels of salt as adults, increasing their chances for HT and cardiovascular diseases (CVDs) (Huybrechts *et al*., 2011).

Although there is sufficient evidence regarding Na and K, there are very few longitudinal studies in the world, especially in Africa. A study conducted in Africa only assessed proportion frequency of Na and K and did not track the changes in Na, K and NA/K ratio over time (Saeid *et al*., 2018). The study conducted in Ellisras rural children by van Den Ende et al. (2014) and Mashiane et al. (2018) focused their study on the association between Body Mass Index (BMI) and dietary intake in children and young adults, respectively. Studies regarding the association between micronutrients such as Na and K and HT and abdominal obesity has received little attention. Thus, highlighting the need for such a study, especially in rural African populations.

1.2. RATIONALE

The rise in the prevalence of HT is quickly becoming a global problem. This significant growth was seen in adults as opposed to children (Kagura *et al*., 2015). This was because HT was a disorder that was seldom discovered in children because of exclusion routine screening in public sectors (Kagura *et al*., 2015). In South Africa, few studies that examined BP profiles in rural and peri-urban black children and adolescents discovered a prevalence of HT ranging from 1% to 25.9%. (Schutte *et al*., 2003; Monyeki *et al*., 2006; Kemp *et al*., 2011; Goon *et al*., 2013). High BP at a young age may be a predictor of HT later in life (Aounallah-Skhiri *et al*., 2012).

However, according to He et al. (2008), lowering the source of Na and K may lead to a reduction in the health issues associated with BP. The two researchers backed up this claim (Beauchamp and Mennella, 2009; Campanozzi *et al*., 2015). There are considerable attempts, mostly in Europe, attempting to discover a technique to minimise Na intake among youngsters to maintain a healthy level of BP throughout life (Grimes *et al*., 2017).

Obesity has lately attracted attention as another prospective health-related result, despite the well-documented deleterious health effects of excessive Na consumption on HT (He *et al*., 2013). Several studies have found a link between Na and adult obesity (Adrogué *et al*., 2007; Aburto *et al*., 2013; Drenjancevic-Peric *et al*., 2011). Furthermore, research from nationally representative populations in South Korea, the United Kingdom, and Australia found a substantial positive connection between salt and obesity in both children and adults (Adrogué *et al*., 2007; Millen *et al*., 2013; Zhang *et al*., 2013). Furthermore, despite the endeavour to reduce Na and the relevance of K, the ability to lower the level of BP and abdominal obesity (obesity), few cross-sectional or longitudinal studies in Africa have studied the patterns of Na and K consumption and their health impacts.

In November 1996, a longitudinal study was started to monitor the growth, health, and lifestyle of a group of children in the Ellisras rural area. These children were within the 3 to 10-year age group at baseline (1996) and are now 26 to 34 years old (born between 1986 and 1994). It is not known how these parameters will change into young adulthood.

This population was part of the target population in the present study. Ellisras was selected as a study site as it is already part of a mixed-longitudinal study that monitors the growth, health, and lifestyle of rural children of South Africa. The data collected on dietary intake (Na and K), BP and abdominal indices during studies conducted in 1996 and between 1999–2015 in Ellisras will be utilised in this current study to determine how micronutrients such as Na and K may lead to the development of HT and abdominal obesity. These data will be included in this study to establish trends over time.

The rationale for carrying out this study in Ellisras is as follows:

- Despite that the Ellisras study has provided Baseline data on the children's physical growth, this study will permit a clear understanding of the relationship between the children's Na, K, and Na/K ratio, BP and abdominal indices over time.
- The Ellisras study concentrates on children's growth but does not examine the possible influence of the children's growth on Na, K, and Na/K ratio on BP and abdominal indices over time. Therefore, this study intends to fill this gap.
- The 24-hr recall questionnaire used at Ellisras has already been validated and standardised. Therefore, if the present study were to be carried out in a different population, the validity and reliability of the measurement procedures will have to be re-established and this would not be cost-effective.
- Sufficient rapport and necessary contacts have been established with the Principals and Teachers of primary schools and traditional leaders and political leaders in the Ellisras rural area. Therefore, it will not be difficult to enlist their cooperation in adding the dimension of Na, K, and Na/K ratio, BP and abdominal obesity over time to the existing Ellisras Longitudinal Study.

This study is not designed only to determine the effect of Na, K and their ratio on HT and abdominal obesity amongst Ellisras children, but to provide a glimpse of data into the consumption of Na, K and their ratio; and the effect they have on HT and abdominal obesity. As the relationship between micronutrients such as Na, K, and HT and abdominal obesity have received little attention in Africa. Because majority of the studies conducted were done in Europe, North America and Asia. Hence, the necessity for such a research, particularly among rural African people.

1.3. AIM AND OBJECTIVES

1.3.1. AIM

To investigate the effect of Na, and K intake and their ratio on HT and abdominal obesity amongst Ellisras rural children (and adults) overtime.

1.3.2. OBJECTIVES

I. To determine the proportion frequency of high dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies.

II. To track the prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies over time.

III. To determine the average concentration level of Na and K and NA/K ratio over time.

IV. To determine which of the selected micronutrients (Na, K, and Na/K ratio) will be associated on BP and abdominal indices over time by using the GEE (linear) technique.

V. To determine which of the selected micronutrients (Na, K, and Na/K ratio) will be the predictor for HT and abdominal obesity.

1.4. RESEARCH QUESTIONS

I. What is the proportion frequency of high dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies over time?

II. What are the prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies over time?

III. Will the average concentration of the dietary intake of Na and K and NA/K ratio increase over time?

IV. What is the effect of Na, K, and Na/K ratio on BP and abdominal indices over time by using the GEE (linear) technique?

V. Which of these micronutrients: Na, low K, and Na/K ratio will be the predictor HT and abdominal obesity?

1.5. HYPOTHESIS

I. Hypothesis 1: The proportion frequency of dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies over time will increase in the population of Ellisras, from childhood to young adulthood.

II. Hypothesis 2: The prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies will show an increase over time.

III. Hypothesis 3: The average concentration of Na and NA/K ratio will increase over time, whilst K intake decrease in the Ellisras rural children into young adulthood.

IV. Hypothesis 4: All selected micronutrients (Na, K, and Na/K ratio) will be associated with BP and abdominal indices over time by using the GEE (linear) technique.

V. Hypothesis 5: Na/K ratio will be found to the predictor of the risk of developing HT and abdominal obesity compared to Na and K alone over time.

1.6. SCIENTIFIC CONTRIBUTION

Although there is sufficient evidence regarding sodium and potassium, there are very few longitudinal studies that track the prevalence of HT, abdominal obesity and the effects of Na and K on BP and abdominal indices. In this regard, this study serves to add new information to the scientific knowledge on how the intake levels of Na and K may have on the development of hypertension and abdominal obesity over time from childhood to adulthood, especially from an African perspective as most literature is based on

American, European and Asian studies. Part of the results from this research project has been presented at the Physiology Society of Southern Africa (PSSA) conference 2021 and Faculty Research Day. Additionally, the results will be presented at the Ellisras non-communicable conference 2022 to peers to promote and further raise awareness in a rural setting on how dietary intake can/may cause the development of HT and abdominal obesity.

1.7. Structure of the dissertation

1. Chapter 1 – Problems and aim of the study

2. Chapter 2 – Literature review

3. Chapter 3 – Materials and methods

4. Chapter 4 – Results and Discussion

5. Chapter 5 – Introduction, summary, conclusion, and recommendations

6. Peer-reviewed articles published in International Journal were compiled as an addendum

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CHAPTER 2

LITERATURE REVIEW

2.6. Generalized estimating equation for longitudinal analysis

2.1. INTRODUCTION

According to the World Health Organization (WHO), noncommunicable diseases (NCDs) are the leading causes of mortality and morbidity in the world, killing 41 million people each year, accounting for 71% of all deaths (WHO, 2012). (WHO, 2012). Several studies have found that nutrition is a significant risk factor for NCDs. Furthermore, the prevalence of NCDs has steadily increased throughout the years (Meier *et al*., 2015; Saeid *et al*., 2018). High amounts of Na and low levels of K can predict HT, obesity, CDVs, cancer, and osteoporosis (Mozaffarian *et al*., 2014).

Although these diseases are more common in adults, there is a growing concern since they are being found in children and adolescents (Patterson *et al*., 2009; Aburto et al, 2013). A progressive rise in BP from infancy or young adulthood to middle age may predict BP levels and the occurrence of HT later in life (Caudarella *et al*., 2009). Elevated Na intake and insufficient K intake may influence the development of HT. Excessive K consumption, frequently found in fruits and vegetables such as bananas, potatoes, and spinach, can mitigate the detrimental effects of high sodium intake on BP (WHO, 2012).

Dietary patterns formed from childhood through maturity have shown that children with high Na and low K intake tend to maintain those levels over time (Aburto *et al*., 2013). Although the relationship between Na and K consumption has only been demonstrated in relation to BP, there have lately been a few studies demonstrating a link between Na intake and body weight in children and adolescents irrespective of total calorie intake (Grimes *et al*., 2013; Rodrigues *et al*., 2014; Woodruff *et al*., 2014; Yoon and Oh, 2013). However, the combined influence of dietary Na and K consumption on the aetiology of other disorders, such as overweight and obesity, is less well understood, particularly in children (O'Donnell *et al*., 2011). Furthermore, there is an urgent need to educate the public about the need of maintaining a low dietary Na intake and enough K in children, as nutrition in childhood can play a considerable role in determining adult dietary patterns (He *et al*., 2008).

2.2. HYPERTENSION

HT, commonly known as high blood pressure, is a disorder in which the blood vessels have continuously high pressure (WHO, 2013). HT is diagnosed if, when it is measured on two different days (WHO, 2021). In children and adolescents, HT is characterised differently than in adults, and there are several phases of HT. In children and adolescents, HT is defined as systolic and/or diastolic blood pressure more than the 95th percentile for height, gender, and age. Furthermore, normal BP is less than the 90th percentile, but pre-HT BP is between the 90th and 95th percentiles for height, gender, and age, or greater than 120/80 mmHg (Parsekar *et al*., 2015). On the hand, normal adult BP is defined as SBP of 120 mm Hg and DBP of 80 mm Hg. HT in adults is defined as a SBP of 140 mm Hg or higher and/or a DBP of 90 mm Hg or higher (WHO, 2013). Normal SBP and DBP levels are critical for the effective function of major organs such as the heart, brain, and kidneys, as well as overall health and well-being (WHO, 2013).

2.2.1. Hypertension globally in adults

HT is the world's most modifiable risk factor for CVDs, disability, and death. It affects around a billion individuals worldwide and is responsible for over 9 million deaths per year (WHO, 2013). Furthermore, HT previously contributed significantly to worldwide disability-adjusted life-years (DALYs) in 2015, accounting for 9.2% of DALYs in men and 7.8% of DALYs in women (GBD 2015 Risk Factors Collaborators, 2015). A systematic review found that childhood HT rose from 1994 to 2018, with the pooled estimate at 4% among people aged 19 and under. In 2015, the incidence of childhood HT varied from 4.32% among children aged 6 to 3.28% among people aged 19 to 7.89% among children aged 14 years (Song *et al*., 2019).

HT is becoming more common, particularly in poor and middle-income nations (LMICs). According to estimates, 31.1% of individuals (1.39 billion) globally had hypertension in 2010. Adult HT prevalence was greater in LMICs (31.5%, 1.04 billion people) than in high-income countries (28.5%, 349 million individuals) (Mills *et al*., 2020). Adults with HT grew from 594 million in 1975 to 1.13 billion in 2015, with the majority of the growth occurring

in low- and middle-income nations. This surge is mostly attributable to an increase in HT risk factors in those groups (WHO, 2021).

2.2.2. Trends of hypertension in the African region

HT was once uncommon in African populations, the burden has continuously increased over the past decades in these populations, which is driven by reduced physical activity, unhealthy diet and obesity (NCD Risk Factor Collaboration, 2017). According to the WHO, the African region's hypertensive prevalence is the highest at 46% in adults aged 25 years and above compared to the American region (WHO, 2011). HT incidence and cardiovascular mortality have been increasing in sub-Saharan Africa over the past years (Ataklte *et al*., 2015) and are projected to nearly double by the year 2030 (Damasceno *et al*., 2009).

African countries have a scarcity of longitudinal data on children and adolescents' BP. Hypertension has previously not been seen as a concern in children but in adults. However, there is growing evidence of increasing prevalence in children (Bugaje *et al*., 2005; Samuels, 2012; Okoh and Alikor, 2013) with many adult HT beginning during childhood. The prevalence of HT in Africa ranges between 0–22.3% in children and adolescents, based on studies conducted in certain Western countries of Africa and South Africa. The increase in the prevalence of HT is due to both urbanisation and westernisation in African countries (Essouma *et al*., 2015).

2.2.3. Trends of hypertension in South Africa

A systematic review of articles published on HT between 2000 and 2013 in sub-Saharan Africa conducted by Ataklte et al. (2015) reported a pooled HT prevalence of 30% in adults and a range from 14.7 to 69.9% depending on the site and age. Studies conducted in South Africa included in this review reported HT prevalence of 14.7% (Steyn *et al*., 2001), 49.8% (Basu & Millett, 2013) and 46.2% (Maseko *et al*., 2011). In addition, Bradshaw et al. (2010) also reported a significant increase in HT from 1998 to 2008, which projects a further increase in strokes and heart attacks in future.

HT has become an evolving health problem in South Africa. South Africa has conducted a few studies that profiled BP in children and adolescents (Kagura *et al*., 2015). The prevalence of HT has been reported to be ranging from 1 to 25.9% in black children and adolescents. However, these studies conducted were cross-sectional studies and mostly conducted in rural settings. These studies were not designed to be established to trace high BP in late adolescents (Kagura *et al*., 2015). Furthermore, Kagura et al. (2015) reported a longitudinal tacking of the prevalence of HT, which ranged from 8.4% in black children aged 5 years to 24.4% at age 18 years. The prevalence of pre-HT and HT reported in South African grade ones reported in the study conducted by Kemp et al. (2011) was 8.5% and 24.9%.

2.2.4. Under-diagnosis of hypertension

The under-diagnosis of HT in children and adolescents is a major concern. Even when accurate documentation of paediatric BP is available, the majority of such instances do not adopt acceptable diagnosis and therapy measures (Hansen *et al*., 2007). This is especially significant given that children and young adolescents with BP higher than the 90th percentile for their age are roughly three times more likely to develop HT than their peers with BP at the 50th percentile (Bernstein, 2007). Furthermore, HT in young people is generally undiagnosed and mistreated, particularly in LMICs (Samuels, 2012). Victims of HT are often unaware that they have the ailment, and as a result, many present with difficulties or unexpected death, earning it the moniker "silent killer" (Ekore *et al*., 2009; Ataklte *et al*., 2015; Adeloye *et al*., 2015).

Although there is an increase in the prevalence of HT, there has been an underdiagnosis of HT in children and adolescents. The difficultly to diagnose HT in children and adolescents is because HT diagnosis requires accurate use of the standardised chart. The chart is specific for age, height and sex (Salman *et al*., 2011). This process is time-consuming for physicians who are already overworked in developing countries (Nkeh-Chungag *et al*., 2015). Because the focus of HT screening has primarily been on adults, the diagnosis of high BP in children and adolescents is uncommon. As HT is a complicated disease, it is difficult to diagnose (Ewald and Haldeman, 2015).

Most developing countries already have a high burden of the disease, as well as a high ratio of patients to physicians. This leads to most children and adolescents being undiagnosed due to a limited number of physicians (Nkeh-Chungag *et al*., 2015). The misconception that HT is only a disease for adulthood has contributed towards the low diagnosis rate in children and adolescents, as they are often excluded from BP screening in adults (Nkeh-Chungag *et al*., 2015).

A limitation of evidence of risk factors associated with HT and the lack of medical resources that enables the diagnosis of HT in children and adolescents has led to a low rate of diagnosis in children and adolescents (Salman *et al*., 2011). Although there are electronic programmes and tables to assist with the diagnosis of HT, it is still difficult for paediatrics clinicians to navigate these tools in their workflow (Hansen *et al*., 2007).

2.2.5. Risk factors associated with hypertension

There is evidence that the onset of HT and its complications are influenced by a variety of factors. Both changeable and immutable factors are included in this classification. However, the main risk factors for HT are modifiable factors (reversible) rather than unmodifiable elements (age, genetics and sex, which are irreversible) (van de Vijver *et al*., 2013). These factors include environmental and lifestyle factors. Behavioural factors that affect HT— tobacco use, excessive alcohol use, physical inactivity, unhealthy diet (high salt intake and, insufficient fruit and vegetable consumption), urbanisation and obesity—have a stronger association with and causal relationship with HT over time. These behaviours are consequences of urbanization in developing countries (van de Vijver *et al*., 2013). The literature clearly demonstrates that hypertension is caused by several risk factors and that, whereas Na and K consumption have a substantial link with HT and are the focus of the current study, they do not induce high BP in isolation. These modifiable risk variables are part of the NCD cycle and should not be ignored while looking at a country's HT pandemic (Swanepoela *et al*., 2014).

While geographical and demographic inequalities in HT prevalence have been documented for decades, the causes for these variances in illness risk remain unexplained. The HT demographic and regional tendencies are comparable to those of

low birth weight providing support for the "Barker Hypothesis" postulates that adult-onset illness has a prenatal basis. In reality, ecological and observational studies conducted all around the world have found substantial connections between low birth weight and higher risks of HT (Jebasingh and Thomas, 2022). Regardless, the processes for the relationship has not been properly explained, and documented. Proposed mechanisms include decreased nephrogenesis, which has some supporting data with a greater pressure natriuresis threshold and increased vulnerability to progressive renal disease, as well as poor endothelial development as well as enhanced glucocorticoid sensitivity (Jebasingh and Thomas, 2022).

Figure 2.2.5.1: The risk factors of hypertension (Ewald and Haldeman, 2015).

2.2.5.1. Modifiable risk factors associated with hypertension

- **Tobacco use**:

Tobacco use has been linked to an increased risk of HT and CVDs such as stroke, thrombosis, and heart attack. Smoking causes an immediate increase in BP, resulting in smokers having higher ambulatory BP levels than non-smokers. Quitting smoking is known to lower the overall risk of CVDs. To reduce smoking at the population level, multi-sectoral interventions such as raising tobacco taxes, prohibiting tobacco advertisements, and prohibiting smoking in public places must be implemented (Beaglehole *et al*., 2011).

- **Alcohol consumption**:

In Africa, alcohol consumption is relatively common. There is a direct relationship between high levels of alcohol consumption and specific patterns of consumption (such as binge drinking) and an increased risk of HT. In Nigeria, the effect of heavy drinking on increasing BP levels has been described (van de Vijver *et al*., 2013). Interventions to reduce alcohol consumption should be implemented across multiple sectors and tailored to the local context. Such interventions, like those used to reduce tobacco use, including raising alcohol taxes and prohibiting alcohol advertising, particularly to young people (Beaglehole *et al*., 2011).

- **Inadequate physical activity**:

Adequate physical activity has been shown to have numerous health-promoting effects, including a direct, independent role in the reduction of HT (Gersh *et al*., 2010). Historically, it was thought that a high level of physical activity could help to explain the low prevalence of chronic diseases in most of Africa. However, the amount of physical activity has been decreasing as a result of the continent's rapid urbanisation (Gersh *et al*., 2010). There have been few studies published on the physical activity patterns of African populations.

- **High salt intake**:

A high Na (salt) intake is common in Africa, owing primarily to the use of salt to preserve or flavour food (Cappuccio *et al*., 2006). In addition, because processed food is uncommon, consumers add salt to already prepared foods. Reduced salt intake not only lowers BP and related CVD risk but also has other beneficial cardiovascular effects that are independent of and additive to its BP-lowering effect (van de Vijver *et al*., 2014). It

has been shown to reduce stroke, left ventricular hypertrophy, aortic stiffness, chronic kidney disease, and proteinuria (Beaglehole *et al*., 2011). As a result, it is reasonable to conclude that reducing salt intake may have a greater overall impact on cardiovascular outcomes than BP reduction alone.

- **Insufficient fruit and vegetable consumption**:

Consumption of fruits and vegetables is one component of a healthy diet that varies greatly across countries, reflecting economic, cultural, and agricultural production environments. The majority of the benefits of fruits and vegetables come from lowering CVD risk factors, specifically HT. Aside from a high salt intake, many Africans eat insufficient fruits and vegetables, resulting in a low K intake. In some patients, this is associated with higher BP; a K intake of 90 mmol/day is recommended (Popkin, 2006).

- **Obesity**:

Insulin resistance, sodium retention, increased sympathetic nervous system activity, activation of renin-angiotensin-aldosterone system, and altered vascular function are some of the hypothesised mechanisms of obesity-related HT (Kotchen, 2010). Renal sodium retention and compromised pressure natriuresis are also linked to obesity-related hypertension (Hall, 2003). Obesity rates in South Africans have increased by 2% in the last decade (2003–2012), from 9 to 11% in men and 13% in women, from 27 to 39% (Shisana *et al*., 2014). However, Egypt and Libya have a higher prevalence of obesity than South Africa in Africa according to World Population Review (2023). This prominent risk factor cannot be overlooked in terms of its contribution to South Africa's HT problem (Swanepoela *et al*., 2014). BMI was identified as the strongest predictor of HT in the large Nurses' Health Study II (Forman *et al*., 2009), with a linear relationship between adiposity and BP (correcting for age and body-fat distribution). Similar correlations with BP were found in a South African study (Schutte *et al*., 2003).

- **Urbanisation**:

Another significant risk factor for the South African population is urbanisation, which has a direct relationship with BP (Steyn *et al*., 2008). The authors of this study also found that

people who lived in urban areas had higher rates of HT than those who lived in rural areas. Overall, urbanisation (caused by demographic and epidemiological change) has an impact on food consumption patterns and has been linked to a diet high in animal products and fats. This then raises BMI, a factor that is a reliable predictor of HT and consequently contributes to HT indirectly (Singh *et al*., 2017).

2.2.5.2. Non-modifiable risk factors associated with hypertension

Harding et al. (2006) discovered a stronger relationship between BP and environmental factors, or an interaction of environmental and genetic factors rather than genetic factors alone. High BP is a treatable risk factor for the most common causes of morbidity and mortality in older people, including stroke, ischaemic heart disease, renal insufficiency, and dementia (Steyn *et al*., 2005; Ferri *et al*., 2011). Lloyd-Sherlock et al. (2014) recently reported a high prevalence of HT among older adults in South Africa and Ghana. Herein below are some examples of non-modifiable risk factors:

- **Gender differences**

In HT experimental models, it was further revealed that men develop HT earlier and more severely than women. Although the effects of gonadal hormones on the arterial, neural, and renal mechanisms that control BP are thought to be contributing factors, the reasons for sex differences in HT remain unknown (Ruixing *et al*., 2008). Gender differences in HT have been seen in both human and animal populations, and these differences are related to both biological and behavioural variables (Sandberg and Ji, 2012). Sex hormones, chromosomal variations, and other biological sex differences that are protective against HT in women are among the biological variables. These biological determinants emerge from youth and continue into adulthood until women approach menopause, when gender differences in HT become proportionally less or non-existent (Everett *et al*., 2015).

- **Genetics**

BP heritability is believed to be 30-50%. Genome-Wide Association Studies were used to identify genetic variations that impact blood pressure levels (GWAS). This strategy is

based on the "common disease-common variant" theory, which has resulted in the identification of many genetic variations that explain just 2-3% of the genetic diversity of hypertension. Part of the missing genetic information might be due to variations that are too rare for GWAS to identify. Exome chips and Next-Generation Sequencing aided in the identification of causal variations (Russo *et al*., 2018).

- **Age**

Clinical studies show a link between ageing and higher blood pressure, with increasing age being a primary non-modifiable risk factor in the development of HT. This is attributed, in part, to vasculature alterations such as endothelial dysfunction, vascular remodelling and increased vascular stiffness. These functional and anatomical alterations constitute the 'vascular phenotype' of HT, which also occurs with age. Endothelial cell damage, increased vascular smooth muscle cell proliferation, cell migration, contraction, extracellular matrix deposition, fibrosis, and calcification are all observed at the cellular level (Harvey *et al*., 2015).

2.2.6. Pathogenesis of hypertension

There are numerous pathophysiologic causes of essential HT. The amount of blood the heart pumps (cardiac output) and the resistance the blood faces in the arterioles (peripheral resistance) both affect BP as shown in Figure 2.2.7.1 (Rolfes *et al*., 2014). Increases in blood volume or heart rate increase cardiac output, and peripheral resistance is primarily influenced by blood viscosity and arteriole diameter. Therefore, the nervous system affects BP by regulating heart muscle contractions, arteriole diameters, and hormonal signals that may result in fluid retention or blood vessel constriction (Guyton and Hall, 2016). The control of the hormones involved in vasoconstriction and the retention of sodium and water by the kidneys also helps to control BP (Rolfes *et al*., 2014).

In addition, in most cases of established primary HT, an increase in peripheral resistance accounts for high BP while cardiac output remains normal (Guyton and Hall, 2016). Many theories have been postulated to explain the increase in peripheral resistance associated with HT. The main cause of increased peripheral resistance is suggested to be a structural

narrowing of small arteries and arterioles (caused by lifestyle factors) (Zieman *et al*., 2005), whereas most evidence suggests either disturbances in renal salt and water handling (particularly abnormalities in the intrarenal renin-angiotensin system as shown in Figure 2.2.7.1) and/or abnormalities of the sympathetic nervous system (Guyton and Hall, 2016). These mechanisms are not mutually exclusive, and both are likely to play a role in most cases of essential HT. Endothelial dysfunction and vascular inflammation may potentially contribute to the increased peripheral resistance and vascular damage in primary HT (Marchesi *et al*., 2008; Versari *et al*., 2009).

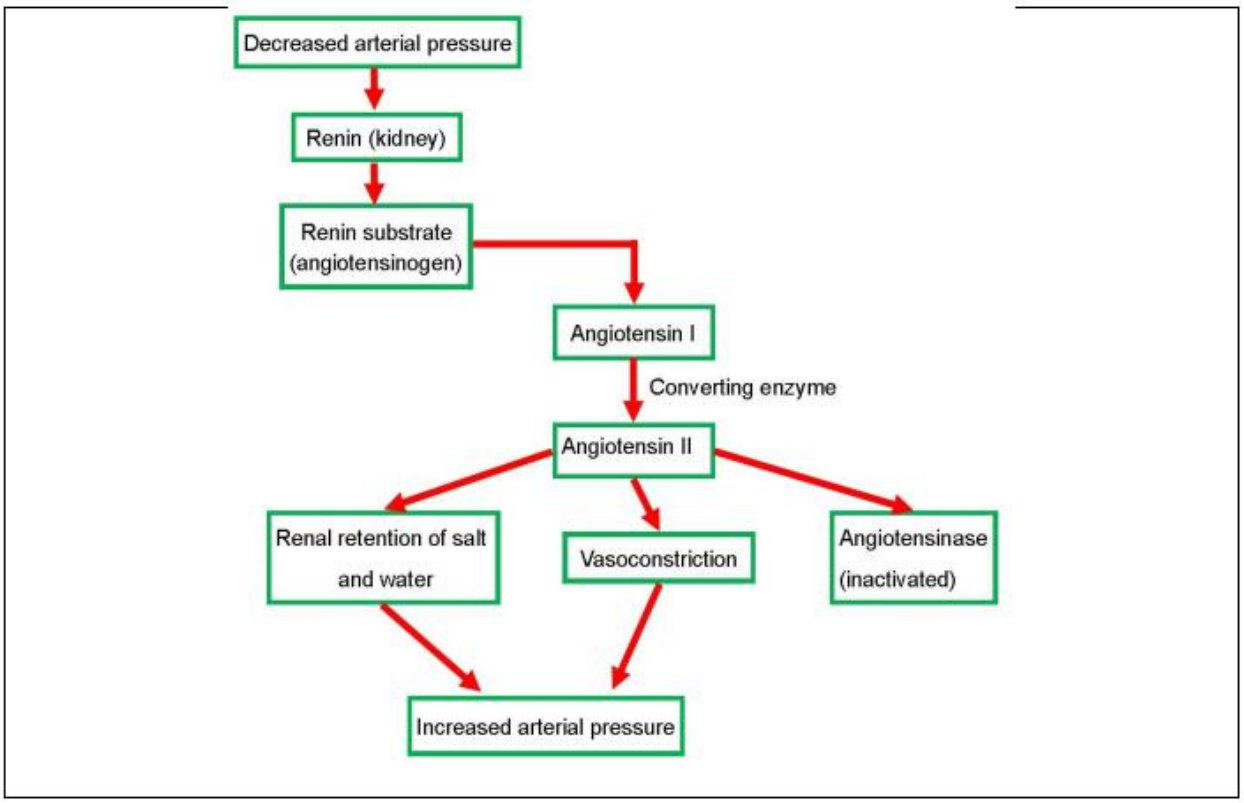

Figure 2.2.6.1: The classic renin-angiotensin vasoconstrictor mechanism for renal retention of sodium and water (Guyton and Hall, 2016).

2.3. ABDOMINAL OBESITY

Obesity is a complex multifactorial disease. The worldwide prevalence of overweight and obesity has doubled since 1980 to an extent that nearly a third of the world's population

is now classified as overweight or obese (Chooi *et al*., 2019). Obesity rates have increased in all ages and both sexes irrespective of geographical locality, ethnicity or socioeconomic status, although the prevalence of obesity is generally greater in older persons and women. This trend was similar across regions and countries, although absolute prevalence rates of overweight and obesity varied widely. For some developed countries, the prevalence rates of obesity seem to have levelled off during the past few years (Chooi *et al*., 2019). Obesity has become a global health issue (Gregg and Shaw, 2017), posing a significant burden on both the health-care system and individuals today (Murray *et al*., 2012; Yang *et al*., 2013; Zhao *et al*., 2008). Obesity, particularly abdominal obesity, is linked to metabolic syndrome (MetS) and CVDs, and it is also a risk factor for all-cause mortality not only in adults but in children (Kivimaki *et al*., 2017). According to the WHO, central obesity is a disorder characterised by an excess of abdominal fat or central fat. Central obesity is defined as a male with an abdomen circumference greater than 90 cm or a female with an abdominal circumference greater than 80 cm (WHO, 2018). In addition, rising evidence suggests that abdominal obesity is relatively frequent in children and is increasing globally (Xi *et al*., 2014), implying an increase in the cardiometabolic risk of the paediatric population. Previous research has demonstrated that childhood obesity and obesity-related comorbidities increase the risk of CVDs (Morrison *et al*., 2007) and diabetes (Morrison *et al*., 2008) in adulthood. Obesity and obesity-related comorbidities require early detection and action for both primary and secondary prevention. BMI and WC are useful markers for detecting childhood obesity. They are not, however, excellent instruments for mass screening since they are age-dependent indices with age-specific diagnostic standards. Cole et al. (2000) and Ma et al. (2010). These age-specific cut-offs will make mass screening less feasible and are not acceptable for self-regulation by non-professionals. WHtR is age-independent and proven to be effective. It may be appropriate for rapid and widespread screening for obesity and MetS, particularly in children and adolescents (Ashwell and Hsieh, 2005; Browning *et al*., 2010). The ratio of WC to height, WHtR, can be thought of as a synchronised adjusted index for WC. Although WC is closely connected to abdominal fat deposits (Balkau *et al*., 2007; Janssen *et al*., 2004), its accuracy in evaluations of relatively tall and relatively short individuals with identical WC is reasonable to doubt.

Recent research found that those who were shorter had a higher risk of MetS and other related diseases than those who were taller. These findings indicated that WC reduction was feasible and should be considered in obesity examinations (Hsieh and Yoshinaga, 1999; Schneider *et al*., 2011).

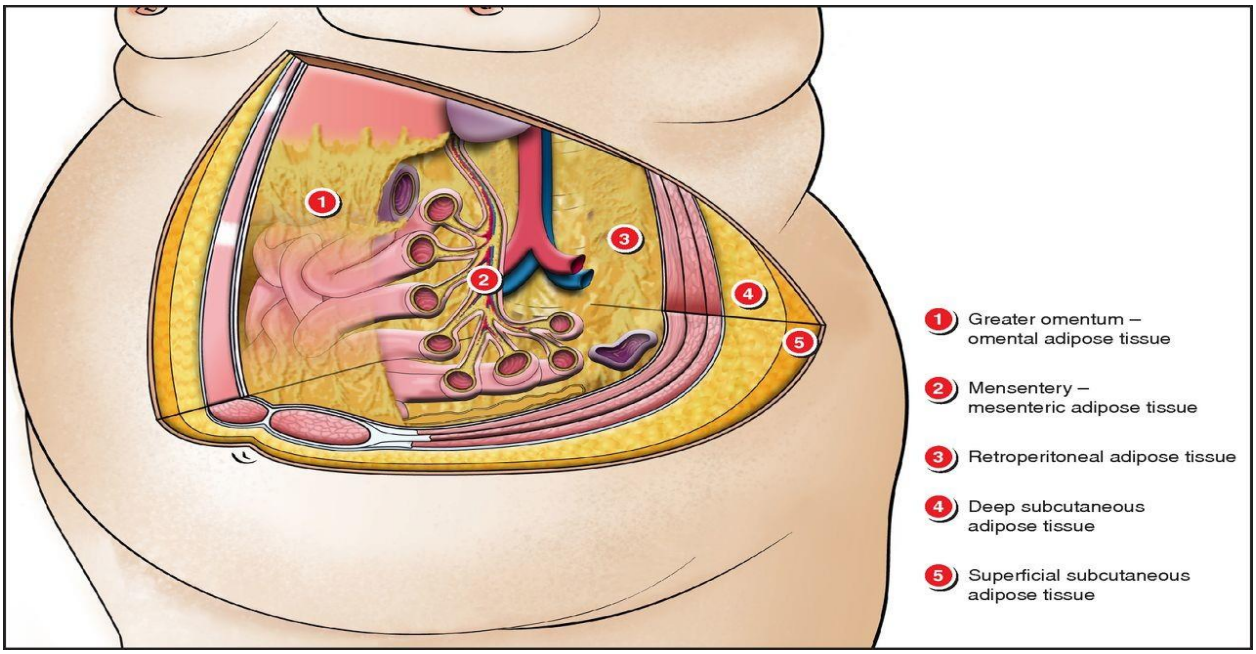

Figure 2.3.1: Representation of the anatomical localization of the main abdominal adipose tissue depots (Tchernof and Després, 2013).

Figure 2.3.2: Example of an a person classified with abdominal obesity (elcaminowomen.com)

2.3.1. Global view of abdominal obesity

Globally, abdominal obesity is increasing at an alarming rate. The rates have reached similarly high levels regardless of whether the country is poor, middle, or high-income (Olinto *et al*., 2017). Spain, Canada, and Germany, for example, have prevalence rates of around 35%, with the United States leading the high-income countries with a worrisome 56%. In low-to-middle-income countries, China and Brazil have rates similar to the three high-income countries, with Mexico leading the pack at a startling 74% (Olinto *et al*., 2017). Another study found that in the United States of America, men are up 46.4% while women are up 65.4% (Beltrán-Sánchez *et al*., 2010). However, some of the Pacific islands countries are reported to have the highest obese prevalence, with islands such as Nauru, Cook Island and Palau having an obesity rate above 55% (World Review Population, 2023).

2.3.2. Abdominal obesity in African and South African region

Obesity has become a global problem, particularly in African countries. While central obesity and overweight have been recorded in numerous nations, there is relatively little evidence on the prevalence of these health issues across most of Africa (Omar *et al*.,

2020). The high incidence of overweight/obesity in adolescents in most communities is coupled by persisting loads of underweight, showing the double burden of malnutrition produced by dietary transition (Kimani-Murage, 2013; Tzioumis and Adair, 2014; Ford *et al*., 2017; Modjadji and Madiba, 2019). A nutrition transition is characterized as a change from conventional whole-food diets to an energy-dense and nutrient-poor diet (Ford *et al*., 2017). According to WHO, 15% of African adolescents are overweight or obese. This is concerning because adolescents are the world's largest population group (1.2 billion), with 90% living in LMICs (Sheen *et al*., 2017). We recently studied trends of overweight/obesity and central obesity in children and adolescents aged 1-20 years in Agincourt, rural South Africa. The prevalence of combined overweight and obesity was considerable in teenage females, peaking at 25% at age 18 years, and central obesity was also prevalent in adolescent females, increasing with pubertal development and peaking at 35% in females (Kimani-Murage *et al*., 2010). Furthermore, a study conducted in South Africa by Debeila et al. (2021) found that 35% of adolescents were overweight/obese, with 25% having abdominal obesity by waist-to-hip ratio (WHR), 21% by WHtR, and 9% by WC. The gender distribution of abdominal obesity varies by nations, but a common tendency suggests that abdominal obesity is more widespread in women than in men—the most pronounced discrepancies in rates are observed in Nigeria, where 39.2% of women and just 3.2% of males are abdominally obese (Olinto *et al*., 2017). According to the Omar et al. (2020) study, central obesity affects 67.8% of young adults. It should be highlighted that the vast majority of them were women. Furthermore, a higher frequency was found in the following countries: Tanzania (Munyogwa and Mtumwa, 2018), Uganda (Kabwama *et al*., 2018), and West Africa (Malik *et al*., 2019).

2.3.3. Risk factors for abdominal obesity

Central obesity is caused by a variety of factors. These include genetic, socio-economic, behavioural, and environmental factors (Olinto *et al*., 2017). Among the aforementioned factors, it has been proposed that the shift toward a western influence dietary pattern and excessive sedentariness are primarily responsible for the recent rise in the global pandemic of obesity (Popkin, 2006; Popkin *et al*., 2012). As for abdominal adiposity, breakfast skipping and regular consumption of western influence fast food, energy-dense

foods, high in salt (Na) and high-calorie beverages (e.g. pastries, sweets, confectionery, salty snacks, processed meat products, sugar-sweetened soft drinks, etc.) have been positively associated with the likelihood of central obesity in children and adolescents, whereas beneficial dietary behaviours (including excessive consumption of dairy products, fruits, and vegetables) have been negatively associated with the likelihood of central obesity (Grigorakis *et al*., 2016). Common (multifactorial) obesity, most likely caused by a coordinated interaction of genetic, epigenetic, and environmental variables, is clearly connected to genetic predisposition by several risk variants, which account for just a modest portion of overall BMI variability (Rohde *et al*., 2019). Among them, the association and causative role played by gut bacteria in obesity represents one of astonishing findings over the past decade (Tseng and Wu, 2019). Although GWAS has opened up new possibilities for unravelling the complex genetics driving common obesity, understanding the biological pathways related to the individual risk factors leading to obesity remains a challenge. Non-genetic variables such as eating habits and physical exercise significantly influence an individual's likelihood of acquiring obesity. Through epigenetic pathways, these variables may interact with a genetic propensity to obesity (Rohde *et al*., 2019).

2.4. THE RELATIONSHIP BETWEEN NUTRITIONAL FACTORS AND HYPERTENSION AND ABDOMINAL OBESITY

Although multiple factors can influence the development of HT and abdominal obesity. Dietary intake has been identified as one of the important risk factors for NCDs. High Na and low K intakes are associated with the early development of chronic diseases (e.g., HT, obesity) (Golpour-Hamedani, 2022). Several mechanisms exist to indicate how Na and K can influence BP. Evidence indicates that an interaction between these micro-nutrients plays a dominant role in the development of primary HT (Adrogué and Madias, 2007; Perez and Chang, 2014). Diets specifically characterised by modern western diet-which are high Na and low K produce a biologic interaction with the kidneys, which results in extreme levels of Na and inadequate concentrations of K in the human body (Adrogué and Madias, 2007; Perez and Chang, 2014). Dietary habits that are formed from childhood are presented in adulthood, children who consume excessive amounts of Na

and little K tend to keep their levels at those levels over time (Aburto *et al*., 2013). In addition, Data on Na and K intake in the African region is insufficient in both children and young adults. Because dietary patterns are frequently regarded as one of the primary causes of NCDs, it is critical to describe the expected nutritional transition to quantify the impact of diet on this group of diseases. However, due to logistical and financial constraints, this work is scarce or inadequately documented in developing countries. Another reason for the scarcity of this work, according to Pisa et al. (2014), is the lack of reliable dietary assessment methodologies, which supports the emerging need for the development, validation, and standardisation of tools for measuring and monitoring food intake in different countries (Pisa *et al*., 2014).

2.4.1. Sodium intake

There is less data on Na intake in children and young people than in adults, and these are mostly limited to the developed countries of Europe and North America. One reason could be the significant methodological challenges in obtaining complete and valid dietary data for children (Livingstone, Robson and Wallace, 2004). The chemical element sodium has the symbol Na and the atomic number 11. People have revered salt throughout history. People we admire are referred to as "the salt of the earth," while those we dislike are referred to as "not worth their salt." The fact that one taste quality (saltiness) is dedicated to identifying Na-containing foods demonstrates the importance of Na (Mattes, 1997). Na is also an essential mineral in humans, where it regulates blood volume, BP, and pH (Rolfes *et al*., 2014). Today, processed foods and restaurant foods account for at least 75% of sodium intake in the industrialised diet (Dötsch *et al*., 2009). The population of South Africa has only been the subject of a small number of studies, but it has been determined that the average South African consumes 6–11 g of salt daily. The studies that served as the foundation for this estimation are summarised elsewhere (Wentzel-Viljoen *et al*., 2013), and the information is somewhat stale. For monitoring and other purposes, South Africa needs more recent data on sodium intake, looking at the trend from childhood to adulthood.

2.4.1.1. The importance of sodium in the body and food

- **Body**:

The main cation in the extracellular fluid is Na. It has many important and diverse roles in mammalian physiology, such as maintaining extracellular fluid volume, and water balance, and generating cell membrane potential. The extracellular fluid contains 95% of the body's total Na content. The majority of dietary Na is consumed in the form of common salt [sodium chloride (NaCl)] (Elliott, P. and Brown, 2007).

- **Food**:

Na in the form of sodium chloride which is known as salt is used to improve flavour and palatability in a variety of foods (Dötsch *et al*., 2009). Na offers two crucial tasks beyond flavour: processing and preservation. The salt added to the product reduces water activity and inhibited microbiological development, making it the earliest application of Na (in the form of salt) (Kilcast and Angus, 2007). It also includes a particular processing function for different food groups. In most bread items, salt modulates gluten production and fermentation, while salt boosts the water-binding capacity of proteins in meat products (Desmond, 2006, Taormina, 2010). Another technical aspect of salt is that it influences the activity of microorganisms and enzymes necessary for cheese maturing (Kilcast and Angus, 2007). As a result, preserving microbiological stability and structural integrity is crucial. However, there are various techniques that businesses may employ to lower Na while retaining the features listed above. Adaptations (gradual sodium reduction), flavours (multisensory principles), salt replacements (usage of mineral salts), and salt boosters are among them (Dötsch *et al*., 2009).

2.4.1.2. Association between sodium intake and BP (hypertension)

A number of adults who consume high Na over time are more likely to develop high BP (Meneton *et al*., 2005; He *et al*., 2013). There is insufficient data and ambiguous conclusions in children and adolescents; some studies have indicated a favourable link between Na intake and BP in adolescents (Maldonado-Martin *et al*., 2002; Yang *et al*., 2012; Rosner *et al*., 2013; Shi *et al*., 2014), while others have not (Palacios *et al*., 2004;

Qunaissa *et al*., 2015). Meta-analyses of experimental trials have revealed that lowering Na consumption in children helps lower BP 27,28 (Aburto *et al*., 2013; Leyvraz *et al*., 2016). Furthermore, He et al, (2013) conducted a follow-up meta-analysis to investigate the effects of a longer-term mild salt decrease. They claimed that the two meta-analyses done by Graudal et al. (2012) looked at papers from short-term trials with a considerable change in salt intake, which is not relevant to the present public health situation. He et al. (2013) reported that a longer-term step-wise reduction in salt intake resulted in large and significant reductions in BP while having no deleterious impact on plasma hormones.

The pathophysiological relationship between Na intake and an increase in BP has long been questioned. Increased salt consumption may cause water retention, resulting in increased flow in arterial arteries. Pressure natriuresis has been proposed as a physiologic phenomenon in which a rise in BP in the renal arteries produces an increase in salt and water excretion (Girardin *et al*., 1980). According to animal model studies, this hemodynamic strain may cause unfavourable microvascular remodelling due to the consequences of elevated BP levels (Dajnowiec *et al*., 2007; Dumont *et al*., 2008). Changes in vascular resistance are associated with high Na consumption and higher BP levels, although the mechanisms governing this phenomenon may not be considered solely as a reflex pressor reaction aimed at boosting Na excretion. Even in normotensive patients, high Na intake can cause microvascular endothelial inflammation, structural remodelling, and functional problems (Marketou *et al*., 2019). Recent research has revealed that variations in Na plasma levels affect not just small resistance arteries but also the function and structure of large elastic arteries. The problem of salt-sensitivity, which refers to individual susceptibility to BP variations as a result of changes in dietary salt intake, has also lately been addressed in terms of its pathophysiological basis and clinical implications (Elijovich *et al*., 2016; Kurtz *et al*., 2017).

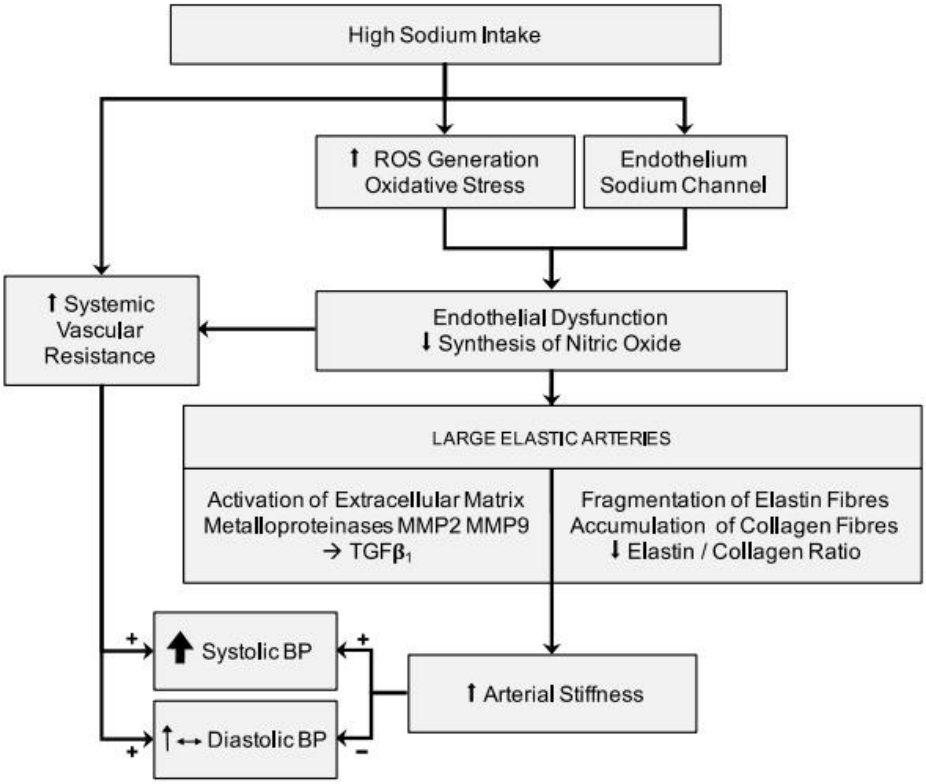

Figure 2.4.1.2.1: Relationship between high salt intake with diet, BP, and arterial stiffness. BP; MMP, matrix metalloproteinases; ROS, reactive oxygen species; TGF, transforming growth factor (Grillo *et al*., 2019).

2.4.1.3. Association between sodium intake and abdominal obesity

Despite the well-known detrimental effects of high Na consumption on HT, obesity has recently gained popularity as another potential medical consequence (He *et al*., 2013). Several recent studies have suggested that dietary Na intake may be linked to weight gain. Na intake has been linked to a variety of adiposity outcomes in both children (Yoo and Oh, 2007; Libuda *et al*., 2012; Zhu *et al*., 2014) and adults (Yoo and Oh, 2007; Larsen *et al*., 2013; Yi and Kansgra, 2014), including BMI or, in the case of children, BMI z-score, weight category, per cent body fat, and abdominal obesity. Cross-sectional studies in children from the United Kingdom, the United States, and Australia found that dietary sodium intake is positively associated with the consumption of energy-rich sugar-sweetened beverages (He *et al*., 2008; Grimes *et al*., 2013). This relationship may be due

to Na's effects on thirst, as experimental studies in both animals and humans show increased fluid intake on a higher Na diet (He *et al*., 2001; Stricker *et al*., 2003).

Because foods high in Na are often also high in energy, reported associations between Na intake and adiposity outcomes may be confounded by energy intake. However, energy intake may act as a moderator in the causal pathway between Na intake and obesity. Adding NaCl (salt) to many foods improves their palatability and encourages greater energy intake (Bolhuis *et al*., 2012). Furthermore, it has been proposed that salt may act as a vehicle that promotes the consumption of dietary fat. This is supported by studies that show a preference for salty and fatty foods is associated with higher total daily energy intakes in adults (Mejean *et al*., 2014), uncontrolled eating (Keskitalo *et al*., 2008), and childhood obesity (Maffeis *et al*., 2008).

2.4.2. Potassium intake

One of the primary electrolytes, or blood minerals, that are necessary for both cellular and electrical function is K. More than 90% of the body's total K stores are found in the cells, which are also the main source of positive ions, or cations, including K. Together with Na, K controls the water balance, and acid-base balance, and plays a crucial part in the transmission of electrical impulses in the heart (Podrid, 1990). The function of the heart and nerves depends on the active transport of K into and out of the cells. K causes an exchange of Na and K across the cell membrane as it enters the cell. As a result, the electrical potential necessary for the transmission of nerve impulses is created within the nerve cells (Podrid, 1990). Once K has left the cell, it restores cell repolarisation, allowing the nerve impulse to proceed. This electrical potential gradient aids in the generation of muscle contractions and the regulation of the heartbeat. Serum K levels should be between 3.6 and 5.0 mmol/L. Even a 1% (35 mmol) decrease in total body K causes a significant imbalance in intracellular and extracellular K. This imbalance changes the electrophysiologic characteristics of the cell membrane, which harms impulse generation and conduction throughout the heart (Podrid, 1990).

2.4.2.1. Association between potassium intake and blood pressure (hypertension)

Until recently, humans ate a K-rich diet. However, with the increased consumption of processed foods that have K removed, combined with a decrease in the consumption of fruits and vegetables, there has been a significant decrease in K intake, which now averages around 70 mmol/day in most developed countries, i.e. only one-third of our evolutionary intake (He and MacGregor, 2008). A large body of evidence suggests that increasing K intake is beneficial to human health. Both epidemiological and clinical studies show that a high-potassium diet lowers BP in people with high BP as well as people with normal BP (He and MacGregor, 2008).

K appears to be an important regulator of BP in epidemiologic, experimental, and clinical studies. Furthermore, similar epidemiologic findings in animal and human research indicated that a shortage of K amplifies the deleterious impact of high Na consumption on the development of HT and other CVDs (Adrogué and Madias, 2007, Adrogué & Madias, 2014; He and MacGregor, 2001; He and MacGregor, 2008). K intakes in populations are often low due to excessive intake of processed foods (He and MacGregor, 2008), as validated in South African research where K levels were shown to differ amongst different demographic groups and only a small fraction of individuals reached the optimum dietary intake (Charlton *et al*., 2005).

2.4.2.2 Association between potassium intake and abdominal obesity

The link between K intake and obesity or MetS has recently been called into question. As a result, a systematic review was conducted to describe the relationship between serum K, Na/K ratio, and obesity/MetS. Murakami et al. (2015) discovered that high K levels measured from 24 hour urinary excretion were also linked to obesity, whereas Shin et al. (2013) and Lee et al. (2013) discovered that K intake did not affect obesity. Nonetheless, the findings of Ge et al. (2015) did not show a significant relationship between 24 h urinary K excretion and MetS. Shin et al. (2013) demonstrated that high K intake was a protective factor in both sexes, according to MetS. However, the protective effect of high K intake on obesity may be due to a high intake of fruits and vegetables, which are high in K and have also been shown to be beneficial to MetS (Esmaillzadeh *et al*., 2006; Murakami *et*

al., 2012). The precise mechanism underlying the link between K intake and obesity or MetS is unknown. Central Obesity is a component of MetS, and the mechanisms behind obesity and MetS are homogeneous. Obesity has been linked to decreased K channel function (Crunkhorn *et al*., 2013; Climent *et al*., 2014). This finding suggests that the link between K and obesity/MetS is a relatively recent topic for nutrition and public health. This new research, however, was limited to Asia and the United States and had small-scale constraints (Cai *et al*., 2016).

2.4.3. Sodium-to-potassium ratio

2.4.3.1. Association between sodium-to-potassium ratio and blood pressure (hypertension)

According to pathogenetic studies, Na and K dependencies influence BP (Adrogué and Madias, 2007). Epidemiological research has demonstrated that high K counteracts the negative effects of high Na on BP levels, reducing the risk of CVD (Aburto *et al*., 2013; Aburto *et al*., 2013; He *et al*., 2013; Gay *et al*., 2016). The evidence points to the interaction between Na and K as the primary factor in the development of HT (Adrogué and Madias, 2007). Biological interaction between the modern diet's high Na and low K content and the kidneys results in the body having too much Na and not enough K (Adrogué and Madias, 2014). Following this, the peripheral resistance will rise and the smooth muscle cells in the arteries will contract, ultimately leading to high BP (Adrogué and Madias, 2014). According to some research (Grimes *et al*., 2011; Mozaffarian *et al*., 2011), increasing the consumption of K-rich foods can lower the dietary Na/K ratio, which may have a greater positive effect on public health. It has also been suggested to substitute the suggested Na/K ratios for the absolute K and Na intake levels (Meneton *et al*., 2009; Yang *et al*., 2011). The findings reported in the 2001 Dietary Approaches to Stop Hypertension (DASH) diet study provide additional support that the Na/K ratio is more strongly associated with BP outcomes than either nutrient alone among prehypertensive and hypertensive participants combined (Sacks *et al*., 2001).

2.4.3.2. Association between sodium-to-potassium ratio and abdominal obesity

A few epidemiological studies have also identified a correlation between adiposity measurements and high Na including low K intake (Ge and Zhang, 2016; Jain *et al*., 2014). The problem is that, in conditions like high BP, the interaction between high dietary Na and low K intake plays a role in the pathogenesis of HT in a way that makes it appear as though the combined effect of high Na and low K diets on BP is greater than the combined effect of each of these factors alone. The urinary Na/K ratio was found to be associated with obesity (Binia *et al*., 2015). However, less is known about the combined impact of dietary Na and K intake in the pathogenesis of other conditions, such as obesity and overweight, particularly in children (O'Donnell *et al*., 2011). There varies ways to estimate dietary intake the following Section provided the detail of different dietary measurements.

2.4.4. Methods used to estimate dietary intake

Dietary information is effective in predicting the risk of cardiovascular disease (Baik *et al*., 2013), and eating a nutrient-dense diet has been linked to a lower risk of all-cause mortality (Streppel *et al*., 2014). In contrast to other lifestyle risk factors (such as smoking), dietary exposures are extremely difficult to quantify because all individuals consume meals, even if the amount and kind of food ingested vary between participants, and people seldom notice what they eat and how much they consume (Willet, 2012). Inadequate dietary assessment may be a significant barrier to understanding the influence of dietary variables on illness.

Table 2.4.4.1: Methodological techniques used to estimate Na and K intake

	Methods	Collected date	Strengths	Limitations
Duplicate diet approach (Shim *et al*., 2014)	Collection and direct examination of duplicate diet samples	Actual intake data over a specific time period	Dietary exposures can be measured (e.g. environmental contaminants)	Large-scale research are not suitable
Food consumption record (Shim *et al*., 2014)	At the household level, trained personnel conduct objective observations	Actual intake data over a specific time period	Easy to use for those with low literacy or who prepare the majority of their meals at home	Individual dietary consumption is inaccurate; not ideal for folks who eat out regularly
Food frequency questionnaire (FFQs) (Shim *et al*., 2014)	Subjective assessment using a predefined, self-administered or interviewer-administered format	Estimates of normal intake over a relatively long time (e.g., 6 months or 1 year)	Assesses typical dietary intake quickly; is cost-effective and time-saving; and is appropriate for epidemiological studies	Specific to study groups and research objectives; use a closed-ended questionnaire; has low accuracy (recall bias); and necessitates accurate evaluation of generated questionnaires
24-hour dietary intake (Shim *et al*., 2014)	A trained interviewer administers open-ended questionnaires as a subjective measure	Actual intake data for the previous 24 hours	Provides detailed intake data; has a low respondent burden (literacy not required)	Potential recall bias; trained interviewer necessary; potential interviewer bias; expensive and time-consuming; numerous days required to assess typical intake; potential dietary changes if repeated measures
Dietary record (Shim *et al*., 2014)	Subjective assessment using self-administered open-ended questionnaires	Actual intake data over a specific time period	Provides detailed intake data; no need for an interviewer; no recall bias	Relatively high respondent burden (literacy and strong motivation required, potential under-reporting); costly and time-consuming; numerous

				days necessary to assess typical intake; potential dietary changes if with repeated measurements
Dietary history (Shim *et al*., 2014)	Subjective measures are taken with open-ended and closed-ended questionnaires administered by a trained interviewer	Estimates of normal intake over a lengthy period of time	Examines typical dietary intake	High expense and time commitment; not appropriate for epidemiological research
Biomarkers (Shin *et* al., 2014; Corella and Ordovás, 2015)	Urine, plasma or serum, teeth, hair, and nail samples are collected for measurements	Dietary data is gathered over hours, days, weeks, months, and years	Free of bias and mistakes; memory independent; No need to specify the type and quantity of food taken by the patient; can be used to substitute dietary intake technique mistakes	It cannot be used alone to collect dietary data because it is affected by absorption and metabolism after eating. It is also affected by homeostasis and illnesses; Inter-individual variables such as age, gender, alcohol use, cigarette use, and physical activity; Storage and collecting of the obtained specimen ; cannot be used to offer dietary recommendations; cannot be utilized to change the food habits of subjects

2.5. DIETARY STRATEGIES TO TREAT AND PREVENT HYPERTENSION OR ABDOMINAL OBESITY

Since the United Nations General Assembly decided in 2011 that NCDs prevention and control should be a priority, member nations have approved the global goal of decreasing salt intake by 30% by 2025, to lower Na intake by less than 2 g per day (WHO, 2013). In 2010, 32 nations implemented some form of sodium reduction plan (Webster *et al*., 2010), and this figure more than quadrupled in 2014, with 75 countries presently using salt reduction programmes (Trieu *et al*., 2015). In 2010, the majority of these nations were in Europe (Webster *et al*., 2010), but by 2014, countries of all economic levels had a plan (Weber *et al*., 2014; Trieu *et al*., 2015). Food reformulation, consumer education, front-of-pack labelling, interventions in public institution settings, and taxes were the five salt reduction implementation approaches used in 75 countries. According to Trieu et al. (2015), 12 nations reported a decrease in population salt intake, up from four reported in 2010 (Webster *et al*., 2016).

In South Africa, the Na reduction strategy arose as a consequence of the national Department of Health formulating a strategic plan to improve efforts to prevent and manage NCDs (based on the recommendations of the WHO). The Department of Health's strategic plan includes goals for decreasing BP in the South African population. National goals, context-specific statistics (Charlton *et al*., 2005; Wentzel-Viljoen *et al*., 2013), and cost-effectiveness assessments inspired this rule (Bertham *et al*., 2012). The rules (R.214) in Table 2.5.1 about the reduction of Na in certain products were gazetted in March 2013 by the Minister of Health under section 15(1) of the Foodstuffs, Cosmetics, and Disinfectants Act, 1972. (Act 54 of 1972). This regulation is part of a wider Na reduction approach that includes Na intake and food supply monitoring, as well as public awareness and education efforts. Salt Watch, an awareness and education campaign on salt use, was founded in 2014 (Wentzel-Viljoen *et al*., 2013).

Table 2.5.1: Regulations to the reduction of Na in certain foodstuffs-R.214

Food category (mg)	2016 target (Na/100g)	2019 target (Na/100g)

Bread	400	380
All breakfast cereals	500	400
All fat and butter spreads	550	450
Savoury snacks, excluding salt-and-vinegar flavoured	800	700
Flavoured potato crisp, excluding salt-and vinegar	650	550
Flavoured ready-to-eat savoury snack and potato crisp, salt-and-vinegar	1000	850
Processed meat – uncured	1300	1150
Processed meat – cured	850	650
Raw-processed meat sausages	800	600
Dry soup powder (not instant type)	5500	3500
Dry gravy powders and dry instant savoury sauces	3500	1500
Dry gravy powders and dry instant noodles to be mixed with a liquid	1500	800
Stocks cubes, - powder, -granules, - emulsions, - pastes or – jellies	18000	13000

HT and obesity are the major cause of CVDs and early mortality. Because of the extensive usage of antihypertensive drugs, worldwide mean BP has stayed constant or slightly lowered during the last four decades (Mills *et al*., 2020). Despite the rising prevalence, the proportions of HT knowledge, treatment, and BP management are low, particularly in low- and middle-income countries, and there are few comprehensive analyses of the economic impact of hypertension (Mills *et al*., 2020). Several studies have revealed that certain dietary variables are linked to high BP and HT. However, there is a limitation on the dietary strategies to mitigate abdominal obesity/obesity in Africa (South Africa) as data regarding the estimation of Na and K are few and there is a need to generate

information on this (Zhao *et al*., 2011). Nevertheless, these following recommendations to treat HT obesity (central obesity) are as follows:

-Dietary factors play a crucial role in the development and progression of HT.

-One of the most important modifiable risk factors for HT is high Na consumption.

-There is strong evidence that a diet high in fruits, vegetables (i.e. increase in K), and low-fat dairy products, as well as low in salt and saturated fat, helps lower BP.

-There are significant discrepancies in the suggested types and intakes of dietary components among hypertension management and preventive guidelines produced by different nations and organisations.

-Population-based dietary interventions for HT should be strongly promoted through the formulation and implementation of national action plans.

2.6. GENERALIZED ESTIMATING EQUATION FOR LONGITUDINAL ANALYSIS

GEE is a general statistical strategy for fitting a marginal model for longitudinal or clustered data analysis that has seen widespread application in clinical trials and biological research (Feng *et al*., 2001; Hardin and Hilbe, 2003; Fitzmaurice *et al*., 2004). There are various basic methods for analysing repeated data, such as ANOVA and MANOVA for repeated measurements. However, variables cannot be incorporated. There are two approaches: mixed-effect models, which are extensively used in practice, and GEE (Crowder, 1995). It is worth mentioning that these two methodologies have various tendencies in model fitting based on the study objectives. The mixed-effect model, for instance, is a person-level method that uses random effects to represent the relationship between observations of the same subject (Crowder, 1995). GEE, on the other hand, is a population-level technique based on a quasi-likelihood function that yields population-averaged parameter estimates. GEE, as is widely known, has various distinguishing characteristics (Diggle *et al*., 2002; Hedeker *et al*., 2006; Fitzmaurice *et al*., 2008), which are as follows:

- Because the variance-covariance matrix of responses is handled as a nuisance parameter in GEE, this model fitting is easier than mixed-effect models. GEE is favoured, in particular, if the total therapeutic outcome is of major concern.
- Even when the "working" correlation structure of responses are mis-specified, the parameter estimates are consistent and asymptotically normally distributed under mild regularity criteria, and the variance-covariance matrix can be calculated thru a resilient "sandwich" variance estimator.
- GEE loosens the distribution assumption, simply requiring the correct specification of the marginal mean and variance, as well as the link function that connects the covariates of interest and marginal means.

2.6. SUMMARY

HT and obesity have both been on the rise in children. Both tracks into adulthood and are linked to an increased risk of cardiovascular disease, which increases the prevalence of heart disease and its associated morbidity and mortality. Dietary intake has been identified as one of the important risk factors for NCDs. High Na and low K intakes are associated with the early development of chronic diseases (e.g., HT, obesity). Several mechanisms exist to indicate how Na and K can influence BP. Diets specifically characterised by modern western diet- which are high Na and low K produce a biologic interaction with the kidneys, which results in extreme levels of Na and inadequate concentrations of K in the human body. Despite the distinguished negative health effects of high sodium intake on HT, obesity has recently gained attraction as another possible health-related outcome. Although the mechanisms underlying the association between Na and obesity have not been well established, the role of soft-drink consumption was explored, that is salt intake is indirectly related to obesity through soft-drink consumption, through its effect on fluid intake. As a food high in Na and usually high in energy intake. However, the relationship between dietary intake with HT and abdominal obesity is inconsistent. Therefore, we designed this study to investigate the effect of Na, and K intake and their ratio on HT and abdominal obesity on the same participants over time in Ellisras.

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CHAPTER 3

MATERIALS AND METHODS

3.1. Geographical area

3.2. Sampling procedure and research design

3.3. Exclusion criteria

3.4. Data collection

3.5. Quality control

3.6. Statistical analysis

3.7. References

3.1. GEOGRAPHICAL AREA

Lephalale, formerly known as Ellisras, is a deeply rural area situated in the province of Limpopo South Africa, bordered by Botswana. It is located in the north-western part of the province (23º 40S 27º 44W), with 42 settlements which are approximately 70 kilometres (Km) from Lephalale town (Monyeki *et al*., 1999). Although it is marked by unemployment and poverty. Lephalale residents are primarily employed at Matimba and Medupi electricity power stations, Exxaro mine (Grootegeluk Coal Mine), shopping centres and Lephalale Mall and with some depending on subsistence farming nurturing cattle whereas the other population is made up of educators and civil servants (Monama, 2020).

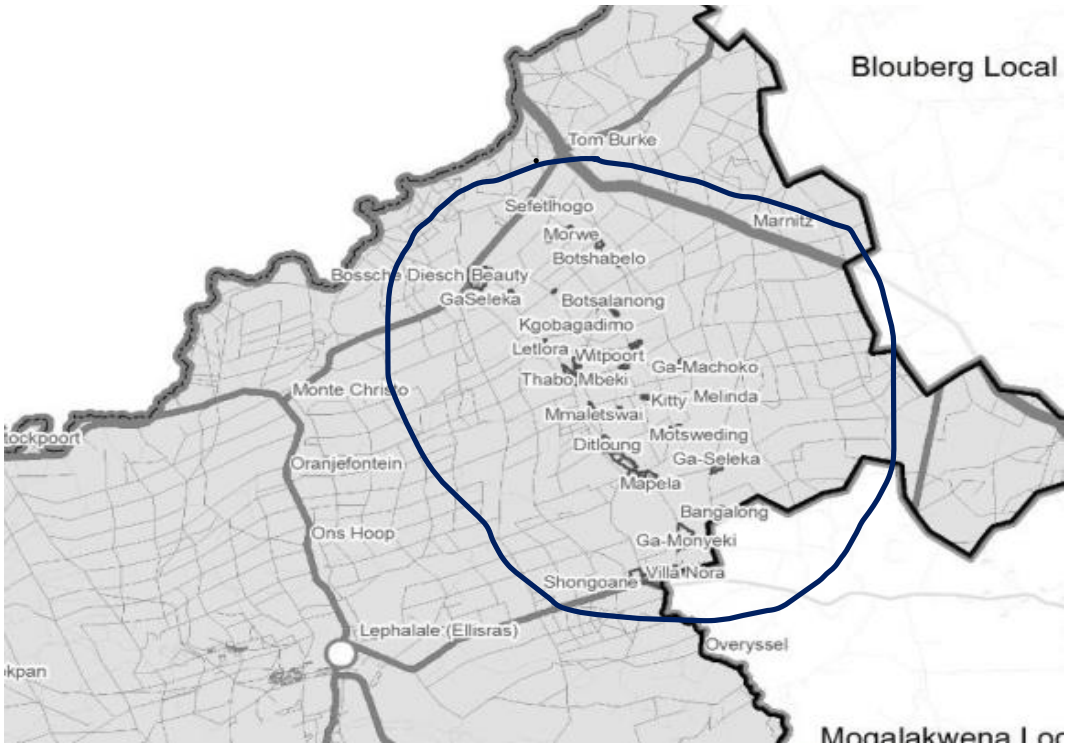


Figure 3.1.1: A part of the Lephalale municipality map showing the villages that were measured (circled)

3.2. SAMPLING PROCEDURE AND RESEARCH DESIGN

This study is part of the ongoing Ellisras Longitudinal Study (ELS), of which the details of the sampling procedure and geographical area were reported elsewhere (Monyeki *et al*., 1999). The ELS initially followed a cluster sampling method. Briefly, the study was undertaken at 22 schools (10 preschool and 12 primary schools) randomly selected from 68 schools within the Ellisras rural area (Monyeki *et al*., 1999). Baseline data were collected in 1996 from children aged 3–10 years, with follow-up exams of dietary intake, BP and anthropometric measurements in 1999, 2001, 2015 and 2018. This study constitutes of the same participants as the study of van Den Ende et al. (2014) and Mashiane et al. (2018) with a total number of 825 (aged 6 to 12; 421 males and 404 females) and 728 (aged 18 to 30; 356 males and 372 females) who were tracked for the changes in Na, K intake and their ratio. In which a total of 325 participants (161 males and 164 females) were found to have complete measurements for the three years of assessment (BP measurements and anthropometric measurements), as shown in Figure 3.2.1 from data collected in 1999, 2001 and 2015. The number of years used for this study were chosen because this is when the dietary intake was collected twice for each year. Ethical clearance was granted by the Turfloop Research Ethics Committee (TREC) of the University of Limpopo before the study was conducted, with an ethical clearance number TREC/88/2021: PG.

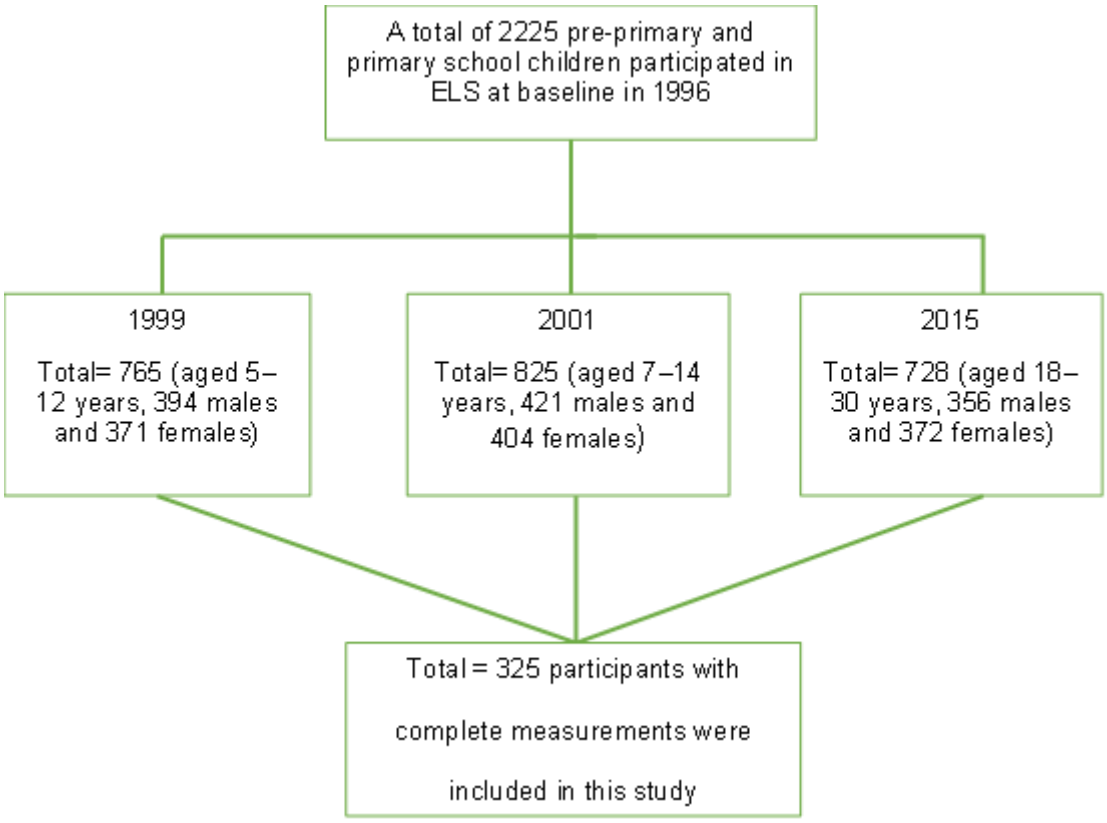


Figure 3.2.1: The flow chart of the study participants over the years

3.3. EXCLUSION CRITERIA

Any participant with the following will be excluded from this study (this does refer to the baseline data):

- Participants who have been diagnosed with HT.
- Participants who fail to provide a signed consent form before measurements.
- Participants who are taking acute or chronic medication for HT or any related CVDs.
- Participants who have incomplete measurements for the three years of assessment.

3.4. DATA COLLECTION

3.4.1. Blood pressure

BP [systolic and diastolic BP (SBP & DBP)] was measured using an Omron electronic micronta monitoring kit (Omron Healthcare Europe B.V, Hoofddorp, Netherlands) (Monyeki *et al*., 2006). The participants were measured three times at a five-minute interval, with their feet on the floor and the right arm was supported on a prop at a heart level in a relaxed position with an appropriate cuff size placed over the brachial artery of the right arm. The average of the three readings (for both SBP and DBP) was calculated and used to determine the prevalence of HT. The cut–off points for high BP or HT as described by National Heart, Lung, and Blood Institute (NHLBI) (2005) were utilised for age, sex, and height (**Table 3.4.1.1**). BP was calculated using the Merck manual-medical calculator for both boys and girls separated (Merck Manual, 2018). This method of calculation differs from the adults as it uses percentiles to calculate BP for children. However, cut-off point guidelines as described by Whelton et al. (2018) were used for adolescents aged eighteen to nineteen and young adults (**Table 3.4.1.2**).

TABLE 3.4.1.1: National Heart, Lung, and Blood Institution guidelines for the diagnosis of HT in children (NHLBI, 2005).

Classification	**Systolic and/or diastolic BP (mmHg)**

Normal BP	< 90th percentile
Prehypertension	90th to < 95th percentile
Stage 1 Hypertension	95th percentile to 99th percentile plus 5 mmHg
Stage 2 Hypertension	> 99th percentile plus 5 mmHg

TABLE 3.4.1.2: Cut-off points for young adults (Whelton *et al*., 2018).

BP Category	**SBP**		**DBP**
Normal	≤ 120 mmHg	And	≤ 80 mmHg
Pre-hypertension	120–129 mmHg	And	<80 mmHg
Hypertension Stage 1	130–139 mmHg	Or	80-89 mmHg
Hypertension Stage 2	≥140 mmHg	Or	≥90 mmHg

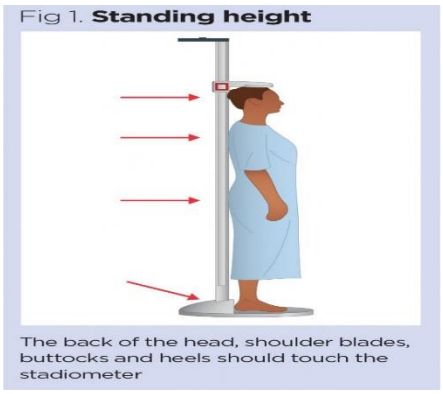
Figure 3.4.1.1: BP measurement

3.4.2. Anthropometric measurements

Anthropometric measurements (height, and WC) were measured according to standard procedures of the International Society for the Advancement of Kinanthropometry (ISAK) (Norton and Olds, 1996). The WHtR was calculated as WC divided by height in centimetres. Waist circumference was measured using a non-stretchable measuring tape to the nearest 0.1 cm and height was measured using Stadiometer to the nearest 0.1 cm. Abdominal obesity was defined as WC (≥ 90th percentile for children and adolescents, ≥ 90 cm for males and ≥ 80 cm for females in young adults) and WHtR (≥ 0.5) (Yoo, 2016).

A-WC measurement

B-Height measurement



C- WHtR measurement

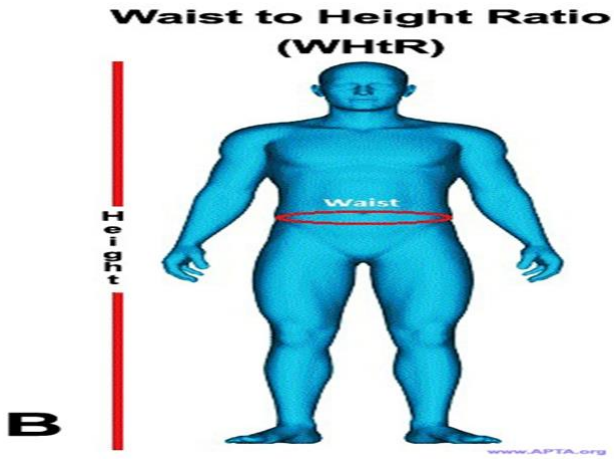


Figure 3.4.2.1: Example of anthropometric measurement (A- www.shutterstock.com; B- Nutrition and Hydration, 2020; C- epos.myesr.org)

3.4.3. Dietary intake

Data on diet (Na and K intake) were collected using a 24-hour recall questionnaire (see attachment), as it is a valid method to determine group dietary intake (Langenhoven, 1991). Trained ELS field workers who were Northern Sotho (Sepedi) speaking and interviewed participants regarding their dietary intake over the past 24 hours. Parents/caregivers of all the children participants were interviewed regarding the dietary intake over the previous 24 hours. The estimated portion size of foods consumed was recorded in as much detail as possible, using a pre-tested questionnaire and food models

simulating average portions of local foods. An average of two days of dietary intake was taken for each participant. One dietary intake was collected during the weekday and another dietary intake was collected for the weekend. This is because food consumption during the week differs from the food consumed during the weekend. People tend to eat a lot more during the weekend than during the week, especially on Saturdays (An, 2016). Dietary intake (Na and K) were analysed using local food tables/composition and South African Food Composition Database System (SAFOODS) (Wolmarans *et al*, 2009) and were compared with the recommended intakes for Na and K as described by Consensus Study Report (2019).

The food composition manuals were used to improve the accuracy of the data collection because they had pictures of actual types, portion sizes and weights of the food items consumed. This accommodated both the interviewer and even those with low literacy to correctly identify and report consumption over the previous 24-hrs (Shim *et al*., 2014). A booklet adapted from the Dietary Assessment and Education Kit was also used to obtain portion sizes (Steyn *et al*., 2006). The WHO recommends a Na/K ratio of 1:1 (or ≤1) to assess the average molar Na/K ratio (WHO, 2012). Since the recommended Na/K ratio is in moles, the Na and K intake in this study was converted from milligrams (mg) to milli-moles (mmol). The following conversion was used to calculate the average molar Na/K ratio (Department of Health and Ageing, National Health and Medical Research Council, 2006):

23 mg sodium = 1 mmol sodium;

39 mg potassium = 1 mmol potassium.

TABLE 3.4.3.1: Sodium Dietary Reference intakes by age, sex, and life-stage group (Consensus Study, 2019).

Life-stage group (years)	**Adequate intake (mg/day)**
Children	
1–3	800

4–8	1000
Males	
9–13	1200
14–18	1500
19–30	1500
31–50	1500
51–70	1500
> 70	1500
Females	
9–13	1200
14–18	1500
19–30	1500
31–50	1500
51–70	1500
> 70	1500

TABLE 3.4.3.2: Potassium Dietary Reference intake by age, sex, and life-stage group (Consensus Study, 2019).

Life-stage group (years)	**Adequate intake (mg/day)**
Children	
1–3	2000

4–8	2300
Males	
9–13	2500
14–18	3000
19–30	3400
31–50	3400
51–70	3400
> 70	3400
Females	
9–13	2300
14–18	2300
19–30	2600
31–50	2600
51–70	2600
> 70	2600

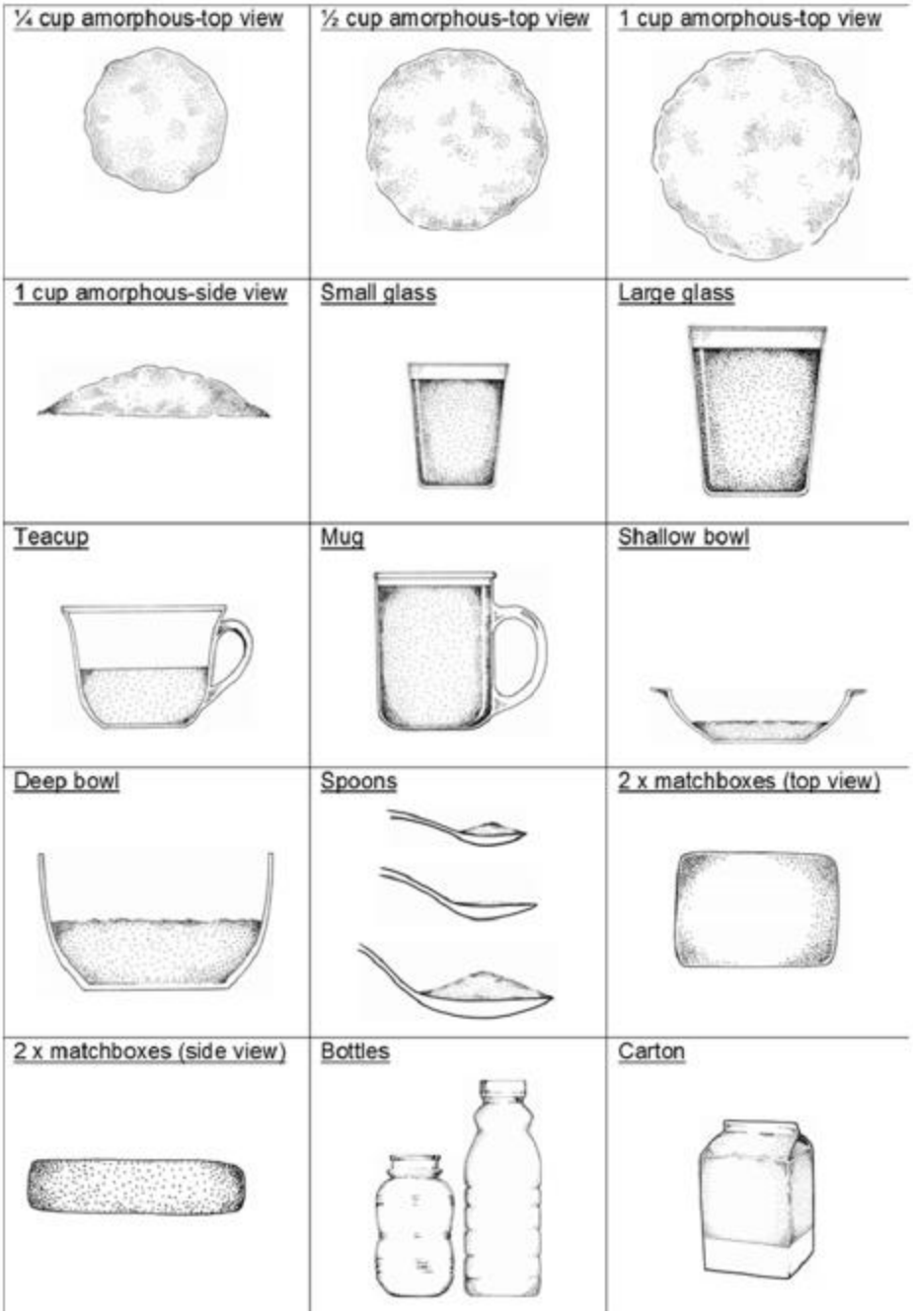


Figure 3.4.3.1: Examples of sketches and measures used in this study (Senekal *et al*., 2020).

3.5. QUALITY CONTROL

Standard procedures as described by ISAK were followed for all training of anthropometric measurement and BP measurements of participants (Norton and Olds, 1996). In addition, BP measurement were done according to the guidelines describes by Whelton et al. (2018) and NHLBI (2005). Fieldworkers underwent testing for the reliability of measurements as part of their training (Monyeki *et al*, 1999). This was done to achieve a technical error of measurement within limits. The training was conducted according to the three-level of criteria as per ISAK guidelines (Norton and Olds, 1996). In brief, the absolute and relative values for intra-tester and inter-tester technical error measurements (%TEM) for height measurements ranged from 0.04-4.16 cm (0.2-5.01%) and WC 0.0-3.4 cm (0-4%) (Monyeki *et al*., 1999; Monyeki *et al*., 2002). In order to determine the validity and reliability which was used in the different African states, definitions and procedure were discussed by each team and consensus drawn so that results can be compared across countries. Considerable amount of time was devoted to this section in the development of the questionnaire before the study commenced. The questionnaires were used by well-trained ELS field workers who were Northern Sotho (Sepedi) speaking. The calculation of Na and K were done using a standardised South African food finder programme, SAFOODS to determine the concentration amount.

3.6. STATISTICAL ANALYSIS

IBM SPSS Statistics software package (version 27.0) was used to conduct statistical analysis. Shapiro–Wilk test was used to assess the variables' normality.

3.6.1. Descriptive characteristics

To describe and characterise the samples, descriptive statistics were calculated for all the variables (Na, K, Na/K, SBP, DBP, WC and WHtR) to indicate frequencies (expressed as percentages), means and standard deviations. Parametric (one-way ANOVA), Chi-square, and Fisher’s exact tests were conducted to determine the difference in males and females over the years; and between the years.

3.6.2. Generalized estimating equation

GEE (linear and binary logistics) were conducted to assess the association of sodium intake, potassium intake; and their ratio with BP, WC and WHtR; and further investigate the association with hypertension and abdominal obesity, to calculate the risk of hypertension and abdominal obesity. A longitudinal tracking GEE technique, which measures the association between an indicator at the first period of measurements and the same indicator at all other periods of measurements were conducted with age and sex included in the model (Twisk *et al*., 1994; Mohammadi *et al*., 2014). The probability value for statistical significance for all tests was set at a p-value ≤ 0.05.

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CHAPTER 4

RESULTS AND DISCUSSION

4.1. Introduction

4.2. Characteristics of the population

4.3. Prevalence of sodium, potassium intake, sodium-to-potassium ratio, hypertension and abdominal obesity

4.4. Association of sodium, potassium intake and sodium-to-potassium ratio with blood pressure and abdominal indices over time

4.5. The risk measure of sodium, potassium intake and sodium-to-potassium ratio causing the development of hypertension and abdominal obesity over time

4.6. Conclusion

4.7. References

4.1. Introduction

This chapter presents the findings of the study, where the relationship between selective micronutrients such as Na, K intake and Na/K ratio with BP and abdominal indices were explored. In addition to this, to explore the risk of Na, K intake and Na/K ratio causing the development of HT and abdominal obesity. This was conducted as studies of this kind are very limited, especially in South Africa/Africa.

4.2. Characteristics of the population

- Demographic characteristics

The characteristics of the population for each year were stratified by sex. This study constitutes a total of 325 participants, with 49.5% (161) males and 50.5% (164) females. The average ages in males were 8.8, 10.8 and 24.8 years, whilst in females were 9.6, 11.6 and 25.5 years, respectively. Bear in mind that this is a longitudinal study, and the age of the participants is meant to change. Hence, there is a statistical difference in the average age in both males ($p< 0.001$) and females ($p< 0.001$) over time.

- Selective micronutrients: Na, K and Na/K ratio

Table 4.2.1. A indicates an increase in the dietary mean of Na intake in both males and females over the years, however, the increase was not significant in males ($p> 0.05$) and females ($p> 0.05$). The dietary mean of K intake significantly decreased from the first year of assessment to the last year of assessment, in both males ($p< 0.001$) and females ($p< 0.001$). Whilst the dietary mean of Na/K ratio significantly increased over the years, in both males ($p< 0.001$) and females ($p< 0.001$). **Table 4.2.1.B** further shows the dietary mean of the micro-nutrients [Na ($p< 0.05$), K intake ($p< 0.001$) and Na/K ratio ($p< 0.001$)] significantly increased over the years for the total population. Both males and females did not meet the recommendation for Na and K intake throughout the years (Consensus Study Report, 2019). Na/K ratio in the first and second assessment ratio met the recommendation as compared to WHO, however in the third year of assessment it was notably above the recommended ratio (WHO, 2012).

Due to the lack of studies that are similar to this study, it is difficult to make comparisons as most are cross-sectional studies and not longitudinal studies (following the same participants from childhood to adulthood). Available longitudinal studies were of an open cohort, which focus mostly on young adults and adults (≥ 18 or 20 years). However, this study concurs with the literature, as dietary habits developed from childhood are taken through into adulthood, which has indicated that children with extreme or increased levels of Na and lower K intake tend to maintain those levels over time (Aburto *et al*., 2013).

- BP measurements

Table 4.2.1.A shows the mean of both SBP and DBP significantly increased from the first year of assessment to the last year of assessment, respectively in both males ($p < 0.001$) and females ($p < 0.001$). In addition, the mean of BP measurements of the total population significantly increased from the first point of measurement, with SBP ($p < 0.001$) increasing to 120.75 mmHg and DBP ($p < 0.001$) to 70.67 mmHg in the last year of measurement, respectively in **Table 4.2.1.B**. Although there is a significant increase in SBP ($p < 0.001$) and DBP ($p < 0.001$), on average the total population were classified with normal BP (NHLBI, 2005; Whelton *et al*., 2018).

Previous longitudinal data from diverse populations have shown different BP tracking patterns. The inconsistencies may be due to differences in study design, baseline age, follow-up period, measuring instruments, intrasubject variability, characteristics of study samples, or analytic methods used. Nevertheless, most studies found significant BP tracking, and some found a weaker tracking for longer follow-up periods and a stronger tracking for SBP than for DBP, which was possibly due to the difficulties in measuring DBP in children and the changes in the recommendations on how to take DBP measurements over time (Chen and Wang, 2008).

- Abdominal obesity

The mean of abdominal indices (WC and WHtR) significantly increased over the years, in both males ($p < 0.001$) and females ($p < 0.001$). Although the increase, males had on average WC and WHtR that were below the 90^{th} percentile or 90 cm and 0.5 for all the years (Yoo, 2016). In addition to this, females on average had a ratio and WC that is

below 0.5 and the 90th percentile, except in 2015 were the ratio and WC were both above 0.5 and 80 cm (Yoo, 2016). The mean of abdominal indices significantly increased from the first measurement (54.44 cm-WC and 0.41-WHtR), with WC increasing to 79.0 cm and WHtR increasing to 0.48 in the last year as shown in **Table 4.2.1B**. Although there was an increase in both abdominal indices, they were however not above the cut-off points of WC and WHtR as stated by Yoo. (2016). Several studies have documented age-related changes in adipose tissue distribution as reflected by an increase in abdominal obesity with age (Tchernof and Després, 2013).

Table 4.2.1.A: characteristics of the population over the years, stratified by sex

Year	1999		2001		2015	
	Males (n=161)	Females (n=164)	Males (n= 161)	Females (n=164)	Males (n=161)	Females (n=164)
Variables	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Age	8.8 ± 1.86	9.6 ± 1.75	10.8 ± 1.86	11.5 ± 1.75	24.8 ± 1.84	25.5 ± 3.13
Na (mg/d)	482.06 ± 593.49	532.84 ± 593.03	482.06 ± 593.49	535.82 ± 592.44	661.45 ± 988.80	717.18 ± 1082.93
K (mg/d)	1043.97 ± 836.65	1165.77 ± 963.26	1043.97 ± 836.65	1175.37 ± 968.15	**756.28 ± 657.31****	**753.27 ± 678.56****
Na/K ratio (mmol)	0.90 ± 1.03	0.89 ± 0.93	0.90 ± 1.03	0.89 ± 0.92	**2.03 ± 2.75****	**1.92 ± 2.67****
SBP (mmHg)	99.86 ± 11.71	102.17 ± 11.43	95.51 ± 9.90	99.39 ± 11.21	**126.72 ± 13.11****	**115.00 ± 10.63****
DBP (mmHg)	61.90 ± 9.89	62.92 ± 9.58	63.58 ± 7.21	64.66 ± 8.04	**71.45 ± 10.17****	**69.93 ± 8.99****
WC (cm)	53.93 ± 3.77	54.96 ± 4.69	56.65 ± 4.14	58.23 ± 4.30	**74.53 ± 9.12****	**84.01 ± 15.39****
WHtR	0.41 ± 0.02	0.40 ± 0.02	0.41 ± 0.04	0.41 ± 0.04	**0.43 ± 0.08****	**0.52 ± 0.12****
Height	130.36 ± 120.08	135.10 ± 10.68	138.51 ± 11.68	143.47 ± 10.77	**173.54 ± 12.83****	**162.79 ± 10.29****

n—number of individuals. Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, WHtR—Waist-to-height ratio, mg/d—milligram per day, mmol/d—millimole per day, cm—centimeter, ****p-value < 0.001**

Table 4.2.1.B: Comparison of means of sodium, potassium intake, sodium-to-potassium ratio, systolic and diastolic BP over the years

Year	1999	2001	2015	p-value
Variables	Mean ± SD	Mean ± SD	Mean ± SD	
Na (mg/d)	507.69 ± 592.89	509.27 ± 592.66	689.84 ± 1036.56	**0.008***
K (mg/d)	1105.43 ± 903.43	1110.48 ± 906.59	754.75 ± 667.16	**0.000****
Na/K ratio (mmol)	0.89 ± 0.98	0.89 ± 0.98	1.97 ± 2.70	**0.000****
SBP (mmHg)	101.02 ± 11.61	97.47 ± 10.74	120.75 ± 13.26	**0.000****
DBP (mmHg)	62.41 ± 9.73	64.12 ± 7.64	70.67 ± 9.60	**0.000****
WC (cm)	54.44 ± 4.27	57.47 ± 4.30	79.40 ± 13.53	**0.000****
WHtR	0.41 ± 0.24	0.41 ± 0.04	0.48 ± 0.11	**0.000****

n—number of individuals. Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, WHtR—Waist-to-height ratio, SD—standard deviation, mg/d—milligram per day, mmol/d—millimole per day, cm—centimeter, *** p-value < 0.05; **p-value < 0.001**

- Most frequent food items

Table 4.2.2 presents the most frequent food items in the diets of the Ellisras participants from 2001 and 2015. In 2001, maize or sorghum porridge was the most stable food amongst the Ellisras children with jam being the least used item. Whilst in 2015, fried chicken with skin was the most used item and pilchards were the least used food item in the Ellisras young adults.

Table 4.2.2: The most frequently used food items for the years 2001 and 2015 in Ellisras, from the most used to the least (Van Den Ende *et al*., 2014; Mashiane *et al*., 2018).

2001	2015 (%)
Maize porridge or Sorghum	Fried chicken with skin (23.8)
Sugar (white)	Pap (22.6)
Homemade bread	Cold drink (16.9)
Chicken	White sugar (14)

Spinach	Vetkoek (5.8)
Non-dietary creamer	Fried beef (4.7)
Beef	Peanut butter (4.4)
Red meat (from goat and wild animals)	Samp (2.6)
Tomato and onion	Yoghurt (2.4)
Cooked dry beans	Spinach (2.0)
Cold drink (mostly coke)	Pilchards (0.5)
Peanut butter	
Sweets	
Mashontja (Mopani worms)	
Bananas and oranges	
Cow milk	
Jam	

4.3. Prevalence of sodium, potassium intake, sodium-to-potassium ratio, hypertension and abdominal obesity

- The proportion frequency of sodium, potassium intake, sodium-to-potassium ratio

The proportion frequency of individuals who consumed above the recommended adequate intake for Na moderately increased from 8.7% in the first year to 11.8% in the third year in males. Whilst in females, it decreased from 11.0% to 10.4% in the third year. In both sexes, there was no statistical difference between the years. The proportion frequency of individuals who consumed below the recommended adequate intake for K in males was 96.9% and in females, it was 93.3% in the first year. In addition to this, K intake slightly decreased to 96.3% in males and significantly increased to 99.4% in females ($p< 0.05$), in the third year, respectively. The proportion frequency of Na/K ratio significantly increases from the first and into the third year of assessment in both males ($p< 0.001$) and females ($p< 0.05$), respectively as shown in **Table 4.3.1.A**

In this study, it was observed that more than 9% of the population each year consumed above the recommended intake of Na. In addition, over 90% of the sample population each year did not adhere to the recommended intake of potassium. We further observed that more than 30% of the sample population each year consumed above the

recommended ratio intake. There was a significant increase in the proportion frequency of micronutrients [K- ($p< 0.05$) and Na/K- ($p< 0.001$)] with an exception of Na as shown in **Table 4.3.1.B**. The increase in the proportion frequency of the selected micronutrient could be possibly explained by the demographic shift, as some rural areas in South Africa are experiencing urbanisation, which is marked by a western diet high in processed food (Van Den Ende *et al*., 2014) and as mentioned before the dietary mean of Na and Na/K ratio increased over time, whilst the mean of K decreased over time. This indicates that this population consumed foods that are high in Na and low in K, this can be demonstrated in **Table 4.2** did the population in 2001 consumed foods that are high in K (such as beans, oranges, bananas, spinach, tomatoes and onions and cow milk), whilst in 2015 consumption of K was low in which the only food that was high in K were yoghurt and spinach. The WHO gives examples of food products with high potassium content: different beans and peas, nuts, green vegetables, root vegetables (carrot, onions, beetroot), other vegetables (tomatoes, cucumbers, pumpkins), and fruits (bananas, papayas, and dates) (WHO, 2012).

- The prevalence of hypertension

Table 4.3.1. A indicates a significant increase in HT from the first year to the third year, in both males ($p< 0.001$) and females ($p< 0.001$), respectively. An increase from 5.0% in males to 22.5% and 4.9% in females to 24.2%. Furthermore, results in **Table 4.3.1.B** shows the prevalence of HT was 4.9% at the first measurement, and it significantly increased to 23.4% in the last year of assessment ($p< 0.001$). Our results are similar to results by Kagura et al, (2015) conducted in South Africa, which reported the prevalence of HT increased from 8.4% in black children aged 5 years to 24.4% at age 18 years, although there is some difference regarding the age group.

- The prevalence of abdominal obesity

The prevalence of abdominal obesity according to WC increased from 36.0% in males and 43.6% in females. Whilst in the third year, the prevalence notably decreased to 10.6% in males ($p< 0.001$) and significantly increased to 58.2% in females ($p< 0.001$). Abdominal obesity was only prevalent in males, with a prevalence of 0.6%. However, in the second

year of assessment, the prevalence increased to 2.5% in males and 6.1% in females. The prevalence in the final year of assessment further increased to 11.9% in males (p< 0.001) and 58.8% in females (p< 0.001), respectively. In addition, the prevalence of abdominal obesity according to WC significantly decreased by 39.8% from the first point of measurement to 34.8% in the last year of assessment (p< 0.001). Whilst the prevalence of abdominal obesity according to WHtR significantly increased from 0.3% at first measurement to 35.7% in the last year of measurement (p< 0.001) for the total population as indicated in **Table 4.3.1.B**. It must be noted that the majority of the participants when they were children in 2001 were classified as underweight as stated by Van Den Ende et al. (2014). Therefore, the high prevalence of abdominal obesity according to WC as compared to abdominal obesity according to WHtR might be due to bloating of malnourishment rather than visceral fat (Raphadu *et al*., 2022), as the prevalence in 2001 was the highest compared to the other two years.

Table 4.3.1.A: The proportion frequency of sodium, potassium, and the prevalence of HT and abdominal obesity stratified by sex

Year	**1999**		**2001**		**2015**	
Dependent variables	Males n (%)	Females n (%)	Males n (%)	Females n (%)	Males n (%)	Females n (%)
Na above the adequate intake	14 (8.7)	18 (11.0)	20 (12.4)	15 (9.1)	19 (11.8)	17 (10.4)
K below the adequate intake	156 (96.9)	153 (93.3)	153 (95.0)	150 (91.5)	155 (96.3)	**163 (99.4)***
Na/K above the recommended intake	57 (35.4)	56 (34.1)	58 (36.0)	56 (34.1)	**84 (52.2)****	**82 (50.0)***
Hypertension	8 (5.0)	8 (4.9)	4 (2.5)	6 (3.7)	**36 (22.5)****	**40 (24.2)****
Abdominal obesity according to WC	58 (36.0)	71 (43.6)	108 (67.1)	131 (79.9)	**17 (10.6)****	**96 (58.2)****

Abdominal obesity according to WHtR	0 (0)	1 (0.6)	4 (2.5)	10 (6.1)	**19 (11.9)****	**97 (58.8)****

n—number of individuals. Na—sodium, K—potassium, WC—waist circumference, WHtR—waist-to-height ratio, mg/d—milligram per day, ***p-value < 0.05, ** p-value < 0.001**

Table 4.3.1.B: The proportion frequency of sodium, potassium, and the prevalence of hypertension and abdominal obesity over the years

Year	**1999**	**2001**	**2015**	**Chi-square p-value**
Dependant Variables	n (%)	n (%)	n (%)	
Na above the adequate intake	32 (9.8)	35 (10.8)	36 (11.1)	0.868
K below the adequate intake	309 (95.1)	303 (93.2)	318 (97.8)	**0.019***
Na/K above the recommended intake	113 (34.8)	114 (35.1)	166 (51.1)	**<0.001***
Hypertension	16 (4.9)	10 (3.1)	76 (23.4)	**<0.001****
Abdominal obesity according to WC	129 (39.8)	239 (73.5)	113 (34.8)	**<0.001****
Abdominal obesity according to WHtR	1 (0.3)	14 (4.3)	116 (35.7)	**<0.001****

n—number of individuals. Na—sodium, K—potassium, WC—waist circumference, WHtR—waist-to-height ratio, ***p-value < 0.05 ** p-value < 0.001**

4.4. The relationship between dependent variables and independent variables

4.4.1. Association of sodium, potassium intake and sodium-to-potassium ratio with blood pressure and abdominal indices over time

Table 4.4.1.1 indicates that there was no positive association between Na intake and blood pressure measurements [SBP- β = -0.507, (95% CL: -4.379 – 3.366) p-value= 0.798; DBP- β = -0.204, (95% CL: -2.287 – 1.807) p-value= 0.818] and abdominal indices [WC- β = -0.735, (95% CL: -4.071 – 2.600) p-value= 0.666; WHtR- β = -0.001, (95% CL: -0.016 – 0.013) p-value= 0.866], even when the model was adjusted for age and sex there was still no association. This could be due to the lower mean intake of Na of the total population, although on average the population did not meet the recommended intake, they were however not above the recommended intake of Na (Consensus Study Report, 2019). Our results are contrary to the literature as increased levels of Na intake may affect

the increase of BP and the development of HT (WHO, 2008). There was no positive association between K intake and BP measurements [SBP- β = 2.033, (95% CL: -2.022 – 6.088) p-value= 0.326; DBP- β = 0.736, (95% CL: -1.738 – 3.211) p-value= 0.560] and WC [β = 2.314, (95% CL: -1.017 – 5.644) p-value= 0.173]. Some previous studies reported no association between K intake and systolic blood pressure, these results are partially similar to our results (Zhang *et al*., 2013).

However, a positive significant relationship between K intake and WHtR over the years was observed, which means an increase in K increases WHtR by 0.019 cm (95% CL: 0.004 – 0.034). Even when adjusted for the relevant covariates such as age and sex, K intake was still associated WHtR (one increase in K intake caused an increase in WHtR by 0.018 cm) over the years. This could probably be due to the notably low mean intake of K throughout the measured years, as mentioned before more than 90% of the sample population each year consumed below the recommended intake. These results could be explained by the lack of intake of fruits and vegetables in 2015 compared to 2001 (e.g. the sample population consumed bananas and oranges, spinach, tomato and onion which are high in K), this might have resulted in the decline in K over time. This significant decrease in K has an effect on the increase in WHtR over time. However, it must be noted that there were no relevant studies in which we can compare the outcome of the results, as this study differs from other studies in the study design. Most studies in this field that have been conducted over the past five years indicate that the K effect on obesity is a new topic, and more robust studies with better design are warranted (Lee *et al*., 2013), especially on abdominal obesity as previous studies mostly focused on BMI and not abdominal indices such as WC and WHtR. However, Binia et al. 2015 reported an association between urinary Na/K ratio and obesity. The precise mechanism between potassium intake and obesity/MetS is unclear. Central obesity is a component of metabolic syndrome, and the mechanisms of obesity and MetS are homogeneous. Obesity is associated with potassium channel function (Climent *et al*., 2014; Crunkhorn, 2013). Despite this, Murakami et al. (2012) stated that a higher intake of potassium may also be associated with a lower risk of obesity mainly due to higher intakes of fruits and vegetables, which are the major sources of potassium, although the effect of fruits and vegetables on obesity is controversial (Vergnaud *et al*., 2012).

Furthermore, the Na/K ratio was positively associated with BP measurements [increasing SBP by 4.236 mmHg (95% CL: 2.056 – 6.595) and DBP by 2.028 mmHg (95% CL: 0.703 – 3.353)], and abdominal indices [increasing WC by 4.191 cm (95% CL: 2.080 – 6.302) and WHtR by 0.0014 cm (95% CL: 0.003 – 0.026)]. However, when the model was adjusted for age and sex, an association was not found between the sodium-to-potassium ratio and blood pressure measurements and abdominal indices, respectively. Our study concurs with the literature (in the case of BP and not abdominal indices) in which Pereira et al. (2019) found independent associations of Na and K intake with BP, however when evaluated in a combined manner, as in the case of the sodium-to-potassium ratio, the effect was potentiated. Na/K ratio is perhaps also a sensitive index to evaluate the risk of obesity and MetS in addition to K intake, although more studies are needed to authenticate the results (Cai *et al*., 2016).

Despite the above-mentioned results, overall it was difficult to compare our results with previous studies because of the differences in the characteristics of participants, methods and age groups. Studies were either cross-sectional studies or longitudinal studies, and those longitudinal studies focused on children or adults and not tracking from childhood to adulthood of the same participants as in this study.

Table 4.4.1.1: The association of sodium intake, potassium intake and sodium-to-potassium ratio with blood pressure and abdominal indices using GEE (linear)

	Na			K			Na/K		
Unadjusted	*β*	95% CI	p-value	*β*	95% CI	p-value	*β*	95% CI	p-value
SBP	-0.507	-4.379, 3.366	0.798	2.033	-2.022, 6.088	0.326	4.326	2.056, 6.595	**<0.001****
DBP	-0.240	-2.287, 1.807	0.818	0.736	-1.738, 3.211	0.560	2.028	0.703, 3.353	**0.003***
WC	-0.735	-4.071, 2.600	0.666	2.314	-1.017, 5.644	0.173	4.191	2.080; 6.302	**<0.001****
WHtR	-0.001	-0.016, 0.013	0.866	0.019	0.004, 0.034	**0.012***	0.014	0.003, 0.026	**0.015***
Adjusted for age and sex									
SBP	0.915	-1.769, 3.599	0.504	0.579	-2.662, 3.821	0.726	0.967	-0.655, 2.589	0.243
DBP	0.311	-1.571, 2.194	0.746	0.283	-2.142, 2.709	0.819	0.798	-0.418, 2.014	0.198
WC	1.052	-0.668, 2.773	0.231	1.247	-0.642, 3.136	0.196	0.469	-0.701, 0.617	0.432
WHtR	0.004	-0.009, 0.017	0.57	0.018	0.002, 0.034	**0.024***	0.005	-0.005, 0.015	0.329

Na-sodium, K-potassium, Na/K ratio- sodium/potassium ratio, SBP-systolic blood pressure, DBP-diastolic blood pressure, WC-waist circumference, WHtR- waist-to-height ratio, mg/d- milligram per day, mmol/d- millimole per day, β – beta coefficient, *** p-value < 0.05, **p-value < 0.001**

4.4.2. The risk measure of sodium, potassium intake and sodium-to-potassium ratio causing the development of hypertension and abdominal obesity over time

Table 4.4.2.1 shows that in this study, it was further observed that the Na/K ratio increased the risk of developing HT [Exp *β*= 1.603, (95% CL: 1.164, 2.207) *p*-value= 0.004] and abdominal obesity [Exp *β*= 1.797, (95% CL: 1.207, 2.677) *p*-value= 0.004] compared to Na and K intake alone. This sample population is 1.603 times more likely to develop HT and 1.797 times more likely to develop abdominal obesity over time. The joint effects of low Na and high K intakes on BP, HT, and related factors may be larger than the effects of either Na or K alone (Sacks *et al*., 2001; Perez and Chang, 2014). The dominance of the Na/K ratio as a predictor of BP variation compared to Na and K as individual predictors have been observed in several studies, most of them conducted in hypertensive patients (Perez and Chang, 2014), higher Na/K ratio may lead to higher BP during follow-up (Okayama *et al*., 2016). Ge et al. (2015) further revealed that the urinary Na/K ratio was also associated with obesity independently, and a high Na/K ratio could increase the risk of obesity. However, no association was found between the Na/K ratio (derived from self-reporting) and obesity (Murakami *et al*., 2015). This is contrary to this study's results as the Na/K ratio was derived from self-reporting dietary in our study and the study conducted by Murakami et al. (2015). When data were adjusted for age and sex, no association was found Na/K ratio and further could not predict HT and abdominal obesity in this sample population. Elevated levels of Na intake and inadequate levels of K intake may affect the development of hypertension (WHO, 2012). To concur with the previous literature stated above, our results showed a significant increase in Na and a significant decrease in K intake over the measured years in this population. Several processes could account for the link between the Na/K ratio and obesity risk. First, diets rich in Na and low in K are frequently high in energy and, as a result, may encourage weight gain. Furthermore, the consequences of thirst and a salty diet may increase sugar-sweetened beverage consumption which is linked to weight gain (He *et al*., 2008; Grimes *et al*., 2013; Grimes *et al*., 2016).

Table 4.2.2.1: The risk measure of sodium, potassium intake and sodium-to-potassium ratio causing the development of hypertension and abdominal obesity according to WC and WHtR using GEE (binary logistic)

	Na			K			Na/K		
Unadjusted	Exp (β)	95% CI	p-value	Exp (β)	95% CI	p-value	Exp (β)	95% CI	p-value
Hypertension	0.959	0.590, 1.558	0.865	1.941	0.762, 4.941	0.164	1.603	1.164, 2.207	**0.004***
Abdominal obesity according to WC	1.131	0.730, 1.754	0.581	0.634	0.321, 1.254	0.190	0.912	0.684, 1.216	0.529
Abdominal obesity according to WHtR	1.066	0.579, 1.964	0.837	1.964	0.589, 6.545	0.272	1.797	1.207, 2.677	**0.004***
Adjusted for age and sex									
Hypertension	0.873	0.530, 1.437	0.593	0.679	0.283, 1.632	0.387	0.968	0.668, 1.403	0.864
Abdominal obesity according to WC	1.136	0.715, 1.805	0.588	0.698	0.362, 1.346	0.283	0.989	0.731, 1.338	0.943
Abdominal obesity according to WHtR	0.654	0.312, 1.372	0.262	0.702	0.221, 2.233	0.549	0.781	0.493, 1.239	0.295

Na-sodium, K-potassium, Na/K ratio- sodium/potassium ratio, SBP-systolic blood pressure, DBP-diastolic blood pressure, WC-waist circumference, WHtR- waist-to-height ratio, mg/d- milligram per day, mmol/d- millimole per day, Exp β – exponential beta, *** p-value < 0.05**

4.6. Conclusion

In conclusion, this current study showed a positive significant relationship between micronutrients and BP measurement and abdominal indices. Where Na intake was positively associated with WHtR over time. In addition to this, the Na/K ratio was also positively associated with BP measurement (SBP and DBP) and abdominal indices (WC and WHtR). An increase in the Na/K ratio was further found to increase the risk of developing HT and abdominal obesity over time. However, more data is crucial in establishing the effectiveness of Na reduction and the increase in K intake, and with the use of more valuable methods to better understand their effect on blood pressure and abdominal weight, especially in Africa. Longitudinal studies in children provide a tremendous global resource to direct prevention strategies for HT and abdominal obesity.

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CHAPTER 5

INTRODUCTION, SUMMARY, PERSPECTIVE, LIMITATIONS AND RECOMMENDATIONS

5.1. Introduction

5.2. Summary

5.3. Perspective

5.4. Limitations

5.4. Recommendations

5.5. References

5.1. INTRODUCTION

This chapter summarises the results of this research study, which includes a discussion of the main findings according to the hypothesis and questions posed in chapter 1. This is followed by limitations of the study and recommendations for future research studies concerning the association of Na, K and Na/K ratio with the risk of developing (BP measurement) hypertension and abdominal obesity (abdominal indices). To further identify which micronutrient (Na, K and Na/K ratio) is a better predictor of HT and abdominal obesity, especially in Africa.

5.2. SUMMARY

Chapter 1 outlines the problems highlighted regarding this research study topic and the motivation for investigating the association of Na, K and Na/K ratio with the risk of developing hypertension (BP measurement) and abdominal obesity (Abdominal indices) from childhood to adulthood in Ellisras rural area. Furthermore, to investigate which micronutrient (Na, K and Na/K ratio) is a better predictor of HT and abdominal obesity. The subsequent objectives of this research study which were stated in chapter 1 were utilised to answer the statements above and are as follows:

I. To determine the proportion frequency of high dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies.

II. To track the prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies over time.

III. To determine the average concentration level of Na and K and NA/K ratio over time.

IV. To determine which of the selected micronutrients (Na, K, and Na/K ratio) will be associated on BP and abdominal indices over time by using the GEE (linear) technique.

V. To determine which of the selected micronutrients (Na, K, and Na/K ratio) will be the predictor for HT and abdominal obesity.

The hypotheses of this study are as follows:

I. Hypothesis 1: The proportion frequency of dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies over time will increase in the population of Ellisras, from childhood to young adulthood.

II. Hypothesis 2: The prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies will show an increase over time.

III. Hypothesis 3: The average concentration of Na and NA/K ratio will increase over time, whilst K intake decrease in the Ellisras rural children into young adulthood.

IV. Hypothesis 4: All selected micronutrients (Na, K, and Na/K ratio) will be associated with BP and abdominal indices over time by using the GEE (linear) technique.

V. Hypothesis 5: Na/K ratio will be found to be the predictor of the risk of developing HT and abdominal obesity compared to Na and K alone over time.

Chapter 2 Literature states that multiple factors can influence the development of HT and abdominal obesity. Dietary intake has been identified as one of the important risk factors for NCDs. High Na and low K intakes are associated with the early development of chronic diseases (e.g., HT, obesity) (Golpour-Hamedani, 2022). Several mechanisms exist to indicate how Na and K can influence blood pressure. Evidence indicates that an interaction between these micro-nutrients plays a dominant role in the development of

primary HT (Adrogué and Madias, 2007; Perez and Chang, 2014). Diets defined by high Na and low K induce a biological interaction with the kidneys, resulting in excessive Na levels and inadequate K concentrations in the human body (Adrogué and Madias, 2007; Perez and Chang, 2014). Childhood dietary patterns are manifested in adulthood; children who consume excessive amounts of Na and minimal K tend to maintain those levels over time (Aburto *et al*., 2013). Furthermore, data on Na and K intake in children and young people in Africa is inadequate. Because dietary patterns are generally viewed as a key cause of NCDs, describing the projected nutritional transition is crucial to evaluate the impact of food on this category of diseases. This work, however, is scarce or badly documented in developing nations due to logistical and financial restrictions. According to Pisa et al. (2014), another explanation for the paucity of this study is a lack of trustworthy dietary assessment procedures, which supports the rising need for the creation, validation, and standardisation of tools for measuring and monitoring food intake in different nations.

Chapter 3 explains the research study design, which was a longitudinal study. It explains how the methodical procedure was followed to collect data and how it was analysed. One way Anova was conducted was to analyse statistical differences of means within males and females over the measured years. A Chi-square test was conducted to analyse the statistical differences in the prevalence between males and females. GEE (linear and binary) was conducted to analyse the association between Na, K and Na/K ratio and BP measurements and abdominal indices; and the association between Na, K and Na/K ratio and the risk of HT and abdominal obesity.

Chapter 4 highlights the description of the results and the discussion of the results. In the Ellisras population it was indicated that a significant positive association was found between K intake and WHtR ($p< 0.05$), and even after age and sex were taken into account in the model, there was still an association with WHtR ($p< 0.05$). The precise mechanism between potassium intake and obesity/MetS is unclear. Central obesity is a component of MetS, and the mechanisms of obesity and MetS are homogeneous. Obesity is associated with potassium channel function (Climent *et al*, 2014; Crunkhorn, 2013). Na/K ratio was significantly associated with SBP (p-value< 0.001), DBP ($p< 0.005$),

WC (p-value< 0.001) and WHtR (p< 0.05), respectively. However, when the model was adjusted for age and sex, an association was not found between the Na/K ratio and BP measurements and abdominal indices, respectively. An increase in the Na/K ratio placed the risk of developing hypertension (p< 0.05) and abdominal obesity (p-value= 0.004) over time. However, when age and sex were taken into account, the Na/K ratio was not found to increase the risk of developing hypertension and abdominal obesity. This concurs with the literature stated by Sacks et al. (2001) and Perez and Chang, (2014) that the joint effects of low Na and high K intakes on BP, HT, and related factors may be larger than the effects of either Na or K alone.

In conclusion, this current study showed a positive significant relationship between micronutrients and BP measurement and abdominal indices. Where Na intake was positively associated with WHtR over time. In addition to this, the Na/K ratio was also positively associated with BP measurement (SBP and DBP) and abdominal indices (WC and WHtR). An increase in the Na/K ratio was further found to increase the risk of developing hypertension and abdominal obesity over time. However, more data is crucial in establishing the effectiveness of Na reduction and the increase in K intake, and with the use of more valuable methods to better understand their effect on blood pressure and abdominal weight especially in Africa to provide us with data on dietary habits of children into their adult life. Since children with high levels of Na and lower levels of K could maintain this dietary habit into adulthood, and this could lead to the development of hypertension and abdominal obesity. Thus, close monitoring of children is needed for better management of their health and this will assist in mitigating premature HT and abdominal obesity.

Interpretation of the main findings and comparison with the relevant literature together with the objectives and hypotheses stated in chapter 1.

Objective 1: To determine the proportion frequency of high dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies.

i. Hypothesis 1: The proportion frequency of dietary intake for Na, K and Na/K ratio based on data collected using questionnaires from previous surveys/studies over

time will increase in the population of Ellisras, over time compared to the recommended intake of the Consensus Report (2019) and WHO (2012).

In this study it was observed that a slight increase in the number of individuals who consumed above the recommended intake of Na, increased from 9.8% in 1999 to 11.1% in 2015, however, the increase was not significant ($p > 0.05$). In addition, the proportion frequency of K increased significantly from 95.1% in 1999 to 97.8% in 2015. We further observed a further significant increase in the Na/K ratio from 34.8% in 1999 to 51.1% in 2015. These results indicate that the number of individuals who complied with the recommendations of the Consensus Report of 2019 and WHO (2012) increased over time for the selective micronutrients (Na, K intake and Na/K ratio). Although our study showed an increase in Na, a study conducted by the Centres for Disease Control and Prevention (CDC) (2011) showed a higher prevalence of Na compared to our study with the prevalence Na intake ranging from 79.1% for U.S. children aged 1–3 years to 95.4% for U.S. adults aged 19–50 years. The reason might due to the different geographical regions and use of methods (age range, statistical analysis and study design) since the U.S. is a developed country and South Africa is a developing country. It is difficult to make the comparison as there are no relevant studies of the same study setting.

In line with the stated results, we, therefore, partially accept this hypothesis.

Objective 2: To track the prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies over time compared to the cut-off measurement of NHLBI (2005) and Whelton *et al,* (2018)

ii. Hypothesis 2: The prevalence of HT and abdominal obesity based on data collected using BP and abdominal indices measurements from previous surveys/studies will show an increase over time compared to the cut-off measurement of NHLBI (2005) and Whelton *et al,* (2018) from childhood to adulthood.

The prevalence of HT was 4.9% in the first year, and it significantly increased to 23.4% in the last year of assessment ($p < 0.001$). In addition, the prevalence of abdominal obesity

according to WC significantly decreased from the first year 39.8% to 34.8% in the last year of assessment ($p< 0.001$). Whilst the prevalence of abdominal obesity according to WHtR significantly increased from 0.3% in the first year to 35.7% in the last year ($p< 0.001$). This result indicates that the prevalence of HT and abdominal obesity according to WHtR increased with time (years), however, the prevalence of abdominal obesity according to WC decreased with time. Our findings are consistent with those of Kagura et al. (2015), who found that the prevalence of HT rose from 8.4% in black children aged 5 years to 24.4% at age 18 years, however, there are minor differences depending on the age group. In addition, a study by Molina et al. (2022) in Southern India (age 6–16 years) reported an increased prevalence of WC from 15.4% to 27.1% during follow-up and the prevalence of WHtR increased from 9.6% to 20.5% during follow-up. This study partially contradicts our study as reported a decrease in the prevalence of WC, however, our prevalence of WC and WHtR during follow-up was higher compared to the study of Molina et al. (2022). Geographical, ethnic and age range differences could explain the differences in the results in comparison to the mentioned study by Molina et al. (2022).

In line with the results observed above, the partially accepted.

Objective 3: To determine the average concentration of Na and K micronutrients and the NA/K ratio over time.

iii. Hypothesis 3: The average concentration of Na will increase and K will decrease, and the NA/K ratio will increase over time in the Ellisras rural children into their young adulthood compared to the recommended intake of the Consensus Report (2019) and WHO (2012).

The dietary mean of Na intake ($p< 0.05$) and Na/K ratio ($p< 0.001$) increased over time, whilst K intake ($p< 0.001$) decreased over time. Na intake significantly increased from 507.69 mg in 1999 to 689.84 mg in 2015. Although there was an increase in the mean intake of Na, it was not above the recommended intake by Consensus Report (2019) throughout the years. However, K's mean intake decreased over time, in which the mean intake was below the recommended intake according to the Consensus Report (2019). K intake significantly decreased from 1105.43 mg in 1999 to 754.75 mg in 2015. The ratio

of Na/K increased over time, and it was above the recommended intake by WHO (2012) in 2015 with an exception of 1999 and 2001, where it was consistent and complied with the recommendations of WHO (2015). In which the mean Na/K ratio significantly increased from 0.89 in 1999 to 1.97 in 2015. The outcome of results may be explained by the change in political, social and economic factors in South Africa has resulted in increased urbanisation and progress (Van De Ende *et al*., 2014). The increased accessibility, availability and affordability of processed foods in South Africa are of concern, as these types of foods are generally considered to be high in fat, sugar or salt (Na) and low in K (Feeley and Norris, 2014; Moodley *et al*., 2015). Our study is partially in line with the study by Lee *et al,* (2013) in South Korea who reported an increase in the mean intake of Na between 1998 and 2009 (4.6 vs 4.7 g per day) and K intake increased significantly (2.6 vs 2.9 g per day). In addition, Lee et al. (2013) reported a Na/K ratio from 1.88 to 1.71. The partial contrary is with K intake and Na/K ratio, as in our study we reported a decrease in K and an increase in Na/K ratio intake over time. However, the mean intake of Na and K in the study of Lee et al. (2013) was higher than our mean intake of Na and K. It must be taken into account that the study of Lee et al. (2013) was conducted in young adults and adults whilst our study focused from childhood to adulthood. This might explain the differences in the results as well as geographical and ethnic differences.

In line with the above results, we can therefore partially accept the hypothesis.

Objective 4: To determine which of the selected micronutrients (Na, K, and Na/K ratio) will be associated on BP and abdominal indices over time by using the GEE (linear) technique.

iv. Hypothesis 4: All selective micronutrients (Na, K, and Na/K ratio) will be associated with blood pressure and abdominal indices over time by using the GEE (linear) technique.

The results of our study indicated that K was significantly associated with WHtR [β = 0.019, (95% CL: 0.004, 0.034) p-value= 0.012]. The outcome of these results could be due lack of intake of fruits and vegetables in 2015 compared to 2001 as indicated in the most frequent food table, which resulted in the decline of potassium over the years. Most

studies in this field that have been conducted over the past five years indicate that the K effect on obesity is a new topic, and more robust studies with better design are warranted (Lee *et al*., 2013), especially on abdominal obesity as previous studies mostly focused on BMI and not abdominal indices such as WC and WHtR. The precise mechanism between potassium intake and obesity/MetS is unclear. Central obesity is a component of metabolic syndrome, and the mechanisms of obesity and MetS are homogeneous. Obesity is associated with K channel function (Climent *et al*., 2014; Crunkhorn, 2013). Na/K ratio was significantly associated with both BP measurements and abdominal indices [SBP-β= 4.326, (95% CL: 2.056, 6.595) *p*-value= < 0.001], [DBP-β= 2.028, (95% CL: 0.703, 3.353) *p*-value= 0.003], [WC-β= 4.191, (95% CL: 2.080, 6.302) *p*-value= < 0.001] and [WHtR-β= 0.014, (95% CL: 0.003, 0.026) *p*-value= 0.015], which was a positive association for both Na and Na/K. Despite the lack of studies that could be compared to this study. Pereira et al. (2019) however found independent associations of Na and K intake with BP, but when evaluated in a combined manner, as in the case of the Na/K ratio, the effect was potentiated. Our results on the hand showed no relationship between Na and BP measurement and abdominal indices. Although Na significantly increased over time, the mean intake over the measured years was not above the recommended average intake. The increase was not significant to have it associated with BP measurement and abdominal indices in this sample population of Ellisras.

In line with the above-stated results, the hypothesis was therefore partially accepted

Objective 5: To determine which selective micronutrients (Na, K, and Na/K ratio) will cause the risk of developing HT and abdominal obesity.

v. Hypothesis 5: Na/K ratio will be the predictor of the risk of developing HT and abdominal obesity compared to Na and K alone

Our results revealed that the Na/K ratio is the predictor of HT and abdominal obesity. This shows that an increase in Na/K ratio will increase the risk of 1.603-folds likely to develop hypertension [Exp β= 1.603, (95% CL: 1.164, 2.207) *p*-value= 0.004] and 1.797-folds likely to developing abdominal obesity [Exp β= 1.797, (95% CL: 1.207, 2.677) *p*-value= 0.004] over time. Due to the lack of sufficient studies, we could not compare this study with relevant studies. However, Ge et al. (2015) also discovered that the urine Na/K ratio

was independently related to obesity and that a high Na/K ratio could increase the risk of obesity. However, no link was discovered between the Na/K ratio (as determined by self-reporting) and obesity (Murakami *et al*., 2015). This contradicts the findings of this study, as the Na/K ratio was calculated using self-reported dietary data in both groups. Because the topic is new, data on the relationship between Na/K and abdominal obesity is inconsistent. On the other hand, our study is consistent with the literature, which states that the Na/K ratio outperforms Na and K as individual predictors of BP change in various investigations, most of them conducted in hypertensive patients (Perez and Chang, 2014), with higher Na/K ratio may lead to higher BP during follow-up (Okayama *et al*., 2016). Elevated levels of Na intake and inadequate levels of K intake may affect the development of hypertension (WHO, 2012). To further agree with this literature, our results indicated an increase in Na over the years and a decrease in K over the years.

In line with the above results, this hypothesis was therefore accepted.

5.3. PERSPECTIVE

This highlights a positive association between K intake and abdominal index (WHtR), it further reveals that the Na/K ratio increases the risk of HT and abdominal obesity. The current study is valuable and informative regarding the status of Na and K intake in a sample of South Africans, from childhood to young adulthood. As tracking the dietary habits of children to adulthood is vital, as children with extremely high levels of sodium intake tend to maintain those levels for a time (Patterson *et al*, 2009). It further provides information on the trend of HT and abdominal obesity from childhood to young adulthood in a rural setting. A progressive increase in BP in childhood or young adulthood through middle age may predict the level of BP and the presence of hypertension later in life (Caudarella *et al*., 2009). Despite the initiative to reduce Na and emphasise the importance of potassium, few studies in Africa and South Africa have explored the trends of Na and K intake and their health effects in cross-sectional or longitudinal studies. More programmes are needed to further the importance of these selective micronutrients (Na, K and Na/K ratio), especially in children as most data in South Africa is focused on young adults and adults.

5.4. LIMITATIONS

It is important to note factors that might have influenced the finding of the current study. This includes factors such as the study design, methodology and statistical analysis. Study limitations are as follows:

- The use of the 24-h recall questionnaire compared to the use of the 24-h urinary excretion, as 24-h excretion is considered the golden standard method of obtaining data on Na and K intake in population surveys and is more accurate than the 24-h recall questionnaire (Saeid *et al*., 2018).
- The second limitation is the small sample size and not having different ethnicities from different geographical regions. Future sodium excretion data over time, from childhood to adulthood within these regional areas will be ideal for evaluating post-legislation salt intakes and the impact on public health (Swanepoela *et al*., 2014). Majority of the study conducted in Europe, Asia and North America that are similar to this study had a large sample size compared to the one presented in this study.
- Not including cofounders such as socio-economic status, energy intake, and physical activity of the sample population.
- Another limitation is that our study did not explore the association between Na, K and Na/K and BP varied according to dietary sources of Na and K.
- In addition, the gap differences between the measured years (this was due to financial constraints), might help in also collecting dietary intake on adolescents to see the effects of dietary intake on HT and abdominal obesity from childhood, adolescent stage into young adulthood.
- Insufficient/lack of relevant studies to compare and contrast with results presented in this research.

The strength of this study

- The use of longitudinal data
- To the best of the author's knowledge, this is the first study of its kind, to investigate the association between Na, K intake and Na/K ratio with the risk of

developing (BP measurement) HT and abdominal obesity (abdominal indices) from childhood to adulthood.

5.4. RECOMMENDATIONS

- Future longitudinal studies are required 24-urinary excretions to measure the association of Na, K intake and Na/K ratio with hypertension (BP measurement) and abdominal obesity (abdominal indices) from childhood to (young) adulthood in South Africa from all ethnic groups and between urban and rural areas.
- To promote interventions and monitor dietary intake, BP measurements and abdominal indices in rural areas especially in children as most children and adolescents go undiagnosed with HT.
- School educational programmes that will inform children and adolescents on the importance of dietary intake.

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ANNEXTURES

ANNEXTURE-A ETHICAL APPROVAL

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TURFLOOP RESEARCH ETHICS COMMITTEE

ETHICS CLEARANCE CERTIFICATE

MEETING: **08 June 2021**

PROJECT NUMBER: **TREC/88/2021: PG**

PROJECT:

Title:	A longitudinal investigation as the effects of sodium and potassium intake have on the development of hypertension from childhood to adulthood amongst Ellisras rural population, South Africa
Researcher:	TT Raphadu
Supervisor:	Prof KD Monyeki
Co-Supervisor/s:	N/A
School:	Molecular and Life Sciences
Degree:	Master of Science in Physiology

PROF P MASOKO
CHAIRPERSON: TURFLOOP RESEARCH ETHICS COMMITTEE
The Turfloop Research Ethics Committee (TREC) is registered with the National Health Research Ethics Council, Registration Number: **REC-0310111-031**

Note:

i) **This Ethics Clearance Certificate will be valid for one (1) year, as from the abovementioned date. Application for annual renewal (or annual review) need to be received by TREC one month before lapse of this period.**

ii) **Should any departure be contemplated from the research procedure as approved, the researcher(s) must re-submit the protocol to the committee, together with the Application for Amendment form.**

iii) **PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES.**

Finding solutions for Africa

ANNEXTURE-B CONSENT FORM

Project title: **A longitudinal investigation as the effects of sodium and potassium intake have on the development of hypertension from childhood to adulthood amongst Ellisras rural population, South Africa.**

Project leader: **Prof KD Monyeki**

Researcher: **Ms TT Raphadu**

hereby voluntarily consent to participate in the following project: “A longitudinal investigation as the effects of sodium and potassium intake have on the development of hypertension from childhood to adulthood amongst Ellisras rural population, South Africa.’’

I understand that:

1. The study deals with anthropometric measurement (weight and height), blood pressure and dietary intake

2. The procedure may hold some risk for me that cannot be foreseen at this stage.

3. The Ethics Committee has approved that individuals may be approached to participate in the study.

4. The research project, aims and methods of the research, has been explained to me.

5. I will be informed of any new information that may become available during the research that may influence my willingness to continue my participation.

6. Access to the records that pertain to my participation in the study will be restricted to persons directly involved in the research.

7. Any questions that I may have regarding the research, or related matters, will be answered by the researcher/s.

8. Participation in this research is voluntary and I can withdraw my participation at any stage.

9. If any medical problem is identified at any stage during the research, or when I am vetted for participation, such condition will be discussed with me in confidence by a qualified person and/or I will be referred to my doctor.

10. I indemnify the University of Limpopo and all persons involved with the above project from any liability that may arise from my participation in the above project or that may be related to it, for whatever reasons, including negligence on the part of the mentioned persons.

Signature of interviewee **Signature of witness**

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Signature of interviewer----------

ANNEXTURE-C 24-HOUR RECALL QUESTIONNAIRE

ELLISRAS LONGITUDINAL STUDY

Subject number: | | | | | | | Birth Date: DD MM YY | | | | | | | Interwdate: DD MM YY | | | | | | |

School name:_____ School number:_____Interviewer_____

DIETARY INTAKE QUESTIONNAIRES (24 HR RECALL)

Instructions:

Now I want you to tell me everything that you ate and drank yesterday. Lets start with when you woke up. Did you have anything to eat or drink?

- Enter each item eaten in grams under the correct interval of the day eaten.
- Make sure that the code is circled.
- Items not on the questionnaire should be looked up in the Quantity Manual or list of food codes.
- Specify fully when new items are entered and look up the code later.

ABBREVIATIONS:

Measures 1t = 1 rounded teaspoon 1T = 1 rounded tablespoon (15ml) 1SP = 1 rounded servingspoon (30ml) C = measuring cup (250ml) s/s = small size m/s = medium L/s = large E = enriched P = plain Milk: SM = skim milk WN = whole milk BL = blend CON = condensed milk ND = non-dairy	Bread Wh = white Br = Brown Ww = wholewheat Meat F = with fat FT = fat trimmed Oil/ Fat B = butter HM = hard margarine Med = medium fat/ light PM = polyunsaturated SO = sunflower oil WF = white fat PM = peanut butter	BR = breakfast (Up to 09h00) IS = in - between snack L = lunch (midday (12h00-14h00) D = dinner (evening (17h00-20h00) AD = after meal Comm = commercial Home = homemade Pot = poatato Cab = cabbage Carr = Carrot Fill = Filling Usually = at least 4x/week

	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
TEA & COFFEE	Tea: 4038; Rooibos 4054	teacup = 180ml; mug = 250ml						
	Coffee 4037	cup = 180ml; mug = 250ml						
	+ Sugar White-3989; Brown-4005;Syrup-3988;Honey-3984	1 t sugar = 6g 1 t honey/syrup = 15g						
	+ Condensed Milk: WM -2714; Cond Milk: SM-2744; Condensed Milk, Non-Dairy-P0042	1t = 10g						
	+ Evaporated WM-2715; Evaporated SM-2827; Lite-P0043	1t = 3g						
	+ Non-Dairy Creamer-2751	1t = 4g						
	+ WM Powder-2831	1t = 4g						
	+ Milk: SM-2719; WM-2718	MEDIUM PORTIONS: 20ml - tea in cup 35ml - tea in mug 40ml - coffee in cup 75ml - coffee in mug						
	BL-2771; 2%-2772							
	Soy-2737; Breast-2741; Goat-2738							
	Formula * (Specify):_____ No of Scoops/Bottle:_____							
	Other (Specify) _____							
MILK & MILK DRINKS	Buttermilk - 2713	s/s = 175ml l/s = 500ml ½c = 125g						
	Maas/Amazi/Sourmilk - 2787							
	Custard: SM-2717; WM-2716	s/s = 350 ml						
	Milk: SM-2719; WM-2718	to drink ½c = 125ml baby bottle = 250ml						
	BL-2771; 2%-2772							
	Soy-2737; Breast-2741; Goat-2738							
	* Formula (Specify):_____ No of Scoops/Bottle:_____							
	+ Sugar White-3989;Brown-4005; Syrup-3988;Honey-3984	1 t sugar = 6g 1 t honey/syrup = 15g						
	+ Ice Cream-3519; Sorbet-3491	1 scoop = 40g						
	+ Sustagen-4079; Complan-4082	2 scoops = 25g; 1T = 15g						
	+ Milo/Cocoa/Horlicks/Ovaltine-2736; Drinking Chocolate-4287	1t = 5g						
	Yoghurt: Plain SM-2734; WM-2757	s/s = 175ml Yogisip = 350ml ½c = 125g						
	Flav-2756; Fruit-2732							
	Flavoured milk - 2774	carton = 250ml s/s plastic = 350ml						
	Other (Specify) _____							
C O	Apple Juice – No Sugar – 3606	Liquifruit s/s = 250ml L/s = 500ml Ceres s/s = 200ml cartons/bottles s/s = 350ml L/s = 500ml						
	Apricot: + Sugar-3539; No Sugar-3610							
	Mango-3683; Granadilla-3680; Grape-3690							
	Orange: +Sugar-3562; No sugar-3638							
	Guava: +Sugar-3554; No Sugar-3629							
	Peach-3642; Pear-3645; Naartjie-3682							
	Cold drinks: Squash-3982	s/s bottle = 350ml L/s bottle = 500ml s/s can = 340ml						
	Mageu-4056							
	Carbonated-3981							
	Diet Cold. & Low-Cal - 3990							

* Infasoy-2808; Isomil-2796; Lactogen 1-2821; Lactogen 2-2822; Nan-2819; Pelargon-2820; Portagen-2799; Pregestimil-2800; Prosobee-2795; S26-2806; S26 Infagro-2810; SMA-2814; Similac-2797; Similac PM-2817

	Dairy Fruit Mix - 2791							
	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
	+ Sugar White-3989;Brown-4005;Syrup-3988; Honey-3984	1t = 6g						
	Syrup (undiluted)-2865; Guava Syrup-2864	1t = 5g						
	Other (Specify) _____							
BREAKFAST CEREALS	Maltabella: Soft-3241; Mabella: Soft-3437	½c = 125g						
	M/Meal: Soft: Plain-3399; Enrich-4277	1c soft = 250g						
	Stiff: Plain-3400; Enrich-4278	1c stiff = 250g						
	Crumbly: Plain-3401; Enrich-4279	1c crumbly = 140g						
	Sour Porridge: Maize with Vinegar-P0001, Maize Fermented- P0002 Mabella with Vinegar-P0003; Mabella Fermented-P0004	½c = 125g 1c = 250g						
	Oats-3239; Tastee Wheat-3240	½c = 125g						
	Corn Flakes-3243; Sugar Frosted-3374	1c = 40g						
	Honey Crunch and Muesli - 3303	½c = 65g						
	Pronutro: Great Start-3438; High Energy-3245; Wholewheat-3436	½c = 50g						
	Puffed Wheat-3325; Sweetened-3376 (Honey Smacks)	½c = 12g						
	Raisin Bran-3373; Fruit Loops-3425	Raisin Bran ½c = 45g Fruit Loops ½c = 18g						
	Special K-3322; All Bran-3242	½c = 25g						
	Rice Crispies-3252; Cocopops-3372	½c = 20g						
	Weetbix – 3244	1 = 25g						
	+ Fat: B -3479; HM-3484; Med-3531; PM-3496; WF-3516	1 t PB = 12g; 1 t marg/oil = 5g						
	Ghee-3525; PB-3485; Butro-3523; SO-3507							
	+ Sugar White-3989;Brown-4005; Syrup-3988;Honey-3984	1 t sugar = 6g 1 t honey/syrup = 15g						
	+ Cond Milk:SM-2744; Cond WM-2714;Cond ND- P0042	1t = 10g						
	+ Evap WM-2715; Evap SM-2827; Evap Light-P0043	1t = 3g						
	+ Non-Dairy Creamer-2751	1t = 4g						
	+ WM Powder-2831	1t = 4g						
	+ Milk: SM-2719; WM-2718	125g – Instant cereal 60g – porridge 180g – Pro Nutro						
	BL-2771; 2%-2772							
	Soy-2737; Breast-2741; Goat-2738							
	Formula (Specify):_____ No of Scoops/Bottle:_____							
	Other (Specify) _____							
BREAD	Bread: Comm & Home: Wh-3210	Wh + Br 10mm = 30g Ww 10mm = 35g Wh + Br 20mm = 60g Ww 20mm = 70g						
	Br-3211							
	Ww-3212							
	Cream Crackers-3230; Provita-3235; Tuc 3331; Crackers Ww-3391	Cr Cracker = 8g; Tuc = 4g; Provita = 6g						
	Maize Meal Bread - 3278	m/s = 30g; L/s = 50g						
	Muffins: Plain-3408; Bran-3407	6cm diam = 35g 8cm diam = 60g						

	Rolls: Wh-3210; Br-3211; Ww-3212	Wh round (10cm) = 30g Wh long (16cm) = 40g s/s = 50g (Roti)						
	Roti: SO-3358; HM-3357							
	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
	Rusks: Comm Wh-3364; Bran-3330	Outspan = 15g; All Bran = 30g						
	Comm Buttermilk: Wh-3329;	Wh = 35g; Ww = 30g						
	Home Buttermilk: Wh-3215; Ww-3255; Bran & Raisins-3380	Wh = 30g; Ww = 30g						
	Scones: (Wh) SM-3411; WM-3237 (Ww) SM-3412; WM-3320	6cm diam = 35g 8cm diam = 60g						
	Vetkoek: Wh-3257; Ww –3324; Dumpling-3210 (no yeast)	8cm diam = 60g						
	Other (Specify) _____							
SPREADS ON BREAD	Beef Fat-3494; Mutton Fat-3497; Lard-3495	Thin: 5; Med: 10; Thick: 15						
	Butter-3479; Butro-3523							
	Ghee-3525; WF-3516;							
	Fishpaste-3109; Liver Spread-2922; Meat Paste-2917	5 / 7 / 10						
	Jam-3985; Honey-3984; Syrup-3988	10 / 20 / 35						
	Marg: H-3484	5 / 7 / 10						
	Med-3531							
	PM-3496							
	Marmite-4030; Meat Spread (Bovril)-4029	2 / 4 / 7						
	Peanut Butter-3485; Sandwich Spread-3522; ChocSpread-P0005	5 / 10 / 20						
	Other (Specify) _____							
EGGS	Eggs: Boiled/Poached - 2867	1 egg = 50g						
	Curried - 2902	1 egg + sauce (IT) = 75g						
	Fried: B-2868; HM-2877; PM-2878	1 egg = 52g						
	SO-2869; Bacon Fat-2870							
	Scrambled/Omelette: SM + B-2886; SM + HM-2887	IT = 35g; 1SP = 80g ½c = 115g (± 2 eggs) omelette = 60g egg (med) 120g (L/s)						
	SM+PM-2888; SM+SO-2889; WM+B-2874							
	WM+HM-2890; WM+PM-2891; WM+SO-2873							
	Other (Specify) _____							
CHEESE	Cheddar-2722;	grated: med = 10g Thick = 15g 1 cheezi = 20g; cubes = 30g 1 slice = 8g						
	Gouda/Sweetmilk-2723							
	Cheese Spread-2730	med = 12g; thick = 25g						
	Cottage Cheese; Creamed-2759; Cream Cheese-2725	thin = 10g med = 20						
	Cottage Cheese: Fat Free-2729; Low Fat-2760	med = 20g; thick = 30g						
	Macaroni Cheese: SM-3343; WM-3301	1T = 45g; 1 SP = 90g; ½c = 115g						
	Pizza (Cheese + Tomato)-3353	S/s = 90g; L/s = 340g						
	Savoury Tart+Asparagus-3367;+Vienna-3326;+Tuna-3366	wedge: small = 65g; med = 75g ; large = 110g						
	Other (Specify) _____							
MEAT	Bacon: Fried: Lean-2915 F-2906	1 rasher = 10g						

	Beef:Corned/Silverside/Cold cuts:F-2924;Bully Beef-2940	138 x 85 x 3 = 20g ½c = 100g						
	Lean-2962; Curry Beef-P0006							
	Fillet: F-2933; FT-2929	100 x 70 x 10 = 90g						
	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
M E	Mince: Pan Fried F-2910; Lean-2961; Curry-3015	T = 40; SP = 85g ½c = 100g						
	- Savoury (Tomato + Onion)-2987							
	- Cottage Pie: WM + HM-3009							
	Roast: F-2944; FT-2960	120 x 60 x 5 = 35g 120 x 60 x 10 = 70g						
	Rump: Fried: F-2908; FT-2959	S/s 130 x 70 x 15 = 125g L/s 165 x 70 x 30 = 270g						
	Sirlion/T-Bone: Grilled: F-2946; FT-2907							
	Stew: Vegetables (Fat Meat)-3006	1 SP = 105g; ½c = 125g						
	: Pot + Carrots + Peas + Onions (Lean Meat)-2909							
	Biltong: Beef-2911; Game-2912	grated 1SP = 10g beefeater = 18g sliced 1SP = 35g						
	Bobotie: Lean, SM, SO-3013; F, WM, S0-2986	1SP = 85g; ½c = 115g						
	Chicken: Boiled + Skin-2926; No Skin-2963; Curry-P0007	breast + skin = 125g thigh = 80g drumstick = 42g foot = 30g wing = 30g pie(comm)=150g home = 90g liver = 30g; stomach = 20g						
	Feet-2997; Giblets-2998; Heads-2999							
	Pie (Comm)-2954							
	Roast + Skin-2925; No Skin-2950; Fried-2925							
	Stew: Vegetables-3005	1SP = 90g; ½c = 125g						
	Tomato + Onion – 2985							
	Batter Dipped-Fried eg. Kentucky-3018	1SP = 105g; ½c = 125g						
	Burger Pattie –2950	1 pattie = 80g						
	+ Bun (4 cm diam)-3210	1 bun = 60g						
	Cornish Pie: (Comm) - 2953	med = 150g						
	Frankfurter-2937	155 x 20 = 45g 168 x 21 = 60g						
	+ Roll (16 cm long)-3210	1 roll = 40g						
	Goat meat: Stewed (plain)-4281; (+ Veg)-4282	120 x 60 x 5 = 35g 120 x 60 x 10 = 70g						
	Fried F-P0008; Fried FT-P0009							
	Grilled F-P0010; Grilled FT-P0011							
	Ham-2967; Ham & Tongue loaf-2990	med slice = 25g						
	Heart: Beef-2968; Sheep-2969	sheep heart = 60g sheep kidney = 30g beef kidney = 85g						
	Kidney: Beef-2923; Sheep-2956							
	Lung: Beef-3019							
	Lasagne: SM-3440; WM-3261	T = 40g; SP = 75g; ½c = 120g						
	Liver: Fried : Beef-2920; Sheep-2955; Patty (Fried) -2971	sheep = 55g chicken = 30g beef = 80g						
	Cooked: Chicken-2970							
	Meat Ball: F + Egg-2965; F-No Egg-2966	50mm = 60; 75mm = 120g						
	Lean + Egg-3033; Lean, No Egg-3034							
	Meat Loaf: F-3035; Lean-3002	80 x 85 x 15mm slice = 80g						

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	Meat Patty: (Hamburger)-2984	s/s = 50g; m/s = 100g						
	+ Bun (4 cm diam)-3210	1 bun = 60g						
	Mutton: Chop (grilled) F-2927; FT-2934	loin chop = 60g rib chop = 40g						
	Roast: F-2947; FT-2973	s/s slice = 30g med = 70g						
	FOOD ITEMS	**QUANTITY (g/ml)**	**BR**	**IS**	**L**	**IS**	**D**	**AD**
MEAT	Stew: Plain-2974; Irish-2916 (Vegetables) Curry-3039; Greenbean-3040	1SP = 105g; ½c = 125g						
	Offal: Cooked-Tripe(Pens&Pootjies)-2951;Vetderm-P0023 (Specify): _____	1SP = 105g; ½c = 125g						
	Oxtail: Stewed-2976							
	Polony-2919	slice 5mm thick = 8g comm slice = 16g						
	Pork: Chop (Grilled) F-2930; FT-2977	chop: 115 x 80 x 20 = 100g schnitzel: 115 x 80 x 20 =110g roast: 110 x 65 x 5 = 30g 1SP = 105g; ½c = 125g 3 ribs = 130g						
	Crumbed-2992; Spareribs-3010							
	Rib, Braised: F-3046; FT-3045							
	Roast: F-2958; FT-2978							
	Salami and Russians-2948	slice 5mm thick = 12g 1 Russian = 50g						
	+ Roll-3210	1 roll = 40g						
	Samoosa: with Veg-3414; Meat-3355	s/s = 42g						
	Sausage: Beef: Dry-2949; Cooked-2931 (Boerewors)	thin x 200mm = 45g thick x 165mm = 90g						
	+ Roll-3210	1 roll = 40g						
	Pork: Cooked-2932	med = 55g						
	+ Roll-3210	1 roll = 40g						
	Roll/Meat Pie (Comm)-2939	25mm pie = 120g roll x 135mm = 165g						
	Spaghetti Bolognaise: Lean-3388; F-3260	T=40g; SP = 75g; ½c = 100g						
	Steak & Kidney: Pie-2957; Stew-2979	comm pie = 120g (30mm) 1SP = 100g; ½c = 135g						
	Tongue: Ox-2935; Sheep:2980	slice 75 x 45 x 10 = 40g						
	Toppers/Imana: Cooked-3196	SP = 85g; ½c = 120g						
	Veal: Cutlet (Fried): Plain-3049; Crumbed-2983	1 chop = 90g						
	Vienna Sausage/Canned Sausage-2936	100mm = 30g; 150mm = 40g						
	+ Roll-3210	1 roll = 40g						
	Worms/Insects:Mopani,Dried-4250;Mopani,Canned-4284; Specify: _____							
	Wild Birds, Animals; Specify: _____							
	Other (Specify) _____							
FISH	Bokkems (Dry Fish)-3097	1 s/s = 25g (120mm) L/s = 40g (135mm)						
	Fatty Fish: Kipper; Galjoen; Snoek; Shad: Fried (SO)-3084; Batter-3094; Grill-3082	small 50 x 55 x 30 = 60g med 100 x 55 x 30 = 120g stew 1 SP = 95; ½c = 140g						
	Salted-3097; Steam-3103; Smoked-3112							
	Curried Stew-3076 (Tomato and Onion) / Pickled /							

	Fish Cakes: (Fried): Home-3098; Comm-3080	65 x 15mm = 50g						
	Fish Fingers: (Fried)-3081	85mm = 35g						
	Haddock: Smoked (Boiled)-3061	70 x 70 x 15 = 65g						
	Mackerel Canned-3113	1 = 80g (15 mm)						
	Pilchards: Tomato Sauce-3102; Brine-3055	1 = 75g						
	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
	Sardines: + Sauce-3087; + Oil-3104	s/s = 7g; L/s = 25g						
	Smoorsnoek-3074	1SP = 55g; ½c = 80g						
	Sole: Fried-3090; Grilled-3073	baby sole: 180mm = 70g						
	Tuna: Oil Pack-3093; Tuna: Water-3054; Salmon-3058	¼c = 50g						
	White Fish: Hake, Haddock, Kingklip; Cod	s/s piece 50 x 55 x 30 = 60g med 100 x 55 x 30 = 120g stew 1 SP = 95g; ½c = 140g						
	: Stew-3076 (Tom + On); Baked+Fat-3092; No Fat-3089							
	: Grilled-3079; Batter-3072; Fried-3060							
	Other: eg Fresh Water Fish; Specify:_____ P0012							
	Other (Specify) _____							
STARCH	M/Meal: Soft: Plain-3399; Enrich-4277	T SP ½c						
	Stiff: Plain-3400; Enrich-4278	stiff 75 120 125						
	Crumbly: Plain-3401; Enrich-4279	crum 30 75 70						
	Mabella Cornrice/Sorghum cooked (soft or stiff)-3437	soft 75 120 125						
	Sour Porridge: Maize & Vinegar-P0001, Fermented-P0002							
	Mabella with Vinegar-P0003; Fermented-P0004							
	Maize Rice (Mealie Rice)-3250	25 45 65						
	Samp: (Cooked) –3250; Fresh Mealies-3725	55 125 125						
	Rice: Wh-3247; Br-3315	25 60 65						
	Spaghetti/Macaroni: (Cooked)-3262	35 70 90						
	Spaghetti + Tomato Sauce -3258	45 80 125						
	Stamped Wheat/Wheat Rice-3249	30 80 80						
	+ Fat: B -3479; HM-3484; Med-3531; PM-3496; WF-3516	1 t PB = 12g; 1 t marg/oil = 5g						
	Ghee-3525; PB-3485; Butro-3523; SO-3507							
	Other (Specify) _____							
LEGUMES	Baked Beans-3176	T SP ½ c						
		50 105 135						
	Beans: (Cooked) Haricot-3185; Sugar-3205; Kidney-3183	50 85 135						
	Breyani: Rice + Lentils + Ghee-3194; +SO-3193	40 80 85						
	Lentils: Cooked/curried-3179	40 80 90						
	Samp and Beans (1:1)-3402; Comm-P0045 (No fat added)	50 125 125						
	Samp & Peanuts (80:20) P0013							
	Soup: Comm (Packets)-3165	125						
	Split Pea-3157; Lentil-3153; Beef + Veg-3159; Bean-3145	35 80 130						
	Sousboontjies (Dried Bean Salad)-3174	40 105 135						

	Stew: Bean + Potato + Onion-3178						60	120	125								
	Other (Specify) _____																
COOKED VEGETABLES		Boil	Fat Added (or Fried)														
		NF	B	HM	PM	SO	T	SP	½C								
	Gr Beans	3696		3788	3789		25	60	80								
	Gr Bean Curry	3791					40	75	120								
	GrBean+Pot+Onion			3792		3794											
	FOOD ITEMS						QUANTITY (g/ml)			BR	IS	L	IS	D	AD		
COOK	Beetroot + Sugar	3699					40	70	80								
	- No Sugar	3698															
	Brinjal	3700		3800		3802	1 slice = 20g (70mm) + batter = 30g										
	- Fried + Egg					3803											
	- + Tomato + Onion			3796		3798	50	100	130								
	Brocolli	3701		3805			25	60	75								
	Brussels Sprouts	3703		3808			50										
		Boil	Fat Added (or Fried)														
		NF	B	HM	PM	SO	T	SP	½C								
	Cabbage	3756		3810		3812	30	55	80								
	Cab + Pot + Onion			3813		3815	35	75	80								
	Carrots	3757		3816	3817		20	50	80								
	Car + Pot + Onion			3822		3824	35	70	105								
	Carrot + Sugar	3818		3819	3820		25	50	85								
	Cauliflower	3716		3825	3826		40	65	80								
	Caul + Cheese Sauce	3715					43	70	90								
	Marogo/imifino* Amaranth leaves	3980					40	105	90								
	Marog + Peanuts Ratio: 80:20	P0014					55	120	105								
	Mealies (corn)	3725					30	60	95								
	Sweetcorn	3726					55	125	135								
	Canned Whole Kernel	3942					55	125	135								
	Mix Veg (Froz)	3727		3835	3836	4269	35	75	75								
	Mushroom (Sliced)	3729		3839		3841	30	65	80								
	Mushroom, Raw					3842	30	65	80								
	Onions (Sliced)	3773		3844		3730	50										
	Onion + Batter					3846	rings: med = 40g										
	Peas	3719		3856			30	65	85								
	Peas, Frozen	4146					30	65	85								
	Peas + Sugar	3720		3859			30	65	85								
	Potato: + Skin	4155					s/s = 60; m/s = 90g										
	: Baked + Skin	3736					s/s = 60g; m/s = 90g										
	: Chips					3740	½c = 50g; med = 80g										

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: Peeled	3737		3867	3868		s/s = 60g; m/s = 90g; (90 x 60 x 40)									
: Sauté			3871		3873	3	50	90							
Potato Cake					3915	1 med = 40g (75 x 30)									
Potato Mash (SM)				3875		50	115	125							
Potato Mash (WM)			3876												
Potato (Roast):Beef Fat-3878; Chicken-3923; Lamb-3736; Pork-3956						1 med = 90g									
* If indigenous, specify local name: _____															

	FOOD ITEMS						QUANTITY (g/ml)			BR	IS	L	IS	D	AD
		Boil	Fat Added (or Fried)												
		NF	B	HM	PM	SO	T	SP	½c						
COOKED VEGETABLES	Pumpkin (Yellow)	4164					45	85	105						
	Butternut	3759													
	Pump + Sugar	3728		3893											
	Pump Fritter					3784	75 x 50 x 9 = 25g								
	Spinach	3913		3898	3899		40	105	90						
	Spinach + Peanuts Ratio: 80:20	P0015					55	120	105						
	Spin + Pot + Onion			3901		3786	50	105	110						
	Squash –Gem	3760					½ gem = 45g 1 SP marrow = 85g								
	Gem Squash + Sugar	3754													
	Squash –Marrow	4179													
	Marrow + Sugar			3885											
	Sw Potato:without skin	3903					50	110	145						
	Sw Potato with Skin	3748													
	Sw Pot + Sugar			3749											
	Tomato + Onion	3925					35	75	140						
	Tom + Onion +Sugar	3910													
	Tomato			3908		3767	1 slice 5mm = 15g (thin); med = 25g								
	Turnips	3911					25	45	90						
	Other (Specify)														
SALAD	Asparagus-3695						med asparagus = 15g								
	Avocado-3656						¼ avo (80 x 50mm) = 40g								
	Beetroot (Grated) + Sugar-3699						1T = 25g; SP = 65g								
	Carrot: (Grated)+ Sugar-3721						1T = 25g;								
	+ Pine + Orange - 3710; + Orange Juice = 3711						1T = 35g; 1SP = 60g								
	Coleslaw + Mayonnaise-3705						T = 20g; SP = 40g; ½c = 50g								

Cucumber Raw/Pickled-3718	med slice = 10g; thick = 15g						
Lettuce-3723	1 med leaf = 30g						
Mixed (Tom + Cucum + Lett) - No Dressing-3921	1T = 40g; 1SP = 85g						
Mixed Green - No Dressing-3927							
Potato Salad + Mayonnaise (Comm), Egg-3928	T = 45g; 1SP = 105g; ½c = 120g						
Tomato (Raw)-3750	med = 120g; slice = 15g						
Other (Specify) _____							

	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
DRESSINGS	French Dressing-3487	1t = 5g; 1T = 15g						
	Mayonnaise: Home-3506; Comm-3488; Low Fat- 3489	1t = 10g 1T = 40g						
	Oil: Olive-3509; Sunflower-3507; Canola-4280	1t = 5g; 1T = 15g						
	Salad Dressing: Cooked-3503; Low-Oil-3505							

FRUIT	Canned + Sugar	Raw	Dry	Stewed							
Apple	3599	3532	3600	3603	1T = 60g; ½c = 120g; 1 med = 150g (52 x 66)						
Apricot	3535	3534	3536	3537	1 med = 35g						
Banana		3540			1 med = 75g						
Dates		3543			1 med = 10g						
Figs		3544	3557		1 med = 40g (45 x 44) 1 dry = 20g						
Fruit Salad	3580	3605	3593	3590	½c = 110g (med)						
Granadilla		3545			1 med = 22g						
Grape Fruit	3547	3546			½ med = 125g						
Grapes	3623	3550			med bunch = 230g; ½c =90g						
Guava	3553	3551			med (6cm) = 95g						
Litchi	3631	3632			med (3cm) = 8g						
Mango	3633	3556			135mm = 350g						
Naartjie	3635	3558			med = (5cm) = 75g						
Orange		3560			med (7cm) = 180g						
Pawpaw		3563			wedge 165 x 26 x 27 = 90g						
Peach	3567	3565	3568	3569	1 med = 150g (60 x 65)						
Pear	3583	3582	3585	3586	1 med (80 x 65mm) = 165g						
Pineapple	3648	3581			1 slice (85 x 10mm) = 40g						
Plum		3570			1 med = 50g (45 x 40)						
Prunes	3676	4230	3596	3564	1T = 50g; ½c = 110g; 1 = 12g						
Raisins		3552			handfull = 27g						
Strawberries	3653	3573			1 med = 12g; ½c = 80g						

	Sweetmelon, Green		3575			1 wedge (145 x 31 x 20mm) = 60g; ¼ = 110g						
	Sweetmelon, Yellow		3541									
	Watermelon		3576			slice (330 x 70mm) = 220g						
	Wild Fruit, Berries: Specify: _____											
	Other Fruit:											

	FOOD ITEMS			QUANTITY (g/ml)	BR	IS	L	IS	D	AD
		SM	WM							
PUDDINGS	Apple + Batter	3345	3327	med serving = 70g						
	Apple Crumble		3334	med serving = 70g						
	Baked Pudd + Syrup	3348	3312	med serving = 30g 30 x 65 x 65 = 50g						
	- No Syrup	3347	3221							
	Blancmange	3282	3281	SP = 75; ½c = 95g						
	Egg Type eg. Bread, Sago	3346	3263	1T = 50g; ½c = 140g; SP = 100g						
	Ice Cream: Commercial Regular-3483			scoop = 40g; 1SP = 65g; ½c = 75g						
	Commercial Rich-3519									
	Ice Lollies-3982									
	Soft Serve-3518			plain = 135g; + flake = 155g						
	Sorbet-3491			1SP = 65g; ½c = 75g						
	Instant Pudding	3314	3266	T = 45g; SP = 95g; ½c = 145g						
	Jelly-3983			1T = 35g; 1SP = 75g; ½c = 110g						
	Jelly + Fruit-4006			1T = 40g; 1SP = 90g; ½c = 125g						
	Jelly Whip	2749	2750	1T = 55g; SP = 95g; ½c = 120g						
	Pancake/Crumpets	3344	3238	1 crumpet = 25g pancake = 70g						
	Trifle-3311; Vermicelli Pudding-3385			½c = 130g (med)						
	Other Puddings (Specify) _____									
SA UC	Cream: Plant-3492; Canned-3499			1T = 13g (not whipped)						
	- Fresh (12%) -3481; Heavy (dessert, 20%)-3480			1T = 30g (whipped)						
	Chocolate Sauce-3129			T = 15g						
	Custard: SM-2717; WM-2716			T = 13g; SP = 40g						

	Sugar-3989	1t = 6g						
	Other (Specify) _____							
CAKE	Banana Loaf: WM + HM-3333; SM + PM-3370	slice = 45g; 90 x 80 x 10mm						
	Cake –Carrot-3392	80 x 40 x 40 = 50g						
	- Plain: SM + HM-3286; PM-3287	single slice = 50g (75 x 75 x 20mm) double slice = 100g (plain) icing = 10g per slice						
	WM + B-3218; HM-3288; SO-3290							
	Cake Icing: HM-4014; PM-4015							
	- Chocolate (No Icing) WM-3289; SM-3339							
	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
CAKE	- Fruit: Comm-3291; Home-3427	home: 70 x 85 x 15mm = 70g comm: 90 x 70 x 15mm = 35g						
	- Sponge (Plain)-3219	100 x 50 x 50 = 40g						
	- Swiss Roll-3292	slice = 60g; 15cm thick						
	Cheese Cake: Baked-3293; Unbaked-3294	slice 95 x 50 x 30mm = 70g						
	Other (Specify) _____							
COOKIES & SPECIAL BREADS	Comm + Fill-3217; Plain-3216; Shortbread-3296	plain = 10g + fill = 15g						
	Home: Plain HM-3233; PM-3341	plain = 15g + fill = 20g hertzog = 50g; cupcake = 35g shortbread = 12g						
	Jam-3295; Oats-3265							
	Custard Slice-3338	110 x 45 x 35mm = 250g						
	Date Loaf; HM-3256; PM-3340	slice 90 x 75 x 10mm = 40g						
	Doughnuts: Jam-3423; Plain-3232	med round = 45g med long = 90g						
	Eclairs + Cream + Chocolate-3268	1 = 120g (160mm)						
	Gingerbread: HM-3253; PM-3371	90 x 75 x 15 = 70g						
	Koeksister-3231	100 x 35 = 60g						
	Pumpernickel Bread-3283	slice 85 x 100 x 10mm = 30g						
	Raisin Bread-3214	slice 85 x 100 x 10mm = 30g						
	Rye Bread-3213	slice 85 x 100 x 10mm = 30g						
	Sweetcorn Bread-3379	slice 85 x 100 x 10mm = 30g						
	Other (Specify) _____							
	Apple: HM-3224; PM-3352	50 x 50 x 50mm = 70g (med)						
	Coconut-3228	wedge 50 x 100 x 30mm= 55g						
	Condensed: HM-3294; PM-3439	95 x 70 x 30mm = 90g						
	Fridge (Fruit): HM-3394; PM-3434							
	Lemon Meringue: HM-3226; PM-3349	100 x 70 x 35mm = 75g						
	Milk (Short) WM + HM-3360; SM + PM-3351	120 x 70 x25mm = 75g						
	Milk (Flaky) WM + B-3443; WM + HM-3229							

	Savoury: Aspar-3367; Tuna-3366; Vienna-3326	120 x 50 x 25 = 75g						
	Tipsy: HM-3323; Jam-3225	87 x 70 x 50mm = 90g						
	Other (Specify) _____							

	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
SWEETS	Bubble/Chewing gum-3993	See Manual						
	Chocolates: Assorted-3992							
	Coated Bars eg. Tex, Lunch, Chomp-3997							
	Milk (White Chocolate)-3987							
	Nuts/Raisins-3994							
	Plain eg Smarties, Flake, Aero-4003							
	Dry Fruit Sweets-3995							
	Fruit Gums-4000							
	Hard/Jelly Sweets eg. Sugus, Jelly Tots, Fruit Drops-3986							
	Ice Lollies-3982							
	Marshmallows-4001							
	Meringues-4008							
	Peanuts: Raw-4285; Peanut Brittle-4002;							
	Roasted, Salted-3458; Roasted Unsalted-3452							
	Peppermints-4004							
	Popcorn: Plain-3332; Sugar Coated-3359							
	Potato Crisps eg. Simba, O=Gradys-3417							
	Raisins, Seedless-4232							
	Snacks – Fritos, Niknaks, Cheese Curls-3267							
	Soft Sweets - Fudge, Toffees, Caramel-3991							
	Other (Specify) _____							
O H	Cheese Sauce: WM + HM-3125; SM + PM-3128	SP = 65g; 1T = 25g						
	Curry Sauce-3130	1T = 25g						
	Chutney-3168; Atjar-3117; Tomato Chutney-3114	1T = 14g; 1T = 60g						
	Gravy: Comm-3119; Meat-3122; NF-3121	1T = 15g; SP = 35g						
	Mustard-4034	1t = 6g						

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	Pickles-3866	1 = 10g						
	Tomato Sauce (Comm)-3139	1t = 6g; 1T = 25g						
	White Sauce: WM + HM-3142; SM + PM = 3141							

	FOOD ITEMS	QUANTITY (g/ml)	BR	IS	L	IS	D	AD
INFANT FOODS	Baby Cereals (dry): Nestum 1-2832; Nestum 2-2834	1t = 2g 1T = 8g ½c = 20g						
	Purity:Mixed-2842;Wholewheat-2861; Rice-2862							
	Cerelac-2836; Nestum Rice & Maize-2835							
	Junior-2833							
	Milk: SM-2719; WM-2718	to drink ½c = 125ml baby bottle = 250ml						
	BL-2771; 2%-2772							
	Soy-2737; Breast-2741; Goat-2738							
	Formula (Specify):_____ No of Scoops/Bottle: _____							
	+ Sugar, White-3989;Brown-4005;Syrup-3988;Honey-3984	1t = 6g						
	First Food Fruit-2852; First Food Veg-2851	jar = 80g; 1t = 11g						
	Fruit Juice (Strained)-2860; Fruit Juice-2866	½c = 125ml						
	Infant Dinners (Dry): Beef + Veg-2841; Chicken+Veg-2840	1t = 5g 1T = 15g ½c = 47g						
	Guava + Custard-2837; Mix Veg-2839							
	Orange + Banana-2838							
	Junior Food (Jar): Veg + Meat-2848; Mix Veg-2849; Pasta + Beef-2850	jar = 200g 1t = 11g ½c = 125g						
	Junior Fruit (Jar): Fruit-2863; Guava-2855							
	Junior Pudding: Fruit+Yog-2858; Vanilla Cust-2859							
	Strained Food (Jar): Macaroni Beef-2845; Veg+Meat-2846	jar = 125g 1t = 11g ½c = 125g						
	Fruit + Yog-2857; Fruit-2854;							
	Av. Pudding-2844; Meat Soup-2847;							
	Veg Soup-2843; Vegetables-2853;							
	Junior Fruit Guava-2856							
OH								

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9. Did this child go to bed hungry last night?	1 Yes	2 No	3 Don't Know
10. Did this child eat from the same pot as the rest of the family at the main meal yesterday?	1 Yes	2 No	3 Don't Know
11. Did this child eat from the same plate as the siblings, at the main meal yesterday?	1 Yes	2 No	3 Don't Know

A. SCHOOL/CRECHé FEEDING SCHEME

3. Name of School/Creché: _____

4. Address: _____

_____

5. Telephone: _____

6. Person to Contact: _____

7. Composition of the Meals/Supplements

ITEMS	CODE	AMOUNT (g)
i)		
ii)		
iii)		

Description:

_____

_____

_____

B. ADD ADDITIONAL ITEMS EATEN TO THE 24-HR RECALL QUESTIONNAIRE

RECIPES

NAME OF DISH	INGREDIENTS	CODES	AMOUNT (g) OR (mg)

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Peer-reviewed articles derived from this research project

Raphadu, T.T., Matshipi, M., Mphekgwana, P.M. and Monyeki, K.D. 2022. Assessing the Association of Sodium, Potassium Intake and Sodium/Potassium Ratio on Blood Pressure and Central Adiposity Measurements amongst Ellisras Undernourished, Rural Children Aged 5–13 Years: South Africa. *Children*, *9*: **422**: https://doi.org/10.3390/children9030422.

Raphadu, T.T., Mphekgwana, P.M., Matshipi, M. and Monyeki, K.D. (Year). Investigating the association of sodium, potassium intake and sodium-to-potassium ratio on the development of hypertension and abdominal obesity over time in Ellisras. *Public Health Nutrition*, **volume**: page no/doi.

Peer-reviewed article 1:

children

MDPI

Article

Assessing the Association of Sodium, Potassium Intake and Sodium/Potassium Ratio on Blood Pressure and Central Adiposity Measurements amongst Ellisras Undernourished, Rural Children Aged 5–13 Years: South Africa

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Abstract: Background: Numerous studies have shown how diet, such as sodium (Na) and potassium (K) intake, is an important risk factor for non-communicable diseases (NCDs). This study aimed to assess the relationship between sodium intake, potassium intake; and sodium/potassium ratio with blood pressure (BP) and abdominal obesity amongst Ellisras rural children. Method: In this cross-sectional study, data on dietary intake of sodium and potassium were collected using a 24-h recall questionnaire from a total of 765 participants, aged 5–13 years. Blood pressure and anthropometric measurements were also collected. Generalised linear models and Pearson correlation were conducted to assess the association of sodium intake, potassium intake; and their ratio with BP, waist circumference (WC), and waist-to-height ratio (WHtR). Results: In both age groups, less than 14.9% of males and 19.8% of females consumed above the recommended adequate intake (AI) of sodium. In addition, both age groups had more than 90% of males and females who consumed below the recommended AI of potassium. Moreover, the sodium/potassium ratio was above the WHO recommended level in more than 30% of males and females. The study found a significant, weak positive correlation of sodium intake with systolic BP (SBP), diastolic BP (DBP), and with WHtR. A significant, weak positive correlation was also found between sodium/potassium ratio and WHtR. In addition, a significant association was found between potassium intake and systolic BP. Conclusion: Although our study found a notable low average intake of sodium and potassium as compared to the recommended values. There was positive correlation found between sodium intake and BP. Furthermore, a positive correlation of sodium intake and sodium/potassium ratio with WHtR was also found.

Keywords: sodium; potassium; sodium/potassium ratio

Citation: Raphadu, T.T.; Matshipi, M.; Mphekgwana, P.M.; Monyeki, K.D. Assessing the Association of Sodium, Potassium Intake and Sodium/Potassium Ratio on Blood Pressure and Central Adiposity Measurements amongst Ellisras Undernourished, Rural Children Aged 5–13 Years: South Africa. *Children* **2022**, *9*, 422. https://doi.org/10.3390/children9030422

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1. Introduction

World Health Organisation (WHO) has reported that non-communicable diseases (NCDs) are the main contributors to mortality and morbidity in the world [1], killing 41 million each year, which is equivalent to 71% of deaths [2]. Evidence reported by various researchers has shown diet as an important risk factor for NCDs. Furthermore, the prevalence of NCDs has greatly and continuously increased over the years [3,4]. Non-communicable diseases that can be predicted by high levels of sodium and low level of potassium are hypertension, obesity, cardiovascular diseases (CDVs), cancer, and osteoporosis [4–6]. Although these diseases are more prevalent in adults, there has been a growing concern as these diseases continue to be detected in children and adolescents [7,8].

A progressive increase in BP in childhood or young adulthood through middle age, may predict the level of BP and the presence of hypertension later in life [5]. Elevated levels

of sodium intake and inadequate levels of potassium intake may affect the development of hypertension. High consumption of potassium, commonly from fruits and vegetables, such as bananas, potatoes, and spinach, can counteract the negative effects of high sodium intake on BP [1]. Dietary habits developed from childhood into adulthood have indicated that children with an extreme level of sodium and lower potassium intake tend to maintain those levels over time [8]. Although the association of sodium and potassium intake has only been shown concerning BP, recently there have been few studies suggesting a link regarding sodium intake in relation to body weight in children and adolescents independent of total energy intake [9–12]. However, the collective effect of dietary sodium and potassium intake in the pathogenesis of other conditions, including overweight and obesity, especially in children; is less known [13]. Moreover, there is a crucial need to educate the public to keep a low dietary sodium intake and adequate level of potassium in children as diet in childhood can play a significant role in determining adult dietary habits [14].

Institutions worldwide have designed dietary guidelines in their national food and nutrition policies [15,16]. These guidelines are designed to curb the increase in NCDs by reducing sodium intake and increasing potassium intake in the population, as it has been identified as one of the most cost-effective strategies to reduce NCDs [4]. A study by He et al. (2008) showed that minimising the source of sodium and increasing the source of potassium may lead to reducing the health complications associated with BP [14]. As a means to maintain a healthy level of BP over the progression of life, there are strong initiatives that tried to find a way to reduce sodium intake among children, mainly in Europe and North America [17–19]. Despite the initiative to reduce sodium and emphasise the importance of potassium, few studies in Africa and South Africa have explored the trends of sodium and potassium intake and their health effects in cross-sectional or longitudinal studies. Studies conducted in South Africa and Africa on sodium and potassium intake have consistently focused on adults, and not on children [15]. These studies have shown high content of sodium and low content of potassium. In addition, data from studies conducted in children are limited, especially those assessing both sodium and potassium intake; and their ratio in health outcomes [1]. Currently there are no studies in South Africa that have looked at the effect of sodium intake, potassium intake and sodium/potassium ratio on hypertension and obesity in children.

Undernutrition is one of the most important public health issue, which has a prevalence of more 900 million people around the world. It is responsible for the highest death rate in children and has long-lasting physiological effects, which includes increased susceptibility to fat accumulation mostly in central region of the body and hypertension [20]. A previous study by Van Den Ende et al. (2014) focused on the relationship between dietary intake and body mass index (BMI) among the same sample (children), whilst Mashiane et al. (2018) focused on the same sample when they were young adults in Ellisras [21,22]. Van Den Ende et al. (2014) reported a high prevalence of underweight (undernourished) amongst the same sample [21]. However, Mashiane et al. (2018) revealed a high prevalence of obesity and overweight as the Ellisras sample grow older [22]. The increase in overweight and obesity in populations where historical undernutrition prevailed is often characterised by a pattern of nutrition transition [23]. However, this study is a cross-sectional study which focused on investigating the relationship of daily intake of sodium, potassium intake, and the daily dietary sodium/potassium ratio with blood pressure and central adiposity measurements amongst Ellisras rural children.

2. Method and Materials

2.1. Sampling Procedure

This study is part of the ongoing Ellisras longitudinal study (ELS), of which the details of the geographical area were reported elsewhere [24]. The ELS initially followed a cluster sampling method. Briefly, the study was undertaken at 22 schools (10 pre-school and 12 primary schools) randomly selected from 68 schools within the Ellisras area [24]. A baseline data that were collected in 1996, with follow-up exams of dietary intake, BP and

anthropometric measurements from 1999 [24]. However, this study deployed a cross-sectional study design in which information on dietary intake was collected from Ellisras in 1999. A total of 765 participants (394 males and 371 females), aged 5–13 years participated in this study.

2.2. Exclusion Criteria

Any participant with the following was excluded from this study:
Participants who failed to provide a signed consent form before measurements.

2.3. Dietary Intake

Data on diet were collected using a 24-h recall questionnaire [25]. Trained ELS field workers interviewed participants regarding their dietary intake over the past 24 h. Parents/caregivers of all the participants were interviewed regarding the dietary intake over the previous 24 h. The estimated portion size of foods consumed were recorded in as much detail as possible, using a pre-tested questionnaire and food models simulating average portions of local foods. An average of two days of dietary intake was taken for each participant. One dietary intake was collected during the weekday and another dietary intake for the weekend. This is because food consumption during the weekdays differs from the food consumed during the weekend. People tend to eat a lot more during the weekend than during the week, especially on a Saturday [26]. The average of sodium intake and potassium intake were analysed using local food tables and the South African Food Composition Database System (SAFOODS) [27] and were compared with the recommended intakes for sodium and potassium as described by Consensus Study Report (2019) (Table 1) [28]. The WHO recommends a sodium/potassium ratio of 1:1 (or ≤1) to assess the average molar sodium/potassium ratio [1]. Since the recommended sodium/potassium ratio is in moles, the sodium and potassium intake in this study were converted from milligrams to milli-moles. The following conversion was used to calculate the average molar sodium/potassium ratio [29]:

23 mg sodium = 1 mmol sodium;
39 mg potassium = 1 mmol potassium.

Table 1. Classification of variables according to sex and age.

Variables	Life Stage Group (Years)			
	Male 4–8 Years	**Female 4–8 Years**	**Male 9–13 Years**	**Female 9–13 Years**
Na adequate intake (mg/d)	≤1000	≤1000	≤1200	≤1200
K adequate intake (mg/d)	≥2300	≥2300	≥2500	≥2300
Na/K ratio	≤1	≤1	≤1	≤1
Hypertension (systolic and/or diastolic)	>95th percentile	>95th percentile	>95th percentile	>95th percentile
Abdominal obesity according to WC	≥90th percentile	≥90th percentile	≥90th percentile	≥90th percentile
Abdominal obesity according to WHtR	≥0.5	≥0.5	≥0.5	≥0.5

2.4. Anthropometric and Blood Pressure Measurements

Blood pressure was measured using an electronic Micronta monitoring kit, at least three blood pressure readings were taken after the participant had been seated for 5 min, as described by the National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents [30]. The cut–off points for high blood pressure or hypertension as described by National Heart, Lung, and Blood Institution (NHLBI) [31] was utilised for age, sex, and height. Blood pressure was calculated using the Merck manual-medical calculator for both boys and girls separately (Merck manual, 2018) (Table 1). Weight, height, and waist circumference were measured according to standard

procedures of the International Society for the Advancement of Kinanthropometry (Norton and Olds, 1996) [32]. The waist-to-height ratio was calculated as WC divided by height in centimetres. Abdominal obesity was defined as waist circumference and waist-to-height ratio (Table 1).

2.5. Statistical Analysis

IBM SPSS Statistics software package (version 26.0) was used to conduct statistical analysis. To describe and characterise the samples, descriptive statistics were calculated for all the variables to indicate frequencies (expressed as percentages) and median. All the variables were stratified by age groups and sex. Shapiro–Wilk test was used to assess variables normality. Parametric (one-way ANOVA), Chi-square, and Fisher's exact tests were conducted to determine the difference between males and females, stratified by age groups (4–8 years and 9–13 years) in all variables. Generalised linear models and Pearson correlation were conducted to assess the association of sodium intake, potassium intake; and their ratio with BP, WC and WHtR. The probability value for statistical significance for all tests was set at a p-value ≤ 0.05.

3. Ethical Clearance

Ethical clearance was granted by the Turfloop Research Ethics Committee (TREC) of the University of Limpopo before the study was conducted. This study is part of the ongoing ELS that started in 1996 with ethical clearance number MREC/P/204/2013:IR.

4. Results

4.1. Characteristics of the Population

This study constitutes 765 participants (394 males and 371 females). In the age group 4–8 years, the mean values of sodium intake were 507.34 mg/d for males and 553.70 mg/d for females. The mean values of potassium intake were 1041.03 mg/d for males and 1020.53 mg/d for females. Furthermore, the mean values of the sodium/potassium ratio for males and females were 0.89 and 1.00, respectively. The mean values of WC for males and females were 52.19 cm and 51.70 cm; and 0.43 and 0.42 for WHtR, respectively. The mean values of SBP and DBP were 97.10 mmHg and 60.91 mmHg for males and 97.00 mmHg and 60.63 mmHg for females, respectively. Among the age group 4–8 years, there were no significant mean differences between males and females.

In children aged 9–13 years, the mean values of sodium intake for males and females were 550.03 mg/d and 598.20 mg/d, whilst the mean values of potassium intake for males and females were 1027.77 mg/d and 1190.11 mg/d, respectively. The mean values of the sodium/potassium ratio were 1.00 and 0.99 for males and females, respectively. In addition, the mean values of WC for males and females were 55.94 cm and 56.25 cm. However, the mean value of WHtR was the same for both males and females, which was 0.40. The mean values of SBP and DBP were 100.77 mmHg and 62.12 mmHg for males and 102.93 mmHg and 62.91 mmHg for females, respectively. There were no statistical mean differences between males and females in children aged 9–13 years except for potassium intake, height and SBP shown in Table 2.

4.2. The Prevalence of Sodium, Potassium Intake, Sodium/Potassium Ratio, Hypertension and Abdominal Obesity according to WC and WHtR

In children aged 4–8 years, 14.9% of males and 19.8% of females consumed above the recommended adequate intake (AI) of sodium, whilst 98.5% of males and 99.0% of females consumed below the recommended AI of potassium. The sodium/potassium ratio was above the WHO recommended ratio in 38.1% of males and 45.5% of females. In addition, the prevalence of hypertension according to high systolic and diastolic blood pressure was 13.4% for males and 6.9% for females. The prevalence of abdominal obesity according to WHtR for males was 0.7% and 2.0% for females. The Chi-square and Fisher's exact test

p-values for all the variables were more than 0.05, suggesting that there was no significant difference in the sample proportions between males and females in Table 3.

Table 2. Descriptive characteristics of the population according to sex and age.

Life Stage Group (Years)	Male 4–8 Years (*n* = 134)	Female 4–8 Years (*n* = 101)	One-Way Anova *p*-Value	Male 9–13 Years (*n* = 260)	Female 9–13 Years (*n* = 270)	One-Way Anova *p*-Value
Variables	**Mean ± SD**	**Mean ± SD**		**Mean ± SD**	**Mean ± SD**	
Na (mg/d)	507.34 ± 578.60	553.70 ± 530.312	0.459	550.03 ± 988.10	598.20 ± 654.51	0.556
K (mg/d)	1041.03 ± 584.66	1020.53 ± 540.19	0.844	1027.77 ± 721.58	1190.11 ± 1020.53	0.032 *
Na/K ratio (mmol/mmol)	0.89 ± 0.95	1.00 ± 0.96	0.282	1.00 ± 1.20	0.99 ± 0.91	0.996
Weight (Kg)	20.69 ± 2.97	20.80 ± 2.97	0.743	28.12 ± 4.36	29.06 ± 5.33	0.024 *
Height (cm)	122.31 ± 7.10	122.99 ± 6.43	0.428	138.79 ± 7.57	139.77 ± 7.66	0.167
WC (cm)	52.20 ± 2.94	51.71 ± 3.24	0.225	55.94 ± 3.47	56.25 ± 4.35	0.370
WHtR	0.43 ± 0.02	0.42 ± 0.26	0.052	0.40 ± 0.02	0.40 ± 0.02	0.661
SBP (mmHg)	97.10 ± 11.20	97.00 ± 11.60	0.542	100.77 ± 9.90	102.93 ± 10.92	0.017 *
DBP (mmHg)	60.91 ± 9.98	60.63 ± 9.33	0.832	62.12 ± 8.52	62.91 ± 9.59	0.318

* *p*-value < 0.05; n—number of individuals. Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, mg/d—milligram per day, mmol/d—millimole per day.

Table 3. The prevalence of sodium, potassium intake, sodium/potassium ratio, hypertension and abdominal obesity according to WC and WHtR.

Life Stage Group (Years)	Male 4–8 Years (*n* = 134)	Female 4–8 Years (*n* = 101)	Chi-Square/Fisher's Exact Test *p*-Value	Male 9–13 Years (*n* = 260)	Female 9–13 Years (*n* = 270)	Chi-Square/Fisher's Exact Test *p*-Value
Variables	***n* (%)**	***n* (%)**		***n* (%)**	***n* (%)**	
Na-Proportion > 1000 (4–8 years) mg/d or >1200 mg/d (9–13 years)	20 (14.9)	20 (19.8)	0.305	26 (10.0)	35 (13.0)	0.305
K-Proportion < 2300 mg/d or <2500 mg/d	132 (98.5)	100 (99.0)	0.444	258 (99.2)	253 (93.7)	0.017 *
Na/K- proportion > 1	51 (38.1)	46 (45.5)	0.238	90 (34.6)	107 (39.6)	0.208
Hypertension according to high SBP and DBP	18 (13.4)	7 (6.9)	0.639	8 (3.0)	21 (7.8)	0.025 *
Abdominal obesity according to WC	19 (14.2)	12 (11.9)	0.622	156 (60.0)	152 (56.3)	0.388
Abdominal obesity according to WHtR	1 (0.7)	2 (2.0)	0.400	0 (0)	1 (0.4)	0.326

* *p*-value < 0.05; n—number of individuals. Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, WC—waist circumference, WHtR—waist-to-height ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, mg/d—milligram per day. AI—adequate intake.

In children aged 9–13 years, 10.0% of males and 13.0% of females consumed above the recommended AI of sodium, whilst 99.2% of males and 93.2% of females consumed below the recommended AI for potassium, respectively. Moreover, the sodium/potassium ratio was above the WHO recommended ratio in 34.6% of males and 39.6% of females. The prevalence of hypertension according to high systolic and diastolic blood pressure was 3.0% for males and 7.7% for females. The abdominal obesity according to WC prevalence in males was 60.0% and 56.3% in females. The prevalence of abdominal obesity according to WHtR for males was 0% and 0.4% for females. Since a vast majority were underweight (undernutrition) in a study conducted by Van Den Ende et al. (2014) on the

same participants [21], the high prevalence of WC as compared to WHtR might due to bloating of malnourishment, rather than visceral fat. The Chi-square or Fisher's exact test *p*-values for potassium intake and hypertension according to high diastolic and systolic blood pressure were less than 0.05, suggesting that there is a significant difference in the sample proportions between males and females in the 9–13 years age group in Table 3.

4.3. The Correlation of Sodium, Potassium Intake and with Systolic Blood Pressure, Diastolic Blood Pressure, Waist Circumference and Waist-to-Height Circumference

Results in Table 4 indicates a significant, weak positive correlation of sodium intake with SBP (r = 0.192, p-value = 0.026), DBP (r = 0.185, p-value = 0.031), and with WHtR (r = 0.176, p-value = 0.041) in males aged 4–8 years. In addition, a significant, weak positive correlation was found between sodium/potassium ratio and WHtR (r = 0.184, p-value = 0.034) in the same age group of males.

Table 4. The correlation of sodium, potassium intake and with SBP, DBP, WC, and WHtR in Pearson correlation.

Variables	**Na**		**K**		**Na/K**	
Male 4–8 Years	***r***	***p*-Value**	***r***	***p*-Value**	***r***	***p*-Value**
SBP (mmHg)	0.192	0.026 *	0.152	0.079	0.073	0.400
DBP (mmHg)	0.185	0.031 *	0.160	0.063	0.006	0.948
WC (cm)	−0.042	0.632	−0.069	0.426	0.004	0.960
WHtR	0.176	0.041 *	−0.013	0.877	0.184	0.034 *
Female 4–8 years						
SBP (mmHg)	0.095	0.342	−0.034	0.733	0.100	0.317
DBP (mmHg)	0.169	0.092	−0.079	0.433	0.166	0.097
WC (cm)	−0.052	0.607	−0.016	0.874	−0.050	0.620
WHtR	−0.065	0.521	−0.059	0.555	−0.048	0.634
Male 9–13 years						
SBP (mmHg)	0.046	0.461	0.039	0.531	0.075	0.227
DBP (mmHg)	0.089	0.155	0.043	0.494	0.098	0.113
WC (cm)	0.063	0.309	0.014	0.820	0.030	0.629
WHtR	−0.016	0.803	0.051	0.414	−0.052	0.408
Female 9–13 years						
SBP (mmHg)	0.011	0.859	0.012	0.0838	0.049	0.426
DBP (mmHg)	0.022	0.715	0.057	0.350	0.043	0.480
WC (cm)	0.075	0.221	0.059	0.338	0.042	0.496
WHtR	0.095	0.121	−0.015	0.807	0.100	0.100

* p-value < 0.05; Na-sodium, K-potassium, Na/K ratio- sodium/potassium ratio, SBP-systolic blood pressure, DBP-diastolic blood pressure, WC-waist circumference, WHtR- waist-to-height ratio, mg/d- milligram per day, mmol/d- millimole per day, cm-centimeter, r-correlation coefficient.

4.4. The Effect of Sodium, Potassium Intake and Sodium/Potassium Ratio on Systolic and Diastolic Blood Pressure, Waist Circumference and Waist-to-Height Circumference

Results in Table 5 indicate a decrease in potassium intake [β = 0.102, (95% CL: 0.004, 0.200), p-value = 0.028] was associated with an increase in SBP. Even when the data were adjusted for age and sex, potassium intake [β = 0.090, (95% CL: −0.007, 0.187), p-value = 0.038] was still associated with SBP.

Table 5. Regression coefficients (β) and 95 % confidence intervals (CI) of the generalised linear model.

Variables	Na			K			Na/K			Na*K		
Unadjusted	**β**	**95% CL**	***p*-Value**	**β**	**95% CL**	***p*-Value**	**β**	**95% CL**	***p*-Value**	**β**	**95% CL**	***p*-Value**
SBP (mmHg)	0.025	−0.126, 0.175	0.746	0.102	0.004, 0.200	0.028 *	0.074	−0.053, 0.200	0.254	−0.031	−0.072, 0.009	0.126
DBP (mmHg)	0.058	−0.093, 0.208	0.453	0.079	−0.019, 0.177	0.115	0.047	−0.080, 0.174	0.466	−0.020	−0.060, 0.020	0.330
WC	0.015	−0.136, 0.166	0.844	0.028	−0.071, 0.126	0.580	0.018	−0.109, 0.145	0.789	0.010	−0.031, 0.226	0.634
WHtR	0.117	−0.034, 0.267	0.130	−0.008	−0.106, 0.091	0.877	−0.049	−0.176, 0.178	0.451	−0.036	−0.077, 0.004	0.077
Adjusted for age and sex												
SBP (mmHg)	0.040	−0.108, 0.187	0.599	0.090	−0.007, 0.187	0.038 *	0.058	−0.066, 0.183	0.358	−0.036	−0.075, 0.004	0.077
DBP (mmHg)	0.064	−0.086, 0.214	0.401	0.074	−0.024, 0.172	0.140	0.040	−0.086, 0.167	0.531	−0.022	−0.062, 0.018	0.282
WC	0.049	−0.086, 0.183	0.478	0.011	−0.077, 0.099	0.812	−0.013	−0.127, 0.100	0.817	−0.002	−0.038, 0.034	0.903
WHtR	0.087	−0.052, 0.226	0.221	0.011	−0.080, 0.103	0.805	−0.020	−0.137, 0.097	0.740	−0.027	−0.064, 0.011	0.160

* *p*-value < 0.05; Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, WC—waist circumference, WHtR—waist-to-height ratio, mg/d—milligram per day, mmol/d—millimole per day, β—beta coefficient and confidence intervals (CI).

Table 6 shows the most frequent food items used in Ellisras rural children aged 5–13 years. This food list is placed in an order from most used to least used food item.

Table 6. The most frequent used food items in children aged 5–13 years in Ellisras.

Maize Porridge or Sorghum Porridge
Tea
Sugar (white)
Brown
Homemade bread
Chicken
Spinach
Non dietary creamer
Beef
Red meat (from goat and wild animals)
Tomato and onion
Cooked dry beans
White bread
Margarine
Fried egg
Fish (canned pilchard or fresh from Lephalale)
Sorghum beer (homemade)
Cooked cabbage
Cold drink (mostly Coke)
Peanut butter
White rice
Sweets
Mashontja (Mopani worms)
Bananas and oranges
Cow milk
Jam

Source adapted [21].

5. Discussion

The purpose of this study was to assess the association between sodium intake and potassium intake; and their ratio with BP, WC, and WHtR. Across two days of dietary intake, in children aged 4–8 years the mean (average) of sodium intake for males were 507.34 mg/d and 553.70 mg/d for females, whilst in children aged 9–13 years the median values of sodium intake for males and females were 550.03 mg/d and 598.20 mg/d. The mean values of sodium intake in both age groups (4–8 and 5–13 years) were notably lower than the recommended AI. A study conducted by Van Den Ende et al. (2014) on the same sample reported a significant large percentage of this population were underweight, while the prevalence of overweight and obesity was very low [21]. This might explain why the levels of sodium intake were notably lower than the recommended. Although that might be the case, our findings were inconsistent compared to similar studies conducted in Morocco, Europe, America, and China, whereby their average intakes were notably higher than the recommended intake. The average of sodium in the Moroccan study was 2235.3 mg/d [4], whilst the average of sodium intake in European countries among children was between 2400 mg/d and 3000 mg/d [33–36]. In addition, America and China reported the highest average of sodium intake at 3100 mg/d and 3400 mg/d [37,38]. This difference might be due to the differences in a geographical area, demographics, and socioeconomic status. Since this study was conducted in a rural area and previous research has shown that the traditional eating habits of most South Africans residing in rural areas consist mostly of a prudent diet which consists of adequate content of sodium and potassium [22,39–42]. This might account for the reason of having a low average of sodium in this population compared with other countries, such as Europe, America, China, and Morocco.

On the other hand, the mean of potassium intake was 1041.03 mg/d for males and 1020.53 mg/d for females in children aged 4–8 years, whilst in children aged 9–13 years the mean of potassium intake for males and females was 1027.77 mg/d and 1190.11 mg/d. This might be due to different food intake. The mean of potassium intake in both age groups were notably lower than the recommended AI. However, it is often challenging to compare values from studies on children especially between boys and girls, mainly due to the different nutritional requirements, particularly when considering potassium recommendations depending on energy needs [43]. Our consumed average intake of potassium in this study, amongst males and females in both age groups were, respectively, lower compared with studies conducted by Campanozzi et al. (2015) and Oliveria et al. (2015) [33,44]. The estimated average of potassium intake was 1530 mg/d in boys and 1400 mg/d in girls in a study conducted by Campanozzi et al. (2015) [33]. In addition, Oliveria et al. (2015) reported that the average of boys and girls were, respectively, 1701.0 mg/d and 1682.0 mg/d [44].

Furthermore, the mean of the sodium/potassium ratio in children aged 4–8 years was 0.89 for males and 1.00 for females. The mean of the sodium/potassium ratio was 1.00 and 0.99 for males and females. The mean of sodium/potassium ratio in both age groups were below or equal to the WHO recommended ratio of $\leq$1. The low average intake of sodium and potassium might have contributed to sodium/potassium ratio being below or equal to the WHO recommended ratio. In comparison with similar studies, the average of sodium/potassium ratio in our study was similar with the finding of a study conducted in North America, but was notably lower as compared to studies conducted in Europe. For instance, the average of sodium/potassium ratio reported in American children is 1.03 [45]. In addition, the average of sodium/potassium ratio in Spanish children (aged 6–14 years) and French children (aged 2–14) were, respectively, 3.6 and 1.64 [46,47]. Countries in Europe did not comply with the recommended sodium/potassium ratio.

Moreover, in our study 14.9% of males and 19.8% of females in children aged 4–8 years; and 10.0% of males and 13.0% of females in children aged 9–13 years consumed above the recommended AI of sodium. A study conducted in Morocco evaluated sodium intake in a population aged 6–18 years, and have shown that sodium intakes were too high and found 41.2% of boys and 58.8% of females consumed above the recommended AI of sodium [4]. In

our study, a total of 98.5% in males and 99.0% in females consumed below the recommended AI of potassium in children aged 4–8 years. At the same time, in children aged 9–13 years, 99.2% of males and 93.7% of females consumed below recommended AI of potassium. These findings are similar to those reported in Italy, where over 96% of boys and 98% of girls consumed lower than the recommended adequate intake of potassium [33]. In our study a total of 35.1% of males and 45.5% of females consumed above WHO recommended sodium/potassium ratio in children aged 4–8 years. At the same time, in children aged 9–13 years, 34.6% of males and 39.6% of females consumed above WHO recommended sodium/potassium ratio.

A significant, weak positive correlation of sodium intake with SBP, DBP, and with WHtR in males aged 4–8 years. In addition, a significant, weak positive correlation was found between sodium/potassium ratio and WHtR in the same age group of males. A decrease in potassium intake was associated with an increase in BP (SBP). Even when the data were adjusted for age and sex, potassium intake was still associated with SBP. Over the years, studies have shown that in children, an increase in BP is associated with high dietary sodium intake [48,49] and reduced BP is associated with lower dietary sodium intake [50]. Evidence on the effect of potassium and sodium intake on BP (SBP or DBP) in children is mixed [19]. Some studies have found a positive correlation between sodium, potassium intake and sodium/potassium ratio with BP [51,52], whilst others have not [53–57]. In our study, there were no significant correlation found among sodium, potassium intake, and sodium/potassium ratio with blood pressure (SBP or DBP) and abdominal obesity (WC and WHtR) in children aged 4–9 years, in females only and in children aged 9–13 years (both males and females). In addition, an increase in sodium intake and a decrease in potassium did not affect the development of abdominal obesity (WC and WHtR) in this population. Few epidemiological studies have indicated a positive correlation of high sodium and low potassium with adiposity measures [18,19]. Diets high in sodium and low in potassium are often high in energy and, therefore, may promote weight gain which may also lead to overweight or obesity [1,13].

As a result of a global situation with excessive levels of sodium intake and deficient levels of potassium intake [4], WHO has developed and implemented global strategies and effective policies for salt consumption, targeting the main sources of dietary sodium intake for all age groups [2]. Due to this situation, South Africa has taken the stand to become the first country in the world to regulate sodium consumption at the manufacturing level for several industries. These new regulations are designed to reduce the level of sodium in certain food products [58].

The main strength of this study could be that we used interviewer-administered questionnaires, which are more effective than self-administered questionnaires [39]. However, a 24-h recall could be considered as a limitation. Because 24-h urinary excretion of sodium and potassium has been considered to be the "golden standard" method of obtaining data on sodium and potassium intake in population surveys and more accurate than the 24-h recall questionnaire [4,59]. Although this research is part of an ongoing research, the fact that the data for research was collected in 1999 may serve as a methodological limitation of this present study.

6. Conclusions

In our study, we found that this population has a low average intake of sodium and potassium. However, a significant, weak positive correlation of sodium intake with SBP, DBP and with WHtR, and also a significant, weak positive correlation was found between sodium/potassium ratio and WHtR. In addition, a decrease in potassium intake had an effect on the increase in SBP. However, more research is needed to further examine how dietary patterns of sodium and potassium can serve as predictors of hypertension and abdominal obesity in South African and African children especially over time; so that those consistent conclusions can be drawn regarding the status of sodium and potassium in children.

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Peer-reviewed article 2:

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Investigating the association of sodium, potassium intake and sodium-to-potassium ratio on the development of hypertension and abdominal obesity over time in Ellisras

Journal:	*Public Health Nutrition*
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Subject Category:	3. Nutritional status and body composition
Keywords:	Sodium intake, Potassium intake, Sodium-to-potassium intake, Hypertension, Abdominal obesity
Abstract:	Background: Hypertension and obesity have both been on the rise in children. Each is associated with an increase in cardiovascular disease risk and both track into adulthood. Objectives: Hence, this study aimed to identify the association of sodium intake (Na), potassium (K) intake, and sodium-to-potassium (Na/K) ratio with hypertension and abdominal obesity amongst the Ellisras rural population over time. Methods: In this longitudinal study, data on dietary intake of Na and K were collected using a 24-h recall questionnaire from a total of 325 participants, from 1999, 2001 and 2015. Blood pressure (BP) and anthropometric measurements were also collected. Parametric (One-way ANOVA) and Chi-square tests were conducted to determine the difference between the years. Generalized estimating equation (GEE) was conducted to assess the association of Na intake, K intake; and their ratio on BP, waist circumference (WC) and waist-to-height ratio (WHtR). Results: Our results indicate a significant positive association between potassium intake and WHtR, and even though the model was adjusted for age and gender there was still an association with WHtR. Na/K ratio was associated with SBP, DBP, WC and WHtR, respectively. In addition to this, sodium-to-potassium ratio was determined to increase the risk of developing hypertension [Exp β= 1.603, (95% CL: 1.164, 2.207) p-

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	value= 0.004] and abdominal obesity [Exp β= 1.797, (95% CL: 1.207, 2.677) p-value= 0.004]. Conclusion: Our study observed that an increase in Na/K indicates the risk of developing hypertension and abdominal obesity over time. Keywords: hypertension, abdominal obesity, longitudinal study

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Abstract

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Keywords: hypertension, abdominal obesity, longitudinal study

1. Introduction

Hypertension and obesity have both been on the rise in children. Both tracks into adulthood and are linked to an increased risk of cardiovascular disease, which increases the prevalence of heart disease and its associated morbidity and mortality[1]. Dietary intake has been identified as one of the important risk factors for non-communicable diseases. High sodium and low potassium intakes are associated with the early development of chronic diseases (e.g., hypertension, obesity)[2].

There are several mechanisms that exist to indicate how sodium and potassium can influence blood pressure. Evidence indicates that an interaction between these micro-nutrients plays a dominant role in the development of primary hypertension[3,4]. Diets specifically characterized by modern western diet- which are high sodium and low potassium produce a biologic interaction with the

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kidneys, which results in extreme levels of sodium and inadequate concentrations of potassium in the human body. These biologic changes result in vascular smooth muscle cell concentration, followed by an increase in peripheral vascular resistance and higher pressure, and finally hypertension[3,4].

Despite the distinguished negative health effects of high sodium intake on hypertension, obesity has recently gained attraction as another possible health-related outcome[5]. Several studies have indicated a significant association between sodium and obesity among adults [3, 6-7]. In addition to this, evidence from nationally representative populations from South Korea, the UK and Australia also identified a significant positive association between sodium and obesity among both the youth and adults [3; 8-9]. Although the mechanisms underlying the association between sodium and obesity have not been well established, the role of soft-drink consumption was explored[10]. This hypothesis contends that higher energy intake from sugar-sweetened beverages consumed to quench the thirst caused by high sodium intake would contribute to obesity development[11], that is salt intake is indirectly related to obesity through soft-drink consumption, through its effect on fluid intake [12]. However, the relationship between dietary intake with hypertension and abdominal obesity is inconsistent. Therefore, we designed this study to investigate the effect of sodium, and potassium intake and their ratio on hypertension and abdominal obesity in the same participants over time in Ellisras.

2. Methodology

2.1. Sampling Procedure

This study is part of the ongoing Ellisras longitudinal study (ELS), of which the details of the geographical area were reported elsewhere[13]. The ELS initially followed a cluster sampling method. Briefly, the study was undertaken at 22 schools (10 pre-school and 12 primary schools) randomly selected from 68 schools within the Ellisras rural area[13]. Baseline data was collected in 1996 aged 3–10 years, with follow-up exams of dietary intake, BP and anthropometric measurements in 1999, 2001, 2015 and 2018. For this study, longitudinal data from 1999, 2001 and 2015 were analysed as shown in Figure 1. A final total of 325 participants (161 males and 164 females) were found to have complete measurements, respectively.

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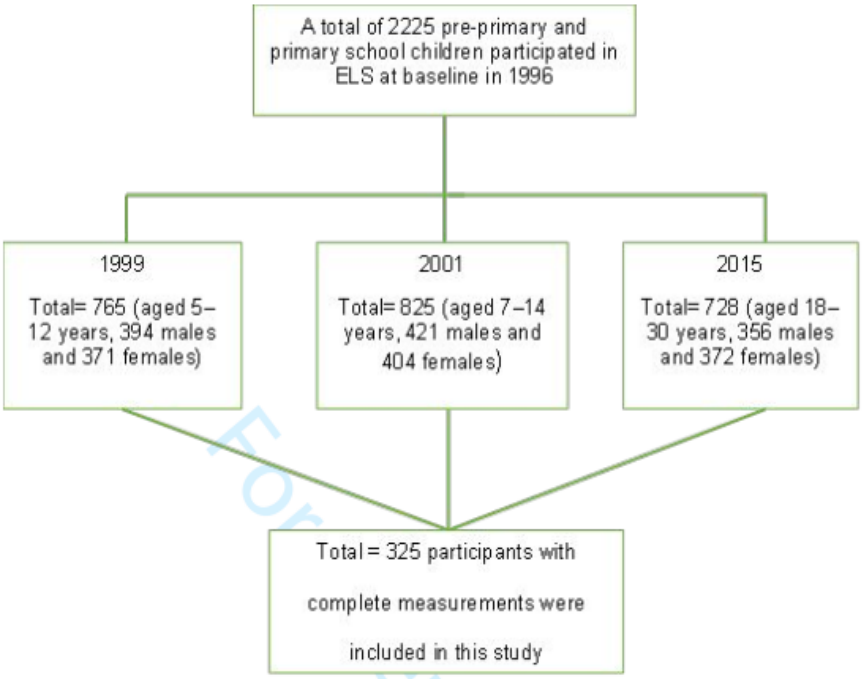

Figure 1: The flow chart of the study participants over the years

2.2. Exclusion Criteria

Any participant with the following was excluded from this study:

- Participants who failed to provide a signed consent form before measurements.

2.3. Blood pressure and anthropometric measurements

Blood pressure was measured using an Omron M2 blood pressure device as described by Whelton *et al*. (2018). The cut–off points for high blood pressure or hypertension as described by National Heart, Lung, and Blood Institute (NHLBI) (2005) were utilised for age, sex, and height[14]. Blood pressure was calculated using the Merck manual-medical calculator for both boys and girls separated[15]. This method of calculation differs from the adults as it uses percentiles to calculate BP for children and adolescents. Cut-off points guidelines as described by Whelton *et al*. (2018) was used for adolescents above eighteen and young adults[16]. Anthropometric measurements (weight, height, and waist circumference) were measured according to standard procedures of the

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International Society for the Advancement of Kinanthropometry[17]. The waist-to-height ratio was calculated as WC divided by height in centimetres. Abdominal obesity was defined as waist circumference (≥ 90th percentile for children and adolescents, ≥ 90 cm for males and ≥ 80 cm for females) and waist-to-height ratio (≥ 0.5)[18,19].

2.4. Dietary Intake

Dietary data were collected using a 24-h recall questionnaire[20]. Trained ELS field workers interviewed participants regarding their dietary intake over the past 24 h. Parents/caregivers of all the participants were interviewed regarding the dietary intake over the previous 24 h. The estimated portion size of foods consumed were recorded in as much detail as possible, using a pre-tested questionnaire and food models simulating average portions of local foods. An average of two days of dietary intake was taken for each participant. One dietary intake was collected during the weekday and another dietary intake for the weekend[21]. This is because food consumption during the weekdays differs from the food consumed during the weekend. People tend to eat a lot more during the weekend than during the week, especially on Saturdays[22]. The averages of sodium intake and potassium intake were analysed using local food tables and the South African Food Composition Database System (SAFOODS)[23] and were compared with the recommended intakes for sodium and potassium as described by Consensus Study Report (2019)[24]. The WHO recommends a sodium/potassium ratio of 1:1 (or ≤1) to assess the average molar sodium/potassium ratio[25]. Since the recommended sodium/potassium ratio is in moles, the sodium and potassium intake in this study were converted from milligrams to milli-moles. The following conversion was used to calculate the average molar sodium/potassium ratio[26]:

23 mg sodium = 1 mmol sodium;

39 mg potassium = 1 mmol potassium.

2.5. Statistical analysis

IBM SPSS Statistics software package (version 27.0) was used to conduct statistical analysis. To describe and characterise the samples, descriptive statistics were calculated for all the variables to indicate frequencies (expressed as percentages), means and standard deviations. Shapiro–Wilk test was used to assess variables' normality. Parametric (one-way Anova), Chi-square, and Fisher's exact tests were conducted to determine the difference in males and females over the years; and

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between the years. Generalized estimating equation (linear and binary logistics) were conducted to assess the association of sodium intake, potassium intake; and their ratio with BP, WC and WHtR; and further investigate the association with hypertension and abdominal obesity. The probability value for statistical significance for all tests was set at a p-value ≤ 0.05.

3. Results

3.1. Characteristics of the population

Table 1A indicates the characteristics of the population for each year stratified by sex, with a total of 325 participants. The dietary mean of Na intake in both males and females over the years, however the increase was not significant in males ($p > 0.05$) and females ($p > 0.05$). The dietary mean of K intake significantly decreased from the first year of assessment to the last year of assessment, in both males ($p < 0.001$) and females ($p < 0.001$). Whilst the dietary mean of Na/K ratio significantly increased over the years, in both males ($p < 0.001$) and females ($p < 0.001$). Furthermore, the mean of both SBP and DBP significantly increased from the first year of assessment to the last year of assessment, respectively in both males ($p < 0.001$) and females ($p < 0.001$). The mean of abdominal indices (WC and WHtR) significantly increased over the years, in both males ($p < 0.001$) and females ($p < 0.001$). Table 1B further shows the dietary mean of the micro-nutrients [Na ($p < 0.05$), K intake ($p < 0.001$) and Na/K ratio ($p < 0.001$)] significantly increased over the years for the total population. In addition, the mean of BP measurements of the total population significantly increased from the first point of measurement, with SBP ($p < 0.001$) increasing to 120.75 mmHg and DBP ($p < 0.001$) to 70.67 mmHg in the last year of measurement, respectively. The mean of abdominal indices significantly increased from the first measurement (54.44 cm-WC and 0.41-WHtR), with WC increasing to 79.0 cm and WHtR increasing to 0.48 in the last year . Furthermore, Table 2 presents the most frequent food items in the diets of the Ellisras participants from 2001 and 2015. In 2001, maize or sorghum porridge were the most stable food amongst the Ellisras children with jam being the least used item. Whilst 2015, fried chicken with skin was the most used item and pilchards were the least used food item in the Ellisras young adults.

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Table 1A: characteristics of the population over the years, stratified by sex

Year	1999		2001		2015	
	Males (n=161)	Females (n=164)	Males (n= 161)	Females (n=164)	Males (n=161)	Females (n=164)
Variables	Mean , SD	Mean , SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Age	8.78 ± 1.86	9.59 ± 1.75	10.78 ± 1.86	11.54 ± 1.75	24.79 ± 1.84	25.52 ± 3.13
Na (mg)	482.06 ± 593.49	532.84 ± 593.03	482.06 ± 593.49	535.82 ± 592.44	661.45 ± 988.80	717.18 ± 1082.93
K (mg)	1043.97 ± 836.65	1165.77 ± 963.26	1043.97 ± 836.65	1175.37 ± 968.15	**756.28 ± 657.31****	**753.27 ± 678.56****
Na/K ratio (mmol)	0.90 ± 1.03	0.89 ± 0.93	0.90 ± 1.03	0.89 ± 0.92	**2.03 ± 2.75****	**1.92 ± 2.67****
SBP (mmHg)	99.86 ± 11.71	102.17 ± 11.43	95.51 ± 9.90	99.39 ± 11.21	**126.72 ± 13.11****	**115.00 ± 10.63****
DBP (mmHg)	61.90 ± 9.89	62.92 ± 9.58	63.58 ± 7.21	64.66 ± 8.04	**71.45 ± 10.17****	**69.93 ± 8.99****
WC (cm)	53.93 ± 3.77	54.96 ± 4.69	56.65 ± 4.14	58.23 ± 4.30	**74.53 ± 9.12****	**84.01 ± 15.39****
WHtR	0.41 ± 0.02	0.40 ± 0.02	0.41 ± 0.04	0.41 ± 0.04	**0.43 ± 0.08****	**0.52 ± 0.12****
Height	130.36 ± 120.08	135.10 ± 10.68	138.51 ± 11.68	143.47 ± 10.77	**173.54 ± 12.83****	**162.79 ± 10.29****

n—number of individuals. Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, WHtR—Waist-to-height ratio, mg/d—milligram per day, mmol/d—millimole per day, cm—centimeter, **p-value < 0.001

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Table 1B: Comparison of means of sodium, potassium intake, sodium-to-potassium ratio, systolic and diastolic blood pressure over the years

Year	1999	2001	2015	p-value
Variables	Mean ± SD	Mean ± SD	Mean ± SD	
Na (mg)	507.69 ± 592.89	509.27 ± 592.66	689.84 ± 1036.56	**0.008***
K (mg)	1105.43 ± 903.43	1110.48 ± 906.59	754.75 ± 667.16	**0.000****
Na/K ratio (mmol)	0.89 ± 0.98	0.89 ± 0.98	1.97 ± 2.70	**0.000****
SBP (mmHg)	101.02 ± 11.61	97.47 ± 10.74	120.75 ± 13.26	**0.000****
DBP (mmHg)	62.41 ± 9.73	64.12 ± 7.64	70.67 ± 9.60	**0.000****
WC (cm)	54.44 ± 4.27	57.47 ± 4.30	79.40 ± 13.53	**0.000****
WHtR	0.41 ± 0.24	0.41 ± 0.04	0.48 ± 0.11	**0.000****

n—number of individuals. Na—sodium, K—potassium, Na/K ratio—sodium/potassium ratio, SBP—systolic blood pressure, DBP—diastolic blood pressure, WHtR—Waist-to-height ratio, SD—standard deviation, mg/d—milligram per day, mmol/d—millimole per day, cm—centimeter, * **p-value < 0.05; **p-value < 0.001**

Table 2: The most frequently used food items for the years 2001 and 2015 in Ellisras, from the most used to the least

2001	2015 (%)
Maize porridge or Sorghum	Fried chicken with skin (23.8)
Sugar (white)	Pap (22.6)
Homemade bread	Cold drink (16.9)
Chicken	White sugar (14)
Spinach	Vetkoek (5.8)
Non-dietary creamer	Fried beef (4.7)
Beef	Peanut butter (4.4)
Red meat (from goat and wild animals)	Samp (2.6)
Tomato and onion	Yoghurt (2.4)
Cooked dry beans	Spinach (2.0)
Cold drink (mostly coke)	Pilchards (0.5)
Peanut butter	

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Sweets	
Mashontja (Mopani worms)	
Bananas and oranges	
Cow milk	
Jam	

Source adapted [27, 28]

3.2. The prevalence of sodium, potassium, hypertension and abdominal obesity

Table 3A indicates a significant increase in hypertension from the first year to the third year, in both males and females, respectively ($p< 0.001$). The prevalence of abdominal obesity according to WC significantly decreased in males, whilst in females, it increased from the first point of measurement to the third year ($p< 0.001$). In addition to this, both males and females prevalence of abdominal obesity according to WHtR significantly increased over the measured years ($p< 0.001$). Results in Table 3B shows the prevalence of hypertension was 4.9% at the first measurement, and it significantly increased to 23.4% in the last year of assessment ($p< 0.001$). In addition, the prevalence of abdominal obesity according to WC significantly decreased by 5% from the first point of measurement to the last year of assessment ($p< 0.001$). Whilst the prevalence of abdominal obesity significantly increased from 0.3% at first measurement to 35.7% in the last year of measurement ($p< 0.001$).

Table 3A: The prevalence of sodium, potassium, hypertension and abdominal obesity stratified by gender

Year	**1999**		**2001**		**2015**	
Dependent variables	Males n (%)	Females n (%)	Males n (%)	Females n (%)	Males n (%)	Females n (%)
Na above the adequate intake	14 (8.7)	18 (11.0)	20 (12.4)	15 (9.1)	19 (11.8)	17 (10.4)
K below the adequate intake	156 (96.9)	153 (93.3)	153 (95.0)	150 (91.5)	155 (96.3)	**163 (99.4)***
Na/K above the recommended intake	57 (35.4)	56 (34.1)	58 (36.0)	56 (34.1)	**84 (52.2)****	**82 (50.0)***

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Hypertension	8 (5.0)	8 (4.9)	4 (2.5)	6 (3.7)	**36 (22.5)****	**40 (24.2)****
Abdominal obesity according to WC	58 (36.0)	71 (43.6)	108 (67.1)	131 (79.9)	**17 (10.6)****	**96 (58.2)****
Abdominal obesity according to WHtR	0 (0)	1 (0.6)	4 (2.5)	10 (6.1)	**19 (11.9)****	**97 (58.8)****

n—number of individuals. Na—sodium, K—potassium, WC—waist circumference, WHtR—waist-to-height ratio, mg/d—milligram per day, ***p-value < 0.05, ** p-value < 0.001**

Table 3B: The prevalence of sodium, potassium, hypertension and abdominal obesity over the years

Year	**1999**	**2001**	**2015**	**Chi-square p-value**
Dependant Variables	n (%)	n (%)	n (%)	
Na above the adequate intake	32 (9.8)	35 (10.8)	36 (11.1)	0.868
K below the adequate intake	309 (95.1)	303 (93.2)	318 (97.8)	**0.019***
Na/K above the recommended intake	113 (34.8)	114 (35.1)	166 (51.1)	**<0.001***
Hypertension	16 (4.9)	10 (3.1)	76 (23.4)	**<0.001****
Abdominal obesity according to WC	129 (39.8)	239 (73.5)	113 (34.8)	**<0.001****
Abdominal obesity according to WHtR	1 (0.3)	14 (4.3)	116 (35.7)	**<0.001****

n—number of individuals. Na—sodium, K—potassium, WC—waist circumference, WHtR—waist-to-height ratio, ***p-value < 0.05 ** p-value < 0.001**

3.3. The association of sodium intake, potassium intake and sodium-to-potassium ratio with dependent variables

The results in Table 4 showed a significant positive association between potassium intake and WHtR [β = 0.019, (95% CL: 0.004, 0.034) p-value= 0.012], and even after age and gender were taken into account in the model, there was still an association with WHtR [β = 0.018, (95% CL: 0.002, 0.034) p-value= 0.024]. Sodium-to-potassium ratio was significantly associated with SBP [β= 4.326, (95% CL: 2.056, 6.595) p-value< 0.001], DBP [β= 2.028, (95% CL: 0.703, 3.353) p-value= 0.003], WC [β= 4.191, (95% CL: 2.080, 6.302) p-value< 0.001] and WHtR [β= 0.014, (95% CL: 0.003, 0.026) p-value= 0.015], respectively. However, when the model was adjusted for

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age and sex, an association was not found between sodium-to-potassium ratio and blood pressure measurements and abdominal indices, respectively. Table 5 shows that in this study, it was further observed that the Na/K ratio increased the risk of developing hypertension [Exp β= 1.603, (95% CL: 1.164, 2.207) p-value= 0.004] and abdominal obesity [Exp β= 1.797, (95% CL: 1.207, 2.677) p-value= 0.004] compared to Na and K intake alone. However, when age and sex were taken into account, sodium-to-potassium ratio was not found to increase the risk of developing hypertension and abdominal obesity as shown in Table 5

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Table 4: The association of sodium intake, potassium intake and sodium-to-potassium ratio with blood pressure and abdominal indices using GEE (linear)

	Na			K			Na/K		
Unadjusted	*β*	95% CI	p-value	*β*	95% CI	p-value	*β*	95% CI	p-value
SBP	-0.507	-4.379, 3.366	0.798	2.033	-2.022, 6.088	0.326	4.326	2.056, 6.595	<0.001**
DBP	-0.240	-2.287, 1.807	0.818	0.736	-1.738, 3.211	0.560	2.028	0.703, 3.353	0.003*
WC	-0.735	-4.071, 2.600	0.666	2.314	-1.017, 5.644	0.173	4.191	2.080; 6.302	<0.001**
WHtR	-0.001	-0.016, 0.013	0.866	0.019	0.004, 0.034	0.012*	0.014	0.003, 0.026	0.015*
Adjusted for age and sex									
SBP	0.915	-1.769, 3.599	0.504	0.579	-2.662, 3.821	0.726	0.967	-0.655, 2.589	0.243
DBP	0.311	-1.571, 2.194	0.746	0.283	-2.142, 2.709	0.819	0.798	-0.418, 2.014	0.198
WC	1.052	-0.668, 2.773	0.231	1.247	-0.642, 3.136	0.196	0.469	-0.701, 0.617	0.432
WHtR	0.004	-0.009, 0.017	0.57	0.018	0.002, 0.034	0.024*	0.005	-0.005, 0.015	0.329

Na-sodium, K-potassium, Na/K ratio- sodium/potassium ratio, SBP-systolic blood pressure, DBP-diastolic blood pressure, WC-waist circumference, WHtR- waist-to-height ratio, mg/d- milligram per day, mmol/d- millimole per day, β – beta coefficient, * p-value < 0.05, **p-value < 0.001

Table 5: The risk measure of sodium, potassium intake and sodium-to-potassium ratio causing the development of hypertension and abdominal obesity according to WC and WHtR using GEE (binary logistic)

	Na			K			Na/K		
Unadjusted	Exp (β)	95% CI	p-value	Exp (β)	95% CI	p-value	Exp (β)	95% CI	p-value
Hypertension	0.959	0.590, 1.558	0.865	1.941	0.762, 4.941	0.164	1.603	1.164, 2.207	0.004*
Abdominal obesity according to WC	1.131	0.730, 1.754	0.581	0.634	0.321, 1.254	0.190	0.912	0.684, 1.216	0.529

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Abdominal obesity according to WHtR	1.066	0.579, 1.964	0.837	1.964	0.589, 6.545	0.272	1.797	1.207, 2.677	0.004*
Adjusted for age and sex									
Hypertension	0.873	0.530, 1.437	0.593	0.679	0.283, 1.632	0.387	0.968	0.668, 1.403	0.864
Abdominal obesity according to WC	1.136	0.715, 1.805	0.588	0.698	0.362, 1.346	0.283	0.989	0.731, 1.338	0.943
Abdominal obesity according to WHtR	0.654	0.312, 1.372	0.262	0.702	0.221, 2.233	0.549	0.781	0.493, 1.239	0.295

190 Na-sodium, K-potassium, Na/K ratio- sodium/potassium ratio, SBP-systolic blood pressure, DBP-diastolic blood pressure, WC-waist circumference, WHtR- waist-to-height ratio,
191 mg/d- milligram per day, mmol/d- millimole per day, Exp β – exponential beta, * p-value < 0.05, **p-value < 0.001

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Discussion

The purpose of this study was to investigate the association of sodium, and potassium intake and their ratio on hypertension and abdominal obesity in the same participants over time in Ellisras. Our study findings showed a positive relationship between potassium intake and WHtR over the years, an increase in potassium increased WHtR by 0.019 cm. Even when adjusted for the relevant covariates such as age and gender, potassium intake is still associated with an increase in WHtR (an increase of 0.018 cm) over the years. This could probably be due to the notably low mean intake of potassium throughout the measured years. As mentioned before, more than 90% of the sample population each year consumed below the recommended intake, and this population had a lower intake of fruits and vegetables in 2015 compared to 2001 (e.g. the sample population consumed bananas and oranges which are high in potassium), resulting in the decline of potassium over the years. Murakami et al. (2012) stated that a higher intake of potassium may also be associated with a lower risk of obesity mainly due to higher intakes of fruits and vegetables, which are the major sources of potassium[29], although the effect of fruits and vegetables on obesity is controversial[30]. In addition, no positive association was found between sodium intake and blood pressure measurements and abdominal indices. Potassium intake was also not associated with BP measurements and abdominal obesity according to WC.

Furthermore, sodium-to-potassium ratio was positively associated with BP measurements (increasing SBP by 4.236 mmHg and DBP by 2.028 mmHg), and abdominal indices (increasing WC by 4.191 cm and WHtR by 0.0014 cm). In our study, we further observed that the sodium-to-potassium ratio was a stronger predictor of hypertension and abdominal obesity compared to sodium and potassium intake alone. Pereira et al. (2019) found independent associations of sodium and potassium intake with BP, however when evaluated in a combined manner, as in the case of the sodium-to-potassium ratio, the effect was potentiated[31]. The dominance of sodium-to-potassium ratio as a predictor of BP variation compared to sodium and potassium as individual predictors have been observed in several studies, most of them conducted in hypertensive patients[32], higher sodium-to-potassium ratio may lead to higher BP during follow-up[33]. Ge *et al.* (2015) further revealed that the urinary sodium-to-potassium ratio was also associated with obesity independently, and a high sodium-to-potassium ratio could increase the risk of obesity[34], which was similar to the results of Jain *et al.* (2014)[35]. However, no association was found

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between sodium-to-potassium ratio (derived from self-reporting) and obesity[36], this is contrary to what we found as our results are derived from self-reporting dietary. When data was adjusted for age and sex, no association was found between sodium-to-potassium ratio and BP measurements and abdominal indices, and further could not predict for hypertension and abdominal obesity in this sample population.

In all the years, there were significant mean differences in males and females for the intake of potassium and sodium-to-potassium ratio. In addition, there were significant mean differences in abdominal measurement for both males and females over the years. On the other hand, BP measurements had a significant mean difference in males and females over the years. Significant differences in the prevalence of hypertension, WC and WHtR were observed in both genders over the years. Over time, there were significant variations in potassium, sodium-to-potassium ratio, hypertension, WC, and WHtR prevalence. It must be noted that the majority of the participants were classified as underweight as stated by Van Den Ende et al. (2014)[27]. Therefore, the high prevalence of abdominal obesity according to WC compared to the prevalence of abdominal obesity according to WHtR 2001 might be due to bloating of malnourishment rather than visceral fat[21]. There was no significant change in the prevalence of sodium over time.

The change in political, social and economic factors in South Africa has resulted in increased urbanisation and progress[27]. The increased accessibility, availability and affordability of processed foods in South Africa are of concern, as these types of foods are generally considered to be high in fat, sugar or salt (sodium)[37,38]. Although our study found a lower intake of sodium and potassium, this is probably due to that this study was conducted in a rural settlement. Excessive intake of sodium and a deficient potassium intake could lead.

There are few limitations regarding this study, is the use of the 24-h recall questionnaire compared to the use of the 24-h urinary excretion, as 24-h excretion is considered the golden standard method of obtaining data on sodium and potassium intake in population surveys and is more accurate than the 24-h recall questionnaire[39]. Another limitation is the small sample size and not having different ethnicities from different geographical regions. Future sodium excretion data over time, from childhood to adulthood within these regional areas will be ideal for evaluating post-legislation salt intakes and the impact on public health[40]. Not including the socio-economic status of the sample population. In addition, the gap differences between the years is a limitation of this

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study. This was due to financial constrain. The strength of this study is the use of longitudinal data. The current study is valuable and informative regarding the status of sodium and potassium intake in a sample of South Africans. As tracking the dietary habits of children to adulthood is vital, as children with extremely high levels of sodium intake tend to maintain those levels for time[41]. These could lead to the development of hypertension and abdominal obesity. Thus, close monitoring of children is needed for better management of their health.

In conclusion, potassium intake was positively associated with WHtR over time. In addition, the sodium-to-potassium ratio was positively associated with BP measurement and abdominal indices. An increase in the sodium-to-potassium ratio was further found to increase the risk of developing hypertension and abdominal obesity over time. However, more data is crucial in establishing the effectiveness of sodium reduction and the increase in potassium intake, and with the use of more valuable methods to better understand their effect on blood pressure and abdominal weight.

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Ethical standards disclosure: Ethical clearance was granted by the Turfloop Research Ethics Committee (TREC) of the University of Limpopo before the study was conducted. This study is part of the ongoing ELS that started in 1996 with ethical clearance number MREC/P/204/2013:IR. Informed consent was obtained from all subjects involved in the study.

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Institutional Review Board Statement: The ethical approval was granted by Turfloop research committee (protocol code TREC/88/2021: PG and 08 June 2021).

Data Availability Statement: Data will be made available upon request.