A Panel Analysis of Health Expenditure and Economic Growth of the Economic Community of West African States

M.O. OCHE

ID: orcid.org/0000-0001-7364-3697

Dissertation Accepted In Fulfillment of the Requirements for the Degree Master of Commerce in Economics at the North-West University (NWU)

SUPERVISOR: PROF. GISELE MAH

Graduation Ceremony July 2018

Student Number: 25748173
ABSTRACT

The West African region have been plagued with huge burden of diseases such as HIV/AIDS, malaria, neglected tropical diseases, Tuberculosis, Ebola among others. Aside these non-communicable and communicable diseases, the region also suffers from malnutrition and poverty which places demand on the kind of medicines needed. One of the many efforts made by African leaders in the fight against poor health outcome in the continent was the Abuja Declaration on public health spending by African countries which request all government to allocate 15% of their income to health. This indicates that, there is need for countries to improve their allocation of resource on health. The main purpose of this study therefore, is to empirically examine the relationship between health expenditure and economic growth of the countries of Economic Community of West African States for the period 1995-2014. To achieve this aim, the Panel cointegration approach as well as the Toda and Yamamoto causality test are utilized as the modelling techniques and it was found that there is long run equilibrium relationship among the variables. The result of the Panel Fully Modified Ordinary Least Squared was found suitable for this study and it indicates that Log of Gross Domestic Product Per Capita, Log of Life Expectancy and Log of Population Growth are positively related to Log of Health expenditure Per Capita while Log of Infant Mortality Rate is negatively related to Log of Health expenditure Per Capita. It was also observed, that the coefficient of Log Gross Domestic Product Per Capita, Log of Life Expectancy and Log of Infant Mortality rate are high compared to Log of Population growth. All these variables followed the a priori expectation. Log Gross Domestic Product Per Capita is positive and statistically significant at 5% significant level while Log of Infant Mortality rate is negative and statistically significant at 5% significant level. Although, log of life expectancy and log of population growth are positive, they not significant at the 5% significance level. The outcome of the Toda and Yamamoto causality test showed that there is bidirectional causality between health expenditure and Gross Domestic Product per capita for the Economic Community of West African States for the period under investigation. The coefficient of Gross Domestic Product Per Capita is less than unit hence; it means that health expenditure in this region is a necessity and not a luxury. This study therefore recommend that the government of these countries should invest more to improve the level of growth as this will equally stimulate investment in the health sector for these economies. The elasticity of the Gross Domestic Product per capita found in this study should be considered as the benchmark for financial outlay of the per capita health expenditure in the policy framework bearing in mind that
health is a capital which would mean that investing more to improve it would result in increase in income which will generally lead to growth of the economy.

**Keywords:** ECOWAS, Panel cointegration, FMOLS, GDP per capita, Health Expenditure Per Capita
ACKNOWLEDGEMENT

My sincere appreciation goes to the Almighty God, the fountain of knowledge who has generously given me the knowledge, understanding and strength (mental, emotion and physical) to carry out this work.

My profound gratitude goes to Prof Gisele Mah (my supervisor) who gave me all the necessary guidance throughout the period of this work. Thank you so much for your patience and motivating words that kept me going throughout this period. Believing in me made a whole lot of difference as regarding this work. I also appreciate all the Lecturers in the Economic Department North West University, Mafikeng campus for their support.

I like to thank my brother (Mr. Patrick Oche) the inspiration behind all my success academically. You are actually the best brother in the whole wide world. Thank you for motivating me for this success. I appreciate your financial support, prayer and guidance. Thank you so much sir.

I also like to appreciate Mr. and Dr (Mrs) Mark Makinde. You are truly my foster parents. Thank you so much for the care, love, support, instruction, guidance and above all your prayers that have made things easy for me and to carry out this work.

My sincere gratitude goes to my parents Mr. and Mrs. Paul Oche and the rest of my siblings (Comfort, Cletus, Mike, John, Abah and Emma) whose words of encouragement have moved me forward. I love you all.

I equally appreciate Mr. Caleb Obotu Ameh (buddy) as he is popularly called for his words of encouragement and for believing in me. This has kept me going through out this period. Thank you Sir.

Finally, my stay in south Africa would not have being worthwhile without Mr. Simon Isaiah, Mr. Oluwaseyi Samuel, My brothers from another mother. You guys are simply the best. Thanks for accommodating my excesses and supporting me all through the period of this work.

I equally acknowledge all RCF members North West University, RCCG Pentecost assembly. Thank you all.
DECLARATION

I, OCHE, Mary Onyemowo, declare that this study titled “A Panel Analysis of Health Expenditure and Economic Growth of the Economic Community of West African States (ECOWAS) Countries From 1994-2014”, is my own work submitted for the award of degree of Master of Commerce in Economics at the North West University. This dissertation has never been submitted for any degree at this or any other university either by me or anyone else. All sources in this study have been indicated and acknowledged by means of direct and indirect references.

_____________________
Signed

_____________________
Date
DEDICATION

This dissertation is dedicated to God, my parents and siblings.
**LIST OF ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>DOLS</td>
<td>Dynamic Ordinary Least Squares</td>
</tr>
<tr>
<td>ECOWAS</td>
<td>Economic Community of West African States</td>
</tr>
<tr>
<td>FMOLS</td>
<td>Fully Modified Ordinary Least Squares</td>
</tr>
<tr>
<td>GDPPC</td>
<td>Gross Domestic Product Per Capita</td>
</tr>
<tr>
<td>HEPC</td>
<td>Health Expenditure Per Capita</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>Human Immuno-Deficiency Virus/Acquired Immune Deficiency Syndrome</td>
</tr>
<tr>
<td>LEXP</td>
<td>Life Expectancy</td>
</tr>
<tr>
<td>MORATE</td>
<td>Infant Mortality Rate</td>
</tr>
<tr>
<td>OAU</td>
<td>Organization of African Unity</td>
</tr>
<tr>
<td>OCCGE</td>
<td>Organization de Coordination et de Cooperation pour la Lutte Contre les Grandes Endemis</td>
</tr>
<tr>
<td>OECD</td>
<td>Organization for Economic Cooperation and Development</td>
</tr>
<tr>
<td>POPG</td>
<td>Population Growth</td>
</tr>
<tr>
<td>PVAR</td>
<td>Panel Vector Autoregression</td>
</tr>
<tr>
<td>WAHC</td>
<td>West African Health Community</td>
</tr>
<tr>
<td>WAHO</td>
<td>West African Health Organization</td>
</tr>
<tr>
<td>WAMA</td>
<td>West African Monetary Agency</td>
</tr>
</tbody>
</table>
# TABLE OF CONTENTS

ABSTRACT .............................................................................................................. ii

DECLARATION........................................................................................................ v

DEDICATION .......................................................................................................... vi

LIST OF ABBREVIATIONS.................................................................................. vii

LIST OF TABLES .................................................................................................. xiv

CHAPTER 1 .............................................................................................................. 1

INTRODUCTION TO STUDY ............................................................................. 1

1.1 INTRODUCTION ............................................................................................ 1

1.2 PROBLEM STATEMENT ................................................................................. 3

1.3 OBJECTIVE OF THE STUDY ......................................................................... 6

1.4 RESEARCH HYPOTHESES ........................................................................... 6

1.6 STRUCTURE OF THE STUDY ......................................................................... 7

CHAPTER 2 .............................................................................................................. 9

AN OVERVIEW OF HEALTH EXPENDITURE AND ECONOMIC GROWTH IN 
THE ECOWAS COUNTRIES ................................................................................. 9
2.1 INTRODUCTION

2.1.1 WEST AFRICAN HEALTH ORGANISATIONS TO IMPROVE HEALTH

2.2 HEALTH ISSUES IN ECOWAS COUNTRIES

2.2.1 The Private Sector

2.2.2 Staffing of health expertise in ECOWAS

2.2.3 Healthcare Financing

2.2.4 Disease outbreak in ECOWAS Countries:

2.2.4.1 Malaria

2.2.4.2 Ebola

2.2.4.4 Tuberculosis

2.2.4.5 Meningococcal Meningitis

2.3 HEALTH EXPENDITURE IN ECOWAS COUNTRIES

2.4 ECONOMIC GROWTH IN ECOWAS COUNTRIES

2.5 GROSS NATIONAL INCOME PER CAPITA FOR THE ECOWAS COUNTRIES FROM 2013-2016

2.6 IMPORTANCE OF HEALTH FUNDING ON THE GROWTH OF THE ECONOMIES

2.7 CONCLUSION

CHAPTER 3
THEORETICAL AND EMPIRICAL LITERATURE ........................................... 30

3.1 INTRODUCTION ........................................................................... 30

3.2 THEORETICAL FRAMEWORK ......................................................... 30

3.2.1 Wagner’s Theory of Expenditure ................................................. 30

3.2.2 Keynesian Theory of Expenditure ................................................. 31

3.2.3 The Neoclassical Growth Model .................................................. 32

3.2.4 Endogenous Growth Model ......................................................... 34

3.3 EMPIRICAL LITERATURE ................................................................. 36

3.4 CONCLUSION ............................................................................... 44

CHAPTER 4 ....................................................................................... 45

RESEARCH METHODOLOGY ................................................................. 45

4.1 INTRODUCTION ........................................................................... 45

4.2 MODEL SPECIFICATION ................................................................. 45

4.3 DATA DESCRIPTION AND THE APRIORI EXPECTATION ................. 46

4.4 MODELLING TECHNIQUES ............................................................ 49

4.4.1 Descriptive statistics ................................................................. 50

4.4.2 Stationarity Investigation ............................................................ 50

4.4.2.1 Visual Inspection for Stationarity ........................................... 50
4.4.2.2 Panel Unit Root Test ................................................................. 51

4.4.3 COINTEGRATION TEST ................................................................. 54

4.4.3.1 Pedroni Test of Cointegration ..................................................... 54

4.4.3.2 Kao Test of Cointegration ......................................................... 57

4.5. RESIDUAL-BASED PANEL FULLY MODIFIED OLS (FMOLS) AND DYNAMIC OLS (DOLS) ................................................................. 58

4.5.1 The Panel Dynamic OLS ............................................................... 59

4.5.2 The Panel Fully Modified OLS (FMOLS) ........................................... 59

4.6 PANEL CAUSALITY TEST ................................................................. 60

4.8 DIAGNOSTIC CHECK ........................................................................ 63

4.9 CONCLUSION ..................................................................................... 64

CHAPTER 5 ............................................................................................. 65

PRESENTATION OF RESULTS AND DISCUSSION OF EMPIRICAL FINDINGS .... 65

5.1 INTRODUCTION ................................................................................ 65

5.2 DESCRIPTIVE STATISTICS ............................................................... 65

5.3 STATIONARITY INVESTIGATION ......................................................... 67

5.3.1 VISUAL INSPECTION FOR STATIONARITY .................................. 67

5.3.2 PANEL UNIT ROOT TEST ............................................................ 70

xi
5.4 COINTEGRATION TEST ........................................................................................................71

5.4.1 Pedroni Test of Cointegration .........................................................................................71

5.4.2 Kao Test of Cointegration ...............................................................................................72

5.5 RESIDUAL-BASED PANEL FULLY MODIFIED OLS (FMOLS) AND DYNAMIC OLS (DOLS) ........................................................................................................... 73

5.7 PANEL CAUSALITY TEST ..................................................................................................77

5.8 NORMALITY CHECK ..........................................................................................................79

5.9 CONCLUSION ....................................................................................................................79

CHAPTER 6 ............................................................................................................................81

SUMMARY, CONCLUSION AND POLICY RECOMMENDATION ........................................81

6.1 SUMMARY AND CONCLUSIONS OF THE STUDY .........................................................81

6.3 LIMITATION OF THE STUDY ..........................................................................................85

REFERENCE ..........................................................................................................................86
<table>
<thead>
<tr>
<th>FIG</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIG 1.1</td>
<td>ECOWAS average health expenditure % GDP</td>
<td>3</td>
</tr>
<tr>
<td>FIG 2.1</td>
<td>Total Health Expenditure in ECOWAS (1995-2014)</td>
<td>21</td>
</tr>
<tr>
<td>FIG 2.2</td>
<td>Progression of Real GDP Growth in African Region (2011-2013)</td>
<td>22</td>
</tr>
<tr>
<td>FIG 2.3</td>
<td>ECOWAS per Capita GDP from (1995-2014)</td>
<td>25</td>
</tr>
<tr>
<td>FIG 4.1</td>
<td>Summary of Logical Steps Taken in the Study</td>
<td>61</td>
</tr>
<tr>
<td>FIG 5.1</td>
<td>Graphical Representations of Variables at Level</td>
<td>66</td>
</tr>
<tr>
<td>FIG 5.2</td>
<td>Graphical Representations of Variables at First Difference</td>
<td>67</td>
</tr>
</tbody>
</table>
LIST OF TABLES

TABLE 2.1 Ebola Epidemics in West Africa 2013-2016 .................................................. 16
TABLE 2.2 Epidemics of Meningococcal Disease from 1994-1997 ............................... 19
TABLE 4.1 Descriptions of Data and Expected Relationship with the Dependent Variable ..... 45
TABLE 5.1 Descriptive Statistics of the Variables .............................................................. 64
TABLE 5.2 Panel Unit Root Result .................................................................................. 68
TABLE 5.3 Pedroni Cointegration Result ........................................................................ 70
TABLE 5.4 Kao Cointegration Result .............................................................................. 70
TABLE 5.5A Dynamic Ordinary Least Square Result ...................................................... 72
TABLE 5.5B Fully Modified Ordinary Least Square Result .............................................. 72
TABLE 5.7 Summary of Lag Length Selection Criterion ................................................ 74
TABLE 5.8 Summary of Panel Causality Test ..................................................................... 75
CHAPTER 1

INTRODUCTION TO STUDY

1.1 INTRODUCTION

The African continent in general has faced an overwhelming burden of communicable and non-communicable diseases. The low and middle income countries bear about 80% of the burden of non-communicable disease according to Alwan (2011). The West African region in particular, has been plagued with a huge burden of diseases such as Human Immuno-Deficiency Virus/Acquired Immune Deficiency Syndrome (HIV/AIDS for short), malaria, neglected tropical diseases, Tuberculosis and Ebola among others. Aside from these non-communicable and communicable diseases, the region also suffers from malnutrition and poverty which places demands on the kind of medicines needed (West African Health Organization, 2014). Although there have been financial interventions that could reduce the burden of these diseases, the scope is too low because of weak health systems. Hence, the World Health Organization (WHO) Regional Committee provides the continent with some orientations to overcome these challenges. However, these orientations cannot be successfully implemented without fighting corruption, having a lasting domestic and external investment in the social sector and a secure internal atmosphere. This situation prompted about Forty-six health ministers from Africa to take up the Ouagadougou declaration which suggested some measures of handling the issues of the health system. The declaration compels the member countries to promote inter-sectoral collaboration and public-private partnership to address wider issues of health and improve production and retention of the health force among other things (Sambo & Kirigia, 2014).

The African region in general has not relented in its efforts to improve the health of the people. One of the many efforts made by African leaders in the fight against poor health outcome in the continent was the Abuja Declaration on public health spending by African countries which requested all governments to allocate 15% of their income to health (Organization of African Union, 2010).

The need for health care is a key need for human existence. The World Health Organization (WHO, 2005) recognized that 50% of the difference in the rate of economic growth of the developed and the developing countries is attributed to low life expectancy and ill-health.
Developed nations believed that the health of their populace is paramount to the growth of their economies thus, they have spent a large proportion of their income on health care. Health improvement is crucial in the millennium development goals such that three of the goals were health related; combat HIV/AIDS, malaria, and other diseases; reduce child mortality; and improve maternal health (United Nations Development Programme, 2003) and because in less-developed countries, the public sector is the main health provider, most countries stress the need for primary health care as a way of reducing inequities.

A common link seems to exist between the health of the populace and development of an economy. An article titled ‘Wealthier is Healthier’ by Pritchett and Summer (1996) as well as World Bank (1993a) observed the relationship between wealth and health of population noting that growth of income is key in improving the health of the population. Bloom and Canning (2003) observed that improvement in health reduces the poverty level of a nation and that; it also has the capacity to bring families out of poverty. Hence, the wealth of an economy can be seen as dependent on the health of those in such an economy as those that are physically and mentally fit tends to be more productive or effective than those who are not. They therefore contribute more to output than the sick and this in turn impacts the growth of the economy positively since if people are healthy there is an increase in life expectancy, which stimulates private savings as well as enhances personal investment in education. This aids both investment and human capital development. Besides, healthy individuals have prospects of finding better ways of gaining from these investments. This expenditure element (health expenditure) has a multiplier effect such that an increase in health can lead to increased aggregate expenditure and total demand. Aside from this, in the health sector a large number of people are employed hence an increased health spending will result in an increased number of those employed and thus the aggregate income of the employed which enhances the level of aggregate expenditure and demand (Mushkin, 1962). This chain reaction is referred to as direct effect and the expected impact should be positive. Sickness makes the sick to be a burden on the family and the nation in general. This will result in low productivity as well as an inadequate flow of funds to other productive areas of the economy such as infrastructure, among others.

The observations made by the World Bank (1993a) and Bloom and Canning (2003) as stated above suggest that these macroeconomic variables, economic growth and health expenditure are interdependent. Hence, this study seeks to examine how health expenditure has been enhanced by the level of growth of the ECOWAS region for the period under investigation.
1.2 PROBLEM STATEMENT

Over the years, various countries have tried to improve the health of the people by spending a certain percentage of their Gross Domestic Product (GDP) on health. As noted by Ke et al. (2014), developing economies spend a lower percentage of their GDP on health. It is emphasized that the developing countries spend about 3% of their GDP on health relative to about 12% of GDP spent on health by developed countries.

One of the many efforts made by African leaders in the fight against poor health outcomes in the continent was the Abuja Declaration on public health spending by African countries which requested all government to allocate 15% of their income to health (OAU, 2001). However, none of the West African countries has met this requirement (Tandon & Cashin, 2010). Besides, as observed by Oyedele and Adebayo (2015) from the cointegration analysis on the convergence of Health Expenditure and Health Outcomes for the ECOWAS countries, there have been differences in health convergence and health expenditure in the region. Hence these authors observed that to curb health challenges in the region, there is a need for countries to improve their allocation of resource on health.

Apart from the divergence in health expenditure in the ECOWAS region, there has also been a decrease in health expenditure since 2009. The average health expenditure was 4.76% in 1995 and in 2009 it reached the peak of 6.75%, but declined to 5.97% in 2014. This is depicted in Figure 1.1.

Figure 1.1 Average Total Health Expenditure of ECOWAS (%GDP)

![AVE TOTAL HEALTH EXPENDITURE OF ECOWAS(%GDP)](image_url)

*Author design*

*Data Source: World Bank Development Indicator*
From Figure 1.1, it is observed that the percentage of GDP spent by the ECOWAS sub-region on health decreased from the peak of 6.75% in 2009 to 5.97% in 2014 and this percentage of the GDP is low when compared with the 12% spent by the developed economies on health.

Moreover, despite the divergence in health expenditure and decrease noticed, there is no comprehensive study on the relationship between health expenditure and economic growth of the ECOWAS countries. Most of the studies relating health expenditure and income or economic growth have focused on countries of the Organisation for Economic Co-operation and Development (OECD) (for instance, Berger and Messer, 2002; Jewel et al., 2003; Baltagi and Moscone, 2010; and Mehrara et al., 2010) and on individual countries in ECOWAS (for example, Bakare and Olubokun, 2011; Boachie et al., 2014).

Mushkin (1962) observed that health spending has the capacity to stimulate growth of an economy. Based on the health led growth hypothesis, health is seen as capital, hence investing more in health will increase income which will in general lead to growth in the economy. In the same vein, Cole and Neumayer (2005) saw poor health as one of the determinants of the presence of underdevelopment in several regions of the world. Thus Baltagi and Morscone (2010), while studying non-stationary and cointegration characteristics among health care expenditure and income, found that health care is not a luxury but a necessity.

Moreover, most developing countries, including ECOWAS countries are labour intensive in nature (Bloom et al., 2004). This means that they depend more on labour supply for their production than they use capital. This implies that there is need for healthier people to improve the level of production. This is important because, as noted by Gyimah-Brempong and Wilson (2004), health capital expands the effectiveness with which people create education, and apparently, different structures of human capital. Hanushek and Dongwook (1995) and Schultz (1999) further suggest that both the intellectual and mental capabilities of individuals are enhanced by health and this will account for improved outcomes of education. By implication, the presence of high amount of non-healthy labour will constrain the impact of a high level of human capital on growth. Put differently, it is possible for a country to have a high level of human capital, which most literature has identified to have contributed significantly to the growth of most economies, and yet the level of growth will remain intangible if there is high amount of non-healthy labour.
Furthermore, the signing of the millennium declaration in 2000 (United Nations, 2000) revealed that low income countries have insufficient resources that could ensure that all people have access to even the very basic health services. The prevalence of threatening diseases such as HIV/ AIDS, tuberculosis, cancer, and malaria in the ECOWAS region tend also to worsen this situation. World Health Report (2010) equally acknowledged that there is a high rate of communicable diseases and child mortality in sub-Saharan Africa (SSA) compared to other regions, and that the health status in this region is lower than the other regions of the world. The alarming issue of health in SSA has drawn the attention of the world at large and this has caused the health funding to curb these problems to increase. The fall in expenditure on health as shown in figure 1.1 from 6.75% to 5.97% in 2009 and 2014 respectively coupled with the health challenges in terms of the continuous spread of diseases as well as the recent outbreak of Ebola in the ECOWAS region further suggests the need for more expenditure on health.

Although the quality of labour in terms of human capital can greatly influence the growth of an economy, most empirical literature has captured the contribution of human capital to the growth of an economy solely in terms of education, neglecting health as important aspect of human capital, which obviously is a vital determinant of growth (Bloom et al., 2000; Becker, 2007). The Integrated Health Association (2011) noted that health is wealth and as observed by Bloom and Canning (2001), improved health will increase wealth. This implies that this aspect of human capital should be given adequate attention so that efforts expended on providing education as a way of developing human capital will result in expected impact on the growth of the economy, as identified in literature. As noted by Schultz (1999), good health allows individuals to properly and effectively utilize skills and knowledge acquired through education. Bloom, et al., (2004) also found that healthier people are equally enriched both mentally and physically and hence more productive as they rarely absent themselves from work. Hourly wages are bound to be less with existence of sickness and this effect is critical in less developed economies where production is labour-intensive compared to advanced countries.

Mankiw et al. (1992), Barro and Sala-i-Martin, (1995) as well as Levine and Renelt (1992) among others have observed that human capital contributes to the promotion of the growth and development of an economy. Human capital has the capacity to foster economic growth; hence, it calls for increased expenditure on the development of the human capital. Ames et al. (2001) showed that accumulation of capital spurs the growth of an economy and as suggested
by Easterly and Rebelo (1993) in the policy agenda of a country to reduce poverty, there must be investment in the health of the country and education, among others, to enhance the growth level. In Bloom and Canning (2003) it is observed that improved health can reduce the poverty level of a nation. This observed relationship therefore lends an explanation to why there should be an increase in health expenditure. Thus, this study seeks to explore the relationship between health expenditure and economic growth of the ECOWAS sub-region.

1.3 OBJECTIVE OF THE STUDY

The main objective of this study is to empirically investigate the relationship between health expenditure and the level of economic growth in 15 ECOWAS countries from 1995-2014. However, to achieve this broad objective, the specific objectives of this study are summarized and highlighted as follows:

1. To empirically study the relationship between health expenditure and economic growth in the ECOWAS region
2. To examine the direction of causation between the health expenditure and economic growth in the region.
3. To highlight the importance of health funding on growth of the economies
4. To recommend policies that will help policy makers and most importantly the West African Health Organisation (WAHO) to improve the health status of people in the region.

1.4 RESEARCH HYPOTHESES

From the above stated objectives the following hypotheses will be examined.

1. H0: Economic growth does not have a positive impact on Health expenditure of the countries
   H1: Economic growth has a positive impact on the Health expenditure of the countries.

2. H0: Economic growth does not Granger-cause Health expenditure
   H1: Economic growth does Granger-cause Health expenditure

1.5 IMPORTANCE OF THE RESEARCH

This study is significant as it will add to academic literature. The study designs a model to examine the relationship between health expenditure and economic growth of the ECOWAS
countries within the framework of Panel Fully Modified Ordinary Least Square (FMOLS), Dynamic Ordinary Least Square (DOLS) and Toda and Yamamoto causality test. This is the first study that employs a comprehensive panel econometric framework for analysing the relationship between health expenditure and growth for all the ECOWAS countries. The aim is to show the long run relationship among the variables investigated as well as provide the direction of causation and how they impact the level of health expenditure.

Although there are studies on health expenditure and growth in some countries in West Africa, this will be one study that will bring all the countries in this region together to study the relationship between health expenditure and economic growth of the various economies, and draw attention to the need for improving the health status of the people in the region as a key need for human existence.

It has also been concluded that sound policies and stability are the preconditions for human capital to increase GDP rate, hence based on this background this study will provide policy makers with information that will enhance policy that will help to boost the health of the populace, which will in turn bring about growth in the economy.

1.6 STRUCTURE OF THE STUDY

This study is structured into six chapters. Chapter one focused on the introduction to the study, the problem statement, the research objective, the research hypotheses and significance of the study.

Chapter two is an overview of health expenditure and economic growth in the ECOWAS countries. The chapter examines health issues in ECOWAS countries, health expenditure in ECOWAS countries, economic growth in ECOWAS countries as well as the importance of health funding on the growth of the economies.

In chapter three, discussion is centred on the literature (theoretical and empirical) underpinning the study. Theories such as Wagner’s theory of expenditure, Keynesian expenditure theory, the neoclassical growth model and endogenous growth model are discussed in this chapter alongside the empirical literature. The literature review will determine the necessary variables to be included in the model of this study so that the gap identified can be filled.

Chapter four focuses on explicit explanations of the methodology of the study. It captures the model specification based on both the theoretical and empirical literature underpinning the
study as examined in the preceding chapter. The chapter equally presents the apriori expectation, data description/source and the modelling strategy employed to achieve the aim of the study. The Panel FMOLS and DOLS Model are utilized as the modelling techniques suitable for this study. Thus, the systemic or logical procedure embarked upon includes: descriptive statistics, visual investigation of the variables followed by the unit root test which will be performed with the Levin et al., and Im Persran unit root test. This will be immediately followed by test of cointegration to determine the long run relationship among the variables after which the Panel FMOLS and DOLS will be carried out to identify the coefficients of the variables in order to determine the elasticities of the long run relationship. Afterwards the Granger causality test will be performed to determine the direction of causality.

In chapter five, the results of all the tests performed as stated in the preceding chapter will be presented and discussed. The results of all the tests will be presented in tables except for the visual investigation which will be displayed in graphic form.

Chapter six presents the summary and conclusion as well as policy recommendations based on the findings of the study. It also captures the limitation of this study.
CHAPTER 2

AN OVERVIEW OF HEALTH EXPENDITURE AND ECONOMIC GROWTH IN THE ECOWAS COUNTRIES

2.1 INTRODUCTION

Chapter two focuses on a short overview of the formation of ECOWAS region, a brief look at the WAHO, the health issues in ECOWAS countries, health expenditure in ECOWAS countries, economic growth in ECOWAS countries and the importance of health funding on the growth of the economies.

The ECOWAS was established on May 28 1975 through the treaty of Lagos. ECOWAS is an economic sub-region made up of fifteen countries at its inception. The membership of this region increased to 16 in 1977 with Cape-Verde joining. However, the membership reduced back to 15 in 2002 because Mauritania left. Hence the current member countries of this region are; Benin, Burkina Faso, Cabo Verde, Côte d'Ivoire, The Gambia, Ghana, Guinea, Guinea Bissau, Liberia, Mali, Niger, Nigeria, Senegal, Sierra Leone and Togo. This 15 member regional group was created with a mandate of promoting economic integration in all fields of activities of the constituting countries. ECOWAS was set up to foster the idea of collective self-sufficiency for its member states. As a trading union, it is also meant to create a single, large trading bloc through economic cooperation (WAHO, 2014)

ECOWAS consists of specialized agencies which include;

- West African Health Organization (WAHO)
- West African Monetary Agency (WAMA)
- Inter- governmental Action Group against Money Laundering and Terrorist Financing in West Africa (GIABA)
- ECOWAS Gender and Development Centre.

These countries, like other parts of the world, have suffered from pandemics of some diseases over the years and examples in this category are HIV/AIDS and the Ebola outbreak as well as malaria and tuberculosis, among others.

To remain within the scope of this study, the WAHO is briefly examined.
2.1.1 WEST AFRICAN HEALTH ORGANISATIONS TO IMPROVE HEALTH

The West African Health Organization (WAHO) was framed in 1987 when the Heads of State and government from each of the fifteen nations in ECOWAS embraced the convention making the organization. The convention, which was accordingly confirmed by every legislature in the sub-region, granted WAHO status as a specific office of ECOWAS and described the organization’s main goal as follows; “the objective of the West African Health Organization shall be the attainment of the highest possible standard and protection of health of the people of the sub-region through the harmonization of the policies of the member states, pooling of resources, and cooperation with one another and with others for collective and strategic combat against the health problems of the sub-region.”

According to Article III, section 1 1987 convention of WAHO (in French), the main impetus behind WAHO’s creation was the incongruence of the plans that were being sought by the two existing inter-governmental health organization in the sub-region, the Francophone Organization de Coordination et de Coopération pour La Lutte Contre les Grandes Endémies (OCCGE) and the Anglophone West African Health Community (WAHC). It was resolved that as issues of health are not bound by linguistic constraints, it would profit the organizations to harmonize their efforts and consolidate assets to upgrade the influence of their programmes in West Africa. In this way, the OCCGE and WAHC amalgamated to form WAHO, an organization devoted to rising above linguistic fringes in the sub-region to serve every one of the fifteen ECOWAS countries. In October of 1998, Bobo-Dioulasso in Burkina–Faso was established as the headquarters of the WAHO by the ECOWAS Heads of State and Government, and both a Director and Deputy Director of the organization were appointed. WAHO started dynamic operations as a health specialist in the sub-region serving ECOWAS countries in March of 2000. WAHO is a pro-active instrument of regional health integration that empowers high-effect and economical intercessions and programmes by; Maintaining viable organisations, Strengthening capacity building, Collecting, interpreting and disseminating information, Promoting collaboration and guaranteeing coordination and advocacy and Exploiting information communication technologies.

From Article III, section 1 1987 convention of WAHO it is observed that the supreme institution of ECOWAS is the authority of Heads of State and Government of member states which is made up of Heads of State and/or Government of the member states. This specialist is charged with the responsibility of directing and controlling the activity in this economic region and takes all necessary actions that guarantee the dynamic advancement and the
realization of the targets of the community. All things considered, it is additionally the highest policymaking body of WAHO. The assembly of ministers is a revolving panel of ministers from ECOWAS member states that is made up of ministers of Integration, Economic planning and Finance, and Foreign Affairs. The group is in charge of the functioning and advancement of the community and makes suggestions on any action regarding the targets of the community to the authority of ECOWAS.

The purview of the assembly of health ministers is primarily restricted to issues of health and more specifically to the technical aspects therein. The assembly decides the general policies of WAHO and makes proper choices to advance or propel the goals of the organization. WAHO is a specialized agency of ECOWAS, however it enjoys financial and administrative autonomy.

This chapter therefore, looks at health issues in the ECOWAS sub-region, examines the expenditures on healthcare, the growth of the region and importance of health expenditure on economies in general.

2.2 HEALTH ISSUES IN ECOWAS COUNTRIES

World Health organisation (1948) defines Health as a state of complete physical, mental, and social well-being of an individual and not merely the absence of disease or infirmity. The issues related to healthcare delivery and access in West Africa are many; poor health infrastructure, poor organisation, inadequate funding are a few of the numerous dilemmas hindering the attainment of sustainable quality health care delivery in West African countries. Hence, the inherent problem is that it is hard to regularly access quality medical care, as many West Africans find it difficult to access in government established healthcare centres while the cost of the private sector can only be affordable to the rich. Among the numerous issues surrounding health in West African sub-region, a few are discussed in this study.

2.2.1 The Private Sector

The issues related to health care delivery and access in ECOWAS are many. Lack of adequate funding, limited workforce, poor organization and a shortage of sustainable private sector solutions are only a few of the numerous issues keeping numerous West Africans from having sustainable access to quality health care. ECOWAS health care is characterised by inadequate, cost-effective private sector market. Much of this is as a result of the dreaded high cost and speculation encompassing the profit motives of prospective investors. Although
these doubts maybe justified due to the insurance industries checkered history in different parts of the world, lack of strong private sector options is another problem that has plagued the accessibility of healthcare in ECOWAS. Medical service seekers are burdened with high out-of-pocket payments hence providers of health care services cannot predict the flow of revenue (WHO, 1998). This absence of consistency has prompted the failure of suppliers to enhance the quality of services. Accordingly, based on other numerous influences, the private sector has stayed underutilized. In all actuality West Africans have demonstrated able to do and prepared to prepay for services. It is apparent in the achievement of the prepaid cell cards. Obviously the healthcare market has numerous complexities, and in comparisons with the mobile telecommunications market, it can be stretched. What is apparent is that the basic idea of pre-payment is not a foreign idea. The issue is in how steady access to quality medical care can be achieved. There is need therefore, need to ask how private insurance can better provide realistic choices to citizens of West Africa. The options available for middle income West Africans and if private sector can play a role for poor in the society. The absence of a viable private health insurance market is major challenge for this region (Global Health Africa, 2013)

2.2.2 Staffing of health expertise in ECOWAS

The World Health Organisation (2016) suggests as a based standard, one doctor per 5,000 residents of a particular geographical area. This criterion has not being adhered to by many West African states. Countries such as Benin, Burkina Faso, Senegal, Niger, Sierre Leone and Mali all have less than ten doctors to 100,000 residents. This challenge of staffing is also applicable to hospital administration and nursing. Although Africa accounts for more than 40% of the world communicable diseases, the total workforce of this region is less than 5%. The number of healthcare workers provided in West African nations is lower than needed. Equally disturbing is the concentration of the available health care workers in the urban centres. This compounds the challenge faced by the rural dweller in accessing quality health care services. The issue regarding the shortage of healthcare worker is partly due to financing problems. Many midwives and nurses are either not duly employed or they are underemployed. The reason has been that nations lack the necessary funds to afford a decent salary for these health workers. The resultant effect of this unemployment or underemployment of the available health workers is the flight of medical expertise and/or professionals from the region to other parts of the world in search of better opportunities. This situation is popularly known as a ‘brain-drain’. It has been found that out of every five
physicians born in Africa and one of ten nurses born in Africa are practicing outside the region (Obiechefu, 2012). According to Akukwe (2006), the emigration of health workers to the western world has posed a serious challenge to health care delivery in Africa as a whole. Hence, many ECOWAS governments rely on just a handful of staff or temporary foreign health workers. This reliance has delivered a framework whereby instead of improving the existing healthcare structure that would create and maintain a bigger labour force, there is a habit of hoping for and needing the availability of foreign assistance to help alleviate an inadequately working course of action (United Nations Programme on HIV and AIDS, 2006; World Health Organization, 2006).

2.2.3 Healthcare Financing

According to the World Health Organization (2013) over 25% of health care funding in one-third of African countries come from the Donors funds. This statistic is equally true for the ECOWAS countries. Most of the funds from foreign source are cyclical in nature though it is the major source of health funding in ECOWAS. Many times, the recipient countries are not notified before the funding is cut, hence their inability to absorb the financial implications. Over 60% of health funding comes from out-of-pocket spending and this is equally alarming. This out-of-pocket payment come in terms of payments made to private health service providers and fees paid at public facilities. Generating a source of revenue to fund healthcare is quite a challenge for many West African countries. One major source of revenue for the governments in West Africa is the user fee. Primary healthcare services such as family planning, immunization and treating communicable diseases are excluded from the payment. Hence, the issue is that many low-income families are not cared for. This source of funding healthcare is unpopular in so many countries of this region thus they seek to abolish it. However, this abolition will create the need for an adequate source of extra revenue but this can be a great obstacle for the poor countries (Akukwe, 2006).

2.2.4 Disease outbreak in ECOWAS Countries:

Some of the most devastating diseases in western Africa include Malaria, Ebola, Tuberculosis, HIV/AIDS, and Meningococcal meningitis, although prevalence may vary from one West African country to another as well as from one geographical area to another within the same country.
2.2.4.1 Malaria

Malaria is a major risk for most travellers to West Africa and poses an increasing risk to their health in West Africa. The development of drug resistance by this endemic disease in West Africa has further compounded the risk to the economic system of West African region (Mandal et al., 2011; Boggild et al., 2015). This life-threatening disease caused by a parasite carried by the female Anopheles mosquitoes has posed serious threat to life (Holt et al., 2002). Several risk factors for acquiring malaria during travel have been identified, of which destination is the most important. West Africa accounts for approximately two thirds of all cases reported in the UK, with travellers to Nigeria and Ghana making up half of all imported Plasmodium falciparum infections (Smith et al., 2008). According to the latest WHO (2017) evaluations, published in December 2016, about 212 million malaria cases were reported in 2015 while there were about malaria deaths 429 000. In 2015, the region was home to 90% of malaria cases and 92% of malaria deaths. In West Africa, malaria has been the primary cause of morbidity and mortality of life also according to statistics by the WHO (2007). The main causes of children’s death under the ages of 5 years (21%) in West Africa is malaria. Most West African countries do not have the framework or the necessary resources to organise sustainable and effective anti-malaria campaigns. Over 30% of malaria cases occurred in this region and out of these, Nigeria and Ghana cases alone accounted for 40%. Equally observed is that of the world malaria cases reported by United Nations International Children's Emergency Fund (UNICEF), Nigeria accounted for 17%, Ghana account for 24%, Burkina Faso (9%), Senegal (7%), Guinea (6%), Mali, Benin and Liberia (5%), and other West African countries (22%) (Cornia Giovanni, 2001; United Nations Fund for Population Activities, 2003; UNICEF, 2005).

2.2.4.2 Ebola

The 2014 West African virus disease outbreak started 2013 December, at Guinea forest (Gatherer, 2014). This is the most destructive, perplexing and biggest ever seen virus crisis which has devastated the communities of West Africa. By 20 April 2014, 242 suspected cases had resulted in a total of 147 deaths in Guinea and Liberia. Till date the West African Ebola outbreak is the deadliest globally. In Guinea, Liberia, Sierra Leone, Nigeria and Mali, not less than 5,176 individuals have died of this virus with a fatality rate of 61% so far. With this percentage of 61%, this West African Ebola virus epidemic is seen as more severe than that in Gabon in 1994 with about 60% fatality rate (Baize et al., 2014; Meltzer et al., 2014). The
Ebola outbreak has spread so fast across the West Africa region. This had prevented West African communities from getting their basic essentials, such as food, water, shelter, healthcare delivery; moreover the requests of option reactions to the outbreak of Ebola have totally overwhelmed the capacity of the government and other partners to respond. (Leroy et al., 2014; Karesh et al., 2012). All efforts proved abortive in the eradication of the Ebola virus. WHO (2016) identified that about 152 health workers in Liberia have been infected with the virus. A total number of 571 health-care workers (HCWs) were found to have contacted this virus out of this total, 332 were from Liberia, 11 from Nigeria, Mali had 2, Guinea had 93, 128 in Sierra Leone; 4 in the United States of America and 1 in Spain. Tambo (2014) reported 325 HCWs dead of this virus infection. The disease is transmitted through direct contact with the bodily fluids of an infected person which include saliva, mucus, blood, sweat, urine, vomit, faeces and breast milk. WHO and respective governments reported a total of 23,253 suspected cases and 9380 deaths as at 15 February 2015, and of these cases the three worst affected West African countries were Liberia Guinea and Sierra Leone. Each of these countries in response notified the WHO of this pandemic (Etuk, 2015). As at the end of February 2016, the Ebola epidemic in West Africa destroyed lives and devastated communities with an astounding total number of cases and deaths reported at 28,663and 11,324, respectively. This led to adverse effects on trade from this region. The acute pressure of the Ebola outbreak on trade has led to a fall in the export trade from the West African economy which is estimated to be down by as much as 30% according to UK’s shipping consultant Drewry. The economic growth in Sierra Leone also fell to 4.0% from the 11.3% projected before the outbreak (Etuk, 2015; Omilabu et al., 2016). This threat to life has called for concern on the devastating effect it will pose to health within the ECOWAS. The update on the Ebola outbreak in the ECOWAS region extracted from the WHO report for 2016 is presented in table 2.1.
Table 2.1 Ebola epidemics in west Africa 2014-2016

<table>
<thead>
<tr>
<th>COUNTRY</th>
<th>CASES</th>
<th>DEATHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liberia</td>
<td>10,678</td>
<td>4,810</td>
</tr>
<tr>
<td>Sierra Leone</td>
<td>14,142</td>
<td>3,956</td>
</tr>
<tr>
<td>Guinea</td>
<td>3,814</td>
<td>2,544</td>
</tr>
<tr>
<td>Nigeria</td>
<td>20</td>
<td>8</td>
</tr>
<tr>
<td>Mali</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Senegal</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>28,663</strong></td>
<td><strong>11,324</strong></td>
</tr>
</tbody>
</table>

Extracted from table 4 of Espinel, et al., 2016.

2.2.4.3. HIV/AIDS

HIV stands for Human Immunodeficiency Virus. It is a virus that can result in AIDS (acquired immunodeficiency syndrome) if it is not treated. This virus attacks the immune system of the body particularly the CD4 cells which are capable of fighting infections. When an individual is infected with this virus, the immune system will be low such that opportunistic infectious diseases and certain cancers may then develop. This infection can be spread through sexual intercourse, from mother to child during birth or during breast-feeding and blood transfusions. When an individual is affected by a continuous progression of such diseases, they are said to suffer from AIDS, the Acquired Immunodeficiency Syndrome (WHO, 2002; Preker, 2004; Cornia Giovanni, 2001). HIV and AIDS is one the main health and human development challenges in recent time. The middle class were the most vulnerable at the early stage of HIV pandemic in the sub-Saharan Africa but as the epidemic gets deepened, this shifted to the poorer population (Gillespie & Greener, 2006). More than three decades after the first reported case of AIDS, sub-Saharan Africa continues to be the most heavily affected region in the world. Although, West Africa is less affected compared to southern and eastern regions of Africa, nonetheless none of the countries in West Africa is
left untouched. However, Côte d’Ivoire is the most affected with 7.1% of its adult population estimated to be HIV positive (UNAIDS/WHO, 2006). The prevalence of HIV varies from 15.9% in The Gambia to 68% in Benin among female sex workers, while it ranged from 9.8% in The Gambia to 34.9% in Nigeria among MSM (men sex workers). HIV prevalence in west Africa remains comparatively low, with the adult prevalence in the general population assessed at 2% or lower except for Côte d’Ivoire and Nigeria where adult prevalence is estimated at 3.4 and 3.5%, respectively (Djomand et al., 2014). In selected geographical area and cities in Ghana, Benin and Togo, 32–84% of prevalent cases among men had been attributed to sexual contact with female sex workers. HIV prevalence in female sex workers varies within and across west African settings, with reported cases estimated over the past 5 years of 15.9% in The Gambia, 20% in Nigeria, 20.1% in Senegal, 25.7% in Bobo-Dioulasso, Burkina Faso, 26.6% in Côte d’Ivoire, 30.4% in Cotonou, Benin, 45.4% in Lome, Togo and up to 68.6% in a study in Ghana and Republic of Benin (Labbe AC et al., 2012; Peitzmeier et al., 2013; Eluwa et al., 2012; Behanzin et al., 2013). HIV prevalence was at least twice as high among women than men in Cameroon, Central African Republic (CAR), Congo, Cote d’Ivoire, Ghana, and Guinea in 2012 and in 2015, 410,000 people became infected with HIV in West and Central Africa, 64% of new infections among young people (15-24 years) in the region occurred among young women. Previous report identified that an estimated 60% of new HIV infections occurred in Nigeria, and another 30% occurred in Cameroon, Chad, Côte d’Ivoire, Democratic Republic of the Congo (DRC), Ghana, Guinea and Mali (USAID, 2012; UNAIDS, 2016). The massive spread of this epidemic can also be traced back to lack of proper health care delivery that will educate local communities in West Africa because when the poor do get infected the vulnerability of their families and communities to the impacts of the disease are generally more severe than is the case for the better off.

2.2.4.4. Tuberculosis

According to WHO (2015), Tuberculosis or TB is a chronic infectious disease caused by a mycobacterium, mainly Mycobacterium tuberculosis (MTB) or Koch’s bacillus (KB). With the emergence of resistance to drugs has made it a high risk to the detriment of human health. Drug-resistant tuberculosis (TB) is a global public health problem. In 2008, a new research network, the West African Network of Excellence for Tuberculosis, AIDS and Malaria (WANETAM), was founded, made up of nine study locations from eight West African countries (The Gambia, Nigeria, Burkina Faso, Mali, Ghana, Guinea-Bissau, Senegal and
There has been a 10% increase in the rate of TB cases in the African region on a yearly basis and in 1999; two thirds of the two million new TB patients were also infected by HIV. It is globally recognised that the only infectious disease that kills more than TB is AIDS (Gehre et al., 2016; Kuaban et al., 2015; Ani et al., 2009; Daniel and Osman, 2011; Otu et al., 2014). West Africa consists of 15 countries with 245 million inhabitants. It was discovered in 2015 globally that 480 000 people developed multidrug-resistant-TB (MDR-TB). There is an increased rate in different geographical areas; Nigeria 48%, Ghana 6%, Cote d’Ivoire 8%, Mali 5%, Burkina Faso 4%, Senegal 4%, Sierra Leone 3% and other West African countries have 22% (WHO, 2007).

2.2.4.5. Meningococcal Meningitis

This is a serious infection of the thin lining that surrounds the brain and spinal cord. Before 2010 and the mass preventive immunization campaigns, Group A meningococcus accounted for an estimated 80-85% of all cases in the meningitis belt, with epidemics occurring at intervals of 7-14 years. The Meningococcal Meningitis is a bacterial form of meningitis and causes severe a serious infection of the meninges which affects membrane of the brain. It can cause severe brain damage and if it is not treated it has fatal in 50% of cases. Several different bacteria can cause meningitis. Neisseria meningitides is the one with the potential to cause large epidemics. Twelve serogroups of N.meningitidis have been recognized and out of these twelve, six (A, B, C, W, X, and Y) can cause pandemics. The distribution and epidemic of this disease in geographical areas varies based on the serogroup. This bacterium is transmitted from a carrier to uninfected person through respiratory droplets. Close contact with an infected person in the form of kissing, sneezing or coughing on someone or living in close quarters with an infected person aids the spread of the disease. The average incubation period is 4 days, but can range between 2 and 10 days.
Table 2.2 Epidemics of meningococcal disease, from 1994-1997

<table>
<thead>
<tr>
<th>COUNTRY</th>
<th>YEAR</th>
<th>NO. OF CASES</th>
<th>CFR</th>
<th>SERO GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nigeria</td>
<td>1996</td>
<td>108,588</td>
<td>11.2</td>
<td>A</td>
</tr>
<tr>
<td>Burkina Faso</td>
<td>1996</td>
<td>42,129</td>
<td>10</td>
<td>A</td>
</tr>
<tr>
<td></td>
<td>1997</td>
<td>22,305</td>
<td>11.3</td>
<td>A</td>
</tr>
<tr>
<td>Mali</td>
<td>1996</td>
<td>7,254</td>
<td>11.5</td>
<td>A</td>
</tr>
<tr>
<td></td>
<td>1997</td>
<td>11,228</td>
<td>10.1</td>
<td>A</td>
</tr>
<tr>
<td>Niger</td>
<td>1995</td>
<td>26,738</td>
<td></td>
<td>A</td>
</tr>
<tr>
<td></td>
<td>1996</td>
<td>16,145</td>
<td>9.9</td>
<td>A</td>
</tr>
</tbody>
</table>


CFR= case-fatality rate

The epidemic wave then spread in West Africa, including Niger (more than 25,000 cases notified in 1995, more than 16,000 cases in 1996), Burkina Faso (more than 40,000 reported cases in 1996, more than 20,000 in 1997), Nigeria (more than 105,000 reported cases in 1996), and Mali (more than 7,000 reported cases in 1996, more than 10,000 in 1997).

2.3 HEALTH EXPENDITURE IN ECOWAS COUNTRIES

The issue of health has being on going for some time now. This led a hundred and eighty-nine heads of states to adopt the millennium declaration in September 2000 as a way of improving both economic and social conditions in poor countries of world by 2015. This declaration led to the formulation of eight goals, which will enable the assessment of the progress made as regards the declaration. Out of these eight goals, three were on health and two others have health components (OAU, 2001; WHO, 2011)
From WHO (2011) given the above stated goals, the heads of states of African union met in Abuja –Nigeria in April 2001 and they all pledged that at least 15% of their annual budget would be devoted to improving the health sector and they also pleaded with the donors countries to render their support of 0.7% of their annual Gross National Product (GNP) to developing nations as ODA (Official Development Assistance). This however, revealed that low-income countries have inadequate resources needed for improvement of health (Tandon & Cashin, 2010; World Health Organization, 2011a)

As regards the three health related millennium development goals (MDGs), twenty-seven countries were found not to have made any significant progress since the goals were set while only three were found to have made progress and described as being on track (WHO, 2011). On the other hand, with respect to the Abuja declaration since 2001, the world health organization report showed that only one African country (Tanzania) met the target of investing at least 15% of the annual budget on health. Eleven countries reduced their expenditure on health while twenty-six countries were found to have increased their expenditure on health but in the remaining nine countries neither increase nor decrease was seen (Tandon & Cashin, 2010; WHO, 2011)

There is increase in the real per capita public spending on health that is financed with domestic source. This increase is from US$ 9.4 to US$13.4 during the period 2001-2011. Thirty-three national governments of Africa spend less than US$33 on health (Tandon & Cashin, 2010; WHO, 2011a). These national governments include all the ECOWAS countries. By implication, ECOWAS countries spend less than US$33 per capita on the health of their population. This amount of US$ 33 is low relative to the amount estimated by the High Level Task Forces to achieve the Millennium Development Goals in low-income countries as well as the US$54 suggested by WHO (2009).WHO (2011) observed that only thirteen countries in the African region spend more than US$ 33 per capita on the health of their population, of which 9 are middle-income countries. It is observed that achieving the MDGs health goals is a function of both the ratio of government spending allocated to health and the level of general government spending. Low per capita health expenditure will result in inability to achieve the MDGs health goals. Nevertheless, if there is increase in the ratio as well as per capita expenditure, then there will be less financial challenge in achieving the MDGs health goals (UN, 2000).

About 25% of healthcare expenditure of a third in Africa comes from donor funding. This is also applicable to the ECOWAS countries. This source of health financing cannot be relied
upon because donors can reduce funding without any prior knowledge by the recipient countries to prepare themselves to cater for the impact of this form of financial cut. In addition, in Africa, about 60% of health funding is made from out-of-pocket, which includes cash payment to traditional healers and private healthcare providers as well as fees paid at public facilities. (WHO, 2010; Novignon et al., 2012).

Figure 2.1 shows the trend of total health expenditure in ECOWAS countries. It is obvious from the graph that all countries have experienced fluctuations in health expenditure for the period under investigation. Equally noticeable from the graph is that all countries experienced a slow but steady decline of total health expenditure from 1995-1999. However, countries such as Nigeria had a sharp fall in 1999 - 2003, Liberia’s health expenditure increased up to 2002 but nose-dived in 2003 while Guinea’s total health expenditure fell in 2005 but began to rise in 2006. Although there was a rise in the total expenditure for almost all countries from 2006, a fall was experienced in 2009 after which it began to rise but this rise is quite slow.

**Figure 2.1 TOTAL HEALTH EXPENDITURE IN ECOWAS**

Data source: World Development Indicator
It is worthy to note that in the period under consideration, the West African community has witnessed the outbreak of the Ebola Virus from 2013-2014. Therefore, in addition to the ongoing fight against HIV/AIDS, Tuberculosis and Malaria, the rise in total health expenditure ought to have been rapid.

2.4 ECONOMIC GROWTH IN ECOWAS COUNTRIES

In simple terms economic growth is taken to mean the rise in the real Gross Domestic Product of an economy over a period. It is usually used to measure the health and performance of the economy under investigation.

The African continent growth rate was expected to increase from 3.4% in 2011 to 4.5% and 4.8% in 2012 and 2013 respectively. This growth on the continent was attributed to the rise in government spending on infrastructure, the gradual recovery in the northern African economies, and internal demand in countries of the continent as well as strong commodity prices. The growth in this continent varies from one region to another and this is shown in the figure below:

Figure 2.2: PROGRESSION OF REAL GDP GROWTH IN AFRICAN REGIONS (2011-2013)

![Graph showing economic growth in African regions](image_url)

Adopted from ECOWAS 2012 annual report
The chart above reveals the variation in the expected growth level of regions. The South Africa region will grow from 3.5% recorded in 2011 to 4.4% in 2012 and 2013. The growth of central Africa will grow from 4% in 2011 to 4.9% in 2012 but will fall slightly to 4.8% in 2013. In North Africa, the expected growth is 0.5% in 2011 to 3.1% and 5.4% in 2012 and 2013 respectively. However, for the eastern region, though the rate of growth is reasonable, there will be a slight fall from 6.4% in 2011 to 6.3 % in 2012 and fall further to 5.8% in 2013. It is also obvious from the graph that like East Africa, West Africa will have a strong growth rate over this period unlike the other regions. The economic growth of this region will be expected to increase from 5.9% in 2011 to 6.9% and 6.4% in 2012 and 2013 respectively.

The growth in 2012 as predicted above is however due to benefits generated from increased exports of natural resources in some of the countries, such as uranium and oil in Niger, iron ore in Sierra Leone and peace in Côte d’Ivoire among other factors.

According to the ECOWAS 2012 annual report, the rate of growth stood at 6.6% and this rose to 7% in 2013. With this increase in 2013, the African Development Bank (AFDB) viewed the region as the economy with most growth in Africa. In the words of the president of the ECOWAS commission, the economic growth is accredited to performance of the secondary sector particularly the mining sector. The highest growth of 13.2% was recorded by Sierra Leone while the lowest of 0.5% was recorded in Cape Verde.

As seen from the foregoing, the growth of this sub-region has fluctuated over the last few years. For example in the individual countries, the per capita GDP of this sub-region lies between USD 800 and USD 4,400 for Niger and Cape Verde respectively (ECOWAS, 2015). The reason for the fluctuation in the growth level in this region can be attributed to prevalence of some diseases in the region. The growth of some countries in this sub-region has been adversely affected with the outbreak of diseases such as Ebola. For instance, Sierra Leone, whose growth was projected to be 11.3% prior to the outbreak, declined to 4.0%. The Gambia was also negatively affected. About 12% of the county’s GDP emanates from tourism but due to prevalence of the Ebola epidemic, hotel reservations for tourism purposes fell by about 65% as noted by Nshimyumuremyi (2014) and World Bank (2014b). The implication of this fall is a decline in growth since even the big economies of the region such as Nigeria, Ghana and Cote d’Ivoire which could help in cushioning this effect were also not left out of the epidemic. In Lagos also, after the first case of Ebola in Nigeria, sales were estimated to have fallen between 20%-40% while in Senegal, there was fall in occupancy in the coastal region to 40%.
From UN-DESA (2015) and ECOWAS (2015), it is observed that the West African region recorded a fall in growth rates, from 6.1% 2014 to 4.2% in 2015. This fall is said to have occurred as a result of the decline in the price of primary commodities particularly oil. The level of growth of the individual countries of this region has also been different. For instance while countries such as the Gambia, Côte d'Ivoire, Senegal, Burkina Faso and Guinea Bissau witnessed a rise in the growth level from 0.5% to 7%, 8.5% to 9.5%, 4.7% to 5.4% and 4% to 4.4% and 2.9% to 4.7%, respectively in 2014 and 2015, other countries such as Benin, Togo, Mali, and Niger experienced a fall in their growth level in 2015 which stood at 5.2%, 5.8%, 4.9% and 4.4% as against their growth in 2014 which was 6.5%, 5.9%, 7.2% and 6.9 respectively. The fall in oil price was expected to negatively affect the growth of Nigeria and Ghana. Nigeria’s growth level fell from 6.3% in 2014 to 4% 2015 while Ghana’s fell from 4% in 2014 to 3.5% in 2015.

It was equally noted that the outbreak of Ebola has adverse effect on the growth of those countries affected. For instance the most affected countries (Liberia, Sierra Leone and Guinea) experienced a fall in their growth. Liberia and Guinea had a growth rate of 0.9% in 2015 against 0.7% and 1.1% in 2014 respectively while Sierra Leone fell from 4.6% in 2014 to -21.5% in 2015 (UN-DESA, 2015 and ECOWAS, 2015). Fig 2.3 shows the trend of growth in the countries of ECOWAS. The trend indicated that from 1994 to 2014 almost all the countries have witnessed some growth over the years. However, it is obvious from the graph that there is fluctuation over this period. For example, in 2009 virtually all countries experienced decline in the per capita GDP. Nevertheless, the fall is more severe in some countries compared to others. Nigeria witnessed a sharp fall in 2009 compared to others. It can also be seen from the graph that after 2009, though the per capita GDP increased for almost all countries, the rise has been slow.
2.5 GROSS NATIONAL INCOME PER CAPITA FOR THE ECOWAS COUNTRIES FROM 2013-2016

The economic performance of a country can be determined by observing the level of the countries income per capita and the GDP per capita. Gross National Income per capita is the total income of country divided by the population of that particular country in a given period of time. In other words it the sum of all value added by all producers resident in a country plus product taxes minus subsidies that are not counted in the estimation of output plus net receipts of primary income from abroad divided by the number of people resident in the country. Since this measures the income an individual earns in a particular country in a given

Data source: World Development Indicator
time period, it thus enhances comparisons of economic performance across economies. It reveals the relative standard of living in those countries compared.

**Fig 2.4 Per Capita Income for the ECOWAS COUNTRIES FROM 2013-2016**

![Per capita income for ECOWAS Countries](image)

*Author’s Design*

*Data Source: World Bank Development indicator*

From the Fig 2.4 above, in line with Diop *et al.*, (2010), it is observed that only a few countries in the ECOWAS region have relatively high income per capita for the period of 2013-2016. Cape Verde has the highest income per capital for the four years examined followed by Nigeria, Ghana, Côte d’Ivoire, Senegal, Benin, Mali, Guinea, Burkina Faso, Guinea Bissau. In Countries such as The Gambia, Niger, the income per capita is below 500US$ for the period examined. In Sierra Leone although the income per capita was rose from 650US$ to 690US$ IN 2013 and 2014, it fell to less than 500US$ for the year 2016.
Togo on the other hand, had less than 500US$ in 2013 but rose slightly to 540US$ the remaining three years. The Liberia has the least income per capita for the period examine. This could be due to that fact that it was adversely affected by the outbreak of Ebola within this period.

2.6 IMPORTANCE OF HEALTH FUNDING ON THE GROWTH OF THE ECONOMIES

It is observed from literatures that there should be a two-way direction of causation between income and health. For instance, Mushkin (1962) and Van Zon and Muysken (2001) regard health expenditure as a form of investment in human capital, and since Lucas (1988) saw human capital as a driver of economic growth, it then implies that increasing the level of investment in health sector or health care has the capacity to increase income level of a country.

Although it is captured in literature that increased national income proxy by GDP can enhance public health, an undeniable link seems to exist between improved health and the level of GDP, but it has not been well acknowledged publicly (Martin, et al., 2012). With increase in health expenditure, there will be an increase in the supply of labour as well as productivity and these will lead to increase in income (Ziesemer, 2003). Martin, et al. (2012) equally observed that diseases and sickness depress both the capacity and size of the work force by inhibiting access to education as well as hindering FDI (foreign direct investment). Accordingly, four major ways were observed for which investment targeted at improving health could impact the level of per capita GPD and these include: populations in good physical and mental state or healthy are productive economically, health expenditure has a multiplier effect based on the Keynesian approach, improvement in the health of the people symbolizes in itself and of itself an outcome of the growth and development, and addressing health issues before they occur will reduce the additional costs of healthcare which occur as a result of treatment of opportunistic infections such as those associated with HIV. Thus, they were of the view that if low investment in health system and health persist, growth in income or the prosperity of the nations will be impeded, such that they call for health to be viewed as a main driver of the economy and that there should be adequate investment in health, more specifically in less developed economies.

The role of health in economic growth cannot be over emphasised as it plays a substantial role in economic growth. For instance, a long-term study for England done by Robert Fogel,
a Nobel Prize winner in Economics, clearly demonstrates this as well as World Bank (2003). Fogel (1991) observed that chronic diseases are not the only channel via which acute malnutrition limits the labour force productivity but that even if the average quantity of calories was low in impoverished nations, the participation rate of labour and their productivity measures will still be low mostly if the labour hours were adjusted for intensity.

Based on World Bank estimates, in developing nations or middle-income nations with considerable acceptable quality of institutions, if health expenditure increases by 10% of the GDP, there will be a fall of about 4.14%, 7% and 0.69% in low weight children below the ages of five, rate of maternal mortality and child mortality respectively (World Bank, 2004).

According to Barro (1996) in a panel study of 100 countries spanning from 1960 to 1990, there is a positive nexus between health and growth. It was found that health positively impacts growth. For instance, it was observed that life expectancy impacts the growth level. Thus a 40% increase in life expectancy will cause the rate of growth to rise by 1.4% annually. In a similar sense, the annual growth was observed to rise by 0.3% with a 10% fall in malaria by Gallup and Sachs (2000) and malnutrition was found to impact negatively on the annual GDP per capita growth globally. This fall in the per capita GDP was observed to between 0.23% and 4.7%. The impact of health on the growth of the Caribbean and Latin America economy was examined by measuring the probability of surviving to the next age bracket, and a strong relationship was found to exist between them (Mayer, 2001b).

Eneji, et al., (2013) examined the relationship between Government expenditure on health, health status and productivity of the Nigerian economic for the period 1999-2012. The study employed both secondary data and questionnaire for this analysis and it was found that in Nigeria at this period of time, there was weak causal relationship between these variables. Hence, they recommended a comprehensive health care coverage such that individuals can easily have access to good health care. They also suggest that investment in innovation and research were essential for innovation in environmental technologies and health care as well as the competitiveness of the economy.

Berger and Messer (2002) investigates the effects of public health expenditure, insurance coverage and other factors on health outcomes within the health production model for twenty OECD economies from 1960 to 1992. The study found that both level of health expenditure and the expenditure mix are important in determining the rate of mortality. They observed that increase in health expenditure leads to lower mortality rate in developed economies but
the share of health care expenditure financed by the government has positive impact on mortality. This positive relationship implies that a rise in the expenditure mix will cause mortality rate to be higher. Thus they suggest that, as much as it is vital to increase the expenditure level, the proportion financed by the government may be limited so as to avoid the rise in mortality rate. To these authors, health is a form of capital; hence it is both an investment good and consumption good so it leads to both indirect and direct benefits. The indirect benefits occur via increase in productivity, increased wages and fewer days of sickness while as a consumption good, it produces direct benefit.

2.7 CONCLUSION

This chapter focused on examining health issues in the ECOWAS sub-region, examined the expenditures on healthcare, the growth of the region as well as the importance of health expenditure on economies in general. It was found that the region has been challenged with some diseases and has set up an institution to address these health issues in member countries. Also observed from this chapter is that all countries have experienced fluctuations in health expenditure for the period under investigation. Although there was a rise in the total expenditure for almost all countries from 2006, a fall was experienced in 2009 after which it began to rise but this rise was quite slow. This fall in health expenditure could lend explanation to the fall in growth experienced in virtually all countries, though the fall is more severe in some countries compared to others such as Nigeria, which witnessed a sharp fall in 2009. It can also be seen from the graph that after 2009, though the per capita GDP increased for almost all countries, the rise has been slow. One quick finding here is that, increased health expenditure will result in increased GDP per capita. However, this will be subject to econometric analysis for verification.
CHAPTER 3

THEORETICAL AND EMPIRICAL LITERATURE

3.1 INTRODUCTION

This chapter focuses on the literature underpinning the study. Theoretical and empirical literature is therefore discussed to give direction to the study. Therefore, the neoclassical, endogenous growth models as well as the Keynesian growth model are explained as the theoretical framework. Also this chapter captures the necessary empirical literature and, on the basis of this literature, the necessary variables for the model of this study will be identified.

3.2 THEORETICAL FRAMEWORK

The focus at this level is a brief review of the various underlying expenditure theories as well as growth theories, particularly the ones which captured the inclusion of human capital in explaining the growth of economies. The term human capital is coined to explain the stock of health and knowledge contained in labour (Acemoglu & Autor, 2011). The idea of human capital springs from the view that investment in health and knowledge is made so as to gain returns as it is expected that investment in physical capital should yield some returns. The investment in human capital is done through on-the-job training, schooling, healthcare and exercise. It is important to know that, like the physical capital, human capital also depreciates over time and this view also holds for health. Hence, increasing the investment in health will help in preventing health capital from wearing down fast. Some of the theories examined in this chapter include the Musgrave theory of expenditure, the Wagner theory of increasing state activities, Peacock and Wiseman theory of expenditure, Keynesian growth theory, neoclassical growth model and the endogenous growth model.

3.2.1 Wagner’s Theory of Expenditure

This theory of state expenditure was propounded by a German economist, Adolph Wagner (1835-1717). This theory postulates that for any economy, the share of government spending will increase with increase in the income. It was predicted that a developed economy would be followed by a rise in the share of government spending in gross domestic product. Based on this law, increase in the real income of an economy will cause that in the long run the share of government expenditure relative to income would increase (Wagner, 1983). This
theory, however, follows the classical school of thought in explaining the relationship between income and public expenditure (Chang et al., 2004). From the classical view point, causality is expected to run from income to public expenditure. This is contrary to the Keynesian view which is of the opinion that public expenditure would stimulate economic growth. By implication, causality runs from public expenditure to economic growth. Wagner (1883) found increased economic growth or the rise in per capita income as the main determinant of public expenditure after observing historical proof for so many developed economies.

Studies such as Peacock and Wiseman (1967) and Musgrave (1969) have supported the view raised by Wagner (1983) that increase in the per capita income has the tendency in the long run to increase the share of public expenditure relative to income by employing econometric techniques. These studies both modelled government expenditure as a function of the Gross Domestic Product. Although there are other versions of Wagner’s law, this study will only follow the version formulated by Peacock and Wiseman.

### 3.2.2 Keynesian Theory of Expenditure

The expenditure theory examined in this study is the Keynesian theory of expenditure. This theory was made popular during the great depression of 1920 – 1930 by John Maynard Keynes. Before the 20th century, it was believed by the classical school of thought that better return can be achieved with the funds in the hands of individuals but the role of government spending in influencing income level in the economy. The theory explains how in the short run, aggregate demand or what is known as the total spending in an economy can influence the output of the economy, particularly in times of economic downturn.

In Keynes view, the economy cannot achieve full employment by itself and effective demand is seen to play a vital role in the growth of the economy which will in turn move the economy towards full employment. Hence Keynes (1936) proposed that the government should intervene in the economy via spending so that the unused savings of private individuals can be put to use. The unused savings of the private individual can be borrowed by the government and put back into the economy through spending to stimulate economic activities. By so doing, in the Keynesian perspective, the government is capable of reversing the economic downturn. However, he suggests that spending by the government should be reduced after the economy has recovered from the depression so that it will not result in inflation which could occur due to growth in the economy. Barro (1990) equally posits that
productive expenditure by government will impact positively on the growth rate on the long run.

3.2.3 The Neoclassical Growth Model

This growth theory is based on the individual growth model developed by Solow (1956) and Swan (1956). This model can be referred to as the Solow-Swan growth model or what is popularly known as the Solow growth model since the contribution of Solow (1956) overshadowed that of Swan (1956). The steady-state equilibrium path of the neoclassical model is explained in terms of output-capital ratio in the Swan model but the capital-labour ratio used in the Solow model overpowered the Swan output-capital ratio.

This model is an extension of the Harrod-Domer (1946) growth model which introduced a new variable known as productivity in the growth model. In this model, labour is included as a factor of production as well as capital-output ratio. However, these are not constant as they were in the Harrod-Domer model. The output and capital-labour ratio were endogenised in the model as was done in Solow and Samuelson (1953) and Pilvin (1953). However, the contribution made was that of an easy but powerful means of finding the steady-state growth path of a single-commodity world.

Aside from the difference in the Solow (1956) and Swan (1956) models highlighted above, these models are basically identical. Growth rate of labour in the Swan model is exogenously determined. In this model also, returns to scale is said to be constant and there is no technical progress such that output grows at a rate intermediate between growths of capital and labour, and any change in the exogenous technical progress will cause output to also change. Solow (1956) also included technology in the production function and accordingly, the growth of output is determined by the steady exogenous technological progress.

According to Solow (1956), the explanation of the growth of an economy is based on the general production function of the form:

$$Y(t) = f(K(t), A(t)L(t)),$$

(3.1)

The t in the above equation represents the period (time). Y is the output at time t, K denotes the capital stock, A represents the technological progress and L represents the Labour supply all in time t. As observed from the function above, the technological progress (A) is labour augmenting. Hence, it is referred to as Harrod-neutral.
This production function assumes a constant return to scale on two arguments; effective labour and capital. Since the time factor enters into the function via capital, labour and knowledge or effectiveness of labour, any increase or change in output over time is dependent on the rate of change in the inputs. To be precise, over time output increases with any given amount of the inputs of capital and labour if there is technological advancement which depends on increase in knowledge.

Solow (1956) indicates that the exogenous steady-state level of per capita income is determined by the savings rate and growth of the population which are assumed to be exogenous in the typical neoclassical production function. With the assumption of a constant return to scale, doubling the amount of capital and labour while technological process is constant will double output. This can be expressed as below:

\[ F(\theta K, \theta AL) = \theta F(K, AL) \quad \text{for all } \theta \geq 0 \]  

(3.2)

The above expression indicates that if the individual arguments are multiplied by a positive constant \( \theta \), the output will also increase by the amount of \( \theta \). The assumption of constant return to scale is informed by two separate assumptions. One of these assumptions is that the economy is large enough so that all that is gained from the specialization is fully utilized which implies that if the factors (capital and labour) are doubled, the current inputs will be employed or utilized in the same manner as existing ones so that output also doubles. Secondly, it is assumed that aside from the three factors (capital, labour and knowledge or technological progress), other factors such as natural resources and land are irrelevant. Hence, only the assumption of a constant return to scale is considered relevant in the growth model.

From equation 3.2, with assumption of constant return to scale, if \( \theta = 1/AL \) then we arrive at

\[ F(1/AL*K, 1/AL*AL) = 1/AL*F(K, AL) \]  

(3.3)

The above equation (3.3) is the output equation with constant returns to scale. And multiplying out the above equation will result in equation 3.4 below:

\[ F(K/AL, 1) = 1/AL F[K, AL] \]  

(3.4)

Where \( K/AL \) is capital—labour ratio (the capital per labour) and \( F[K, AL]/AL = Y/AL \) is the output per labour. If \( y = Y/AL \), \( k = K/AL \) and \( F(k) = F(K/AL, 1) \) then equation (3.4) can be expressed as:
This implies that output per labour is a function of the capital-labour ratio. Based on this theory, change in technology has a greater impact on the economy, thus the economic growth cannot increase without improvement in technology.

In essence, the theory suggests that capital accumulation in an economy, and how this is used by people is crucial for growth of the economy. That is, the capital/labour relationship in an economy determines the growth or output of that economy. Based on the theory, technology is said to be labour augmenting meaning that it enhances or increases the productivity of labour.

Mankiw et al. (1992) found that, although the neoclassical model has done well from an empirical view point and given insight to the relative importance of the capital accumulation and technological advancement in explaining the growth level of a country, it is not without limitations. Some of these limitations are the complex picture of the convergence rather than as it is suggested in the model – only non-convergence was explained by the model and vast income differences across economies by technology. The model failed to explain the vast income variation across countries. Also, that technology which was identified as the main determinant of growth is assumed to be exogenous. It is equally observed that technology is just a residual and as seen, economic growth in the long run cannot be explained by the accumulation of capital. Other factors such as human capital accumulation, and investment in infrastructure, among others could be the drivers of growth hence the next section examines the endogenous growth model.

3.2.4 Endogenous Growth Model

The endogenous growth model is that model which was developed in responding to the pitfalls of the neoclassical growth model. As earlier stated, the neoclassical growth model identified the long-run growth as dependent on the rate of technological change, which is assumed to be exogenously determined (Solow, 1956, 1957). However, as observed by Romer (1987, 1989), the influence of savings on growth is way too large and he attributed this to positive externalities or spill over accruing from accumulation of capital. Lucas (1988) also opined that the difference in the growth of population cannot explain any significant changes in the GDP per capita across economies as suggested by the Solow model.

Works done by Arrow (1962), Uzawa (1965), and Sidrauski (1967) formed the basis for the research into a model that would replace that of the exogenous growth model. However,
further work in this regard by Romer (1986) and Lucas (1988) among others, overlooked technological change in the growth model and they are of the view that growth is dependent on the investment in human capital that has a spill-over effect on the economy and can reduce the diminishing return on the accumulation of capital (Barro and Xavier, 2003). The endogenous model is of the opinion that long-run growth of an economy is explicitly determined within the system or the model.

Lucas (1988) identified spill-over effects of investment in human capital as that which enhances the rate of technology and not investment in physical capital. Romer (1986), however, posits that spill over emanates from research efforts from private individuals that lead to improvement in the entire stock of knowledge. The major thrust of the endogenous growth model is the departure from the presumption of decreasing returns to capital and this was only achieved by including knowledge capital and human capital in the definition of capital.

According to Romer (1990), the growth of the economy is driven by the rate of technical change arising from within the economy due to investment decisions made by agents within the economy. The technological change here is neither a public good nor a conventional good; rather it is a partially excludable good and at the same time non-rival. Romer concluded that, the rate of growth is determined by the stock of human capital. He is of the opinion that having a substantial populace, that is; growth of population is inadequate to achieve growth but argued that integrating to the world markets will improve the rate of growth. Thus, growth in the volume of trade will positively affect the income level.

The basic Romer model is specified as below:

$$Y_i = AK_i^{\alpha}L_i^{1-\alpha}K^{-\beta}$$

But for simplicity all industries are assumed to be the same therefore an individual industry will employ similar quantities of the inputs (capital and labour). The aggregate production function is thus stated as:

$$Y = AK^{\alpha+\beta}L^{1-\alpha}$$

The factors included in the model by Romer are said to make $\beta$ equal zero so that the GDP per capita and the capita growth rate increase. The endogenous growth in this model is said to be dependent one investment and saving and not the exogenous factor (productivity). Based on Romer’s model, investment (technical spill over) will enable the idea of diminishing
return on capital to be checked. Basically, in the endogenous model $\alpha + \beta = 1$ implying that there is increasing returns to scale.

The major finding of the endogenous growth model is that it explicitly models the determinant of technology as endogenous and not taken to be exogenous as is the case of the neoclassical model. As Verbeck (2000) noted, the growth of the economy occurs as a result of advancement in technology, which basically relates to the capacity of the organizations to beneficially use its production facilities more adequately over time. This capacity is developed from the learning process to utilize the recently available productive resources more efficiently or figuring out how to adapt to quick change in the structure of production which industrial progress must infer.

### 3.3 EMPIRICAL LITERATURE

The empirical discourse on the relationship between health care expenditure and economic growth has gained so much attention since the publication of the paper by Newhouse (1977) whereby the relationship between income and medical care expenditure was examined for thirteen developed economies. Regressing per capita medical expenditure on the GDP per capita, this author found that medical expenditure had more than unit income elasticity.

Following this part, Hitiris and Posnett (1992) found a positive relationship between health expenditure per capita and income while Hitiris (1997) found the income elasticity of health expenditure to be between 1.14 and 1.17 for ten OECD economies for the period 1960 to 1991. It is worthy of note that researchers in this period used time series and they viewed health expenditure as a luxury good.

According to WHO’s Commission on Macroeconomics and Health (2001), the link of health to poverty reduction and economic growth is quite strong. Nevertheless, most low income countries, most especially in Sub-Saharan Africa (SSA), have been burdened with disease which has impeded the growth of the economies hence the need for this to be thoroughly addressed in any developmental strategy (World Health Organization, 2001).

In the current century, studies on the relationship between health expenditure and income for OECD have classified health care expenditure as a necessary good. Some of the studies such as Okunade and Murthy (2002) used time series methodology in analysing the relationship between health expenditure and income, and found a statistically significant and stable relationship between these variables. Jewel et al., (2003) employed panel analysis of the
relationship between health expenditure and GDP for 20 OECDs for the period 1960-1997 and they found that both variables are stationary about one or two breaks. Chakroun (2009) estimated both time and country specific income elasticities for seventeen OECDs for the period 1975-2003 with panel threshold regression model and found that health care is not a luxury but a necessity. Other studies such as Baltagi and Moscone (2010) adopted the non-stationary and cointegration approach to examine the relationship between health care expenditure and income for 20 OECDs. They found that health care expenditure is a necessary good and not a luxury. Mehrara et al., (2010) also used panel data for all OECDs during the period 1993-2007 in investigating the relationship between health care expenditure and income, and their results suggest that the income elasticity is about 2.59 for all the OECDs. These authors also found that this income elasticity of health expenditure is unvarying across the countries and over time.

Nyamwange (2012) follows a time series approach to investigate the effect of GDP per capita (GDPPC) on public health care expenditure in Kenya for the period 1982-2012. The main aim of the study was to examine the kind of relationship between these variables and to suggest appropriate policy measures for the country. Hence, in this study, healthcare expenditure was modelled as a function of per capita GDP alongside other control variables such as POP (population growth), inflation and number of physicians per 100,000 population. The results found indicate that health care is a necessary good. It was equally observed that a one percent increase in Gross Domestic Product per capita (GDPPC) will caused healthcare expenditure to increase by 0.024%. The study employed the Ordinary Least Square (OLS) regression techniques and test for cointegration and Granger causality. The Granger causality test showed that GDP per capita Granger cause public health care expenditure. Hence, it was concluded that public healthcare expenditure is a right endogenous variable.

A study by Barnay and Damette (2012) employed the time series approach to study the role of GPD per capita on health expenditure for the French economy during the period 1950-2009 accounting for structural breaks and non-linear long run relationship of healthcare expenditure and GDP. They controlled for ageing population, price effect, medical density and innovation proxy on the role of GDP per capita on healthcare expenditure per capita and the results found, indicate that the non-linear long run relationship among the variables emanates from the structural breaks and medical density at the end of the 70’s. They found low GDP elasticity to be explained by exogenous shock linked with health system policies in the mid-80’s and the changes through medical density in the country.
Santiago et al., (2013) investigated the relationship between health expenditure and income for thirty-one OECDs for the period 1970-2009 and focused on the variation among long and short run elasticity as well as the adjustment process through which health expenditure responds to both cyclical and trend component of the per capita GDP. In this study the health expenditure is modelled as a function of the lagged value of health expenditure, GDP per capita and percentage of the population above 64 years. The results showed that the income elasticity is 0.3 in the short run while it is 1.1 in the long run. They conclude that at 95% significance level, the null hypothesis for the long run can be rejected but it is accepted at 98% level. They equally found that health expenditure respond faster to the cyclical component than the trend component of GDP. It was discovered that health expenditure adjusted faster to changes in GDP per capita if the share of private expenditure on health care is higher. Nevertheless, there is no significant high income elasticity in the long run.

Bedir (2016) in the study on health expenditure and economic growth in developing countries adopted the modified version of the Granger (1969) causality test suggested by Toda and Yamamoto (1995), and Dolado and Lütkepohl (1996) for the emerging countries of Europe, the middle East, Africa and Asian countries and found that income is vital in the explanation of the variations in the healthcare expenditures among the countries. By implication, increased income in some of the emerging countries seems to stimulate expenditure on health care. A two-way causality was observed for the Czech Republic and Russian Federation. However, while the health view over income was supported by the evidence from Egypt, Hungary, the Korean Republic, South Africa, and the Philippines, the income view over health was supported by the evidence from Greece, the United Arab Emirates, Poland, Indonesia, China, and the Korean Republic.

Study performed by Wang (2010) showed that total health expenditure stimulates the growth of the economy. This study employed the panel and quantile regression technique in investigating the link between total health expenditure and economic growth for many countries. Results from the panel regression indicated that the growth of expenditure will cause a rise in economic growth. However, economic growth was found not to contribute essentially to the growth in total healthcare expenditure. The positive link between healthcare expenditure and economic growth is only obvious for countries with high and medium levels of economic growth while it is not so for countries with low level growth.

Autoregressive Distributed lag techniques were used by Mehrara and Musai (2011) to study the link between healthcare expenditure and economic growth in Iran for the period of 1970 –
2007 and the result showed that there is a long run relationship among the dependent variables: real GDP and the independent variables (capital stock, oil revenue, health spending and education). The marginal contribution of health spending to economic growth was found to be insignificant, nevertheless, it was discovered that healthcare spending is vital in reducing infant mortality in Iran.

Aurangzeb (2003) applied the co-integration and error correction modelling approach in studying the relationship between GDP and health expenditure per capita for Pakistan between the years 1973-2001 in the augmented Solow growth model. The study incorporated health expenditure as proxy for health capital in the model alongside openness variable which was used to capture the effect of technological advancement on growth. The study found that both in the long run and short run these variables (GDP and health expenditure) are positively and significantly related.

Elmi and Sadeghi (2012) equally examined the link between GDP and healthcare expenditure in developing countries from 1990-2009 using the panel cointegration technique and the Granger causality test within the framework of Vector Error Correction Model. While on the short run, causality was found to run from GDP to health expenditure, there was no causality from health care expenditure to GDP. Nevertheless, the long run result showed that there is cointegration among the variables and a bilateral causality was observed. Based on the existence of bilateral relationship between the variables found, it implies that the level and growth of health expenditure across these developing countries is influenced by the income of these countries on the long run and health expenditure is a vital factor in explaining economic growth of these countries.

Bakare and Olubokun (2011) employed the OLS estimation techniques to study the link between healthcare expenditure and growth in Nigeria between 1970-2008. The study investigated the trend in health expenditure within the country and further examined the rate at which this expenditure has impacted the growth of the country. The result of the analysis indicated that health expenditure has a positive impact on growth. Hence the authors were of the opinion that annual budgetary allocation for the health sector should be increased. And they also noted that it is not just enough to increase the expenditure, but that the funds should be properly allocated and used transparently.

Baldacci (2004) investigated the role played by health expenditures on the growth of one hundred and twenty emerging countries for the period 1975 - 2000. The panel data set was
used and the result indicates that expenditure on health within a period impacts on the growth of the economy within such period, but the lag expenditure shows no effect on growth. Hence, it is deduced that the effect of health expenditure is rather a flow not a stock effect.

Baldacci et al. (2008) investigated the channels that link social spending, human capital and growth for one hundred and eighteen countries for the period 1971-2000 with the use of panel data set. They built a different model for education and health capital, explicitly controlling for governance and incorporating nonlinearity, and the observed result showed that, spending on education and health has a significant positive effect on education and health capital which they found has a positive impact on growth. They however suggests that, to attain the millennium development goals (MDGs), apart from higher spending on education and health, there is a need to also intervene in other policies such as curtailing inflation and improving governance.

Bloom et al. (2004) examined the effect of health on economic growth using the approach of the production function. They included two variables: health and work experience in the aggregate model of growth, which are fundamental components of human capital. These variables were added alongside the basic growth components. Thus, they modelled growth (GDP) as a function of total factor productivity (TFP), labour force (L), physical capital (K) and human capital. They employed panel data analysis for countries which were observed every ten years from 1960 to 1990. The result indicates that there is a statistically significant positive relationship between health and economic growth. This outcome supports the need for increased investment in health, which is a form of human capital.

The World Bank (2004) examined the relationship between health expenditure and infant mortality by employing a panel analysis of states in India between 1980 and 1999 and found that health expenditure does not have any effect on the mortality rate after including linear time trend and the individual fixed effects in the model. This study also reduced the sample size by using fourteen states and four variables (14*4=56=N), included female literacy variable as well as an interaction term between income and health expenditure in the model, and a negative effect of health expenditure on the mortality rate was observed for poor states.

Boachie et al., (2014) used time series data to investigate the determinants of healthcare expenditure for Ghana for the period of 1970-2008. They employed the Elliot Rothenberg and Stock (ERS) point optimal unit root test and Engle-Granger cointegration approach to analyse the stationarity and cointegration property respectively. In this study, health
expenditure is modelled as a function of Real Gross Domestic Product (proxy for income), the level of healthiness is measured as life expectancy (LEXP), crude birth rate (CRB) which captured the yearly births per 1000 population, the environmental pollution is measured in terms of CO₂ emissions, urbanization (URP) was used to measure population living in the urban areas while Rup captured the number of the population residing in the rural areas and lastly inflation was included to measure the general price level in the economy. The FMOLS was used to generate the long run estimates since the variables were found to be a mixture I(1) and I(0)s and the result of this study indicates that there is long run cointegration among the variables. All the independent variables followed the a priori expectation except for the urbanization variable which is negative. There is a statistically significant positive relationship between Real GDP, life expectancy and crude birth rate and health expenditure, while there is insignificant positive relationship between inflation and CO₂ and health expenditure. The independent variables therefore, account for about 92 percent of the variation in health expenditure in Ghana for the period under investigation. Based on the results found, these authors were of the view that the variables included in the study, especially the RGDP, LEXP and CBR should be given more attention so as to improve health expenditure in the country.

Rajagopal (2016) employed the Bayesian approach within the framework of panel data due to the likely heterogeneity of the states in India over time. The state and time effects are taken in different intercept terms and the coefficient of regression reveals how Infant Mortality Rate responds to rise in state health expenditure. Health expenditure is proxy by the proportion of GDP allotted to healthcare and all calculations are performed with deviations from the mean. The pooled regression is used and this omits state and time heterogeneity. The coefficient of the pooled regression was used as a preceding coefficient for the real slope coefficient. The coefficients of the regression with a normal prior and a uniform prior with a fixed mean and homogeneously distributed variance were compared and it was concluded that a rise in the state expenditure has a negative effect on the rate of infant mortality for the Indian states.

Yaqub et al., (2012) employed the OLS approach to examine the relationship between health expenditure and three health outcomes, namely infant mortality rate, under-5 mortality rate and life expectancy in Nigeria, and captured the impact of governance using corruption perception index for the period 1980-2008. In this study, each of the health outcomes had two equations. In the first equation, the health outcomes were modelled as a function of GDP per capita, health expenditure and population, while in the second equation, each health outcome
was modelled as the first one but with inclusion of the corruption index to capture the impact of governance. The results obtained showed that health expenditure has a significant inverse relationship with infant mortality rate in the equation. It showed that, if health expenditure increases by 1%, infant mortality would decrease by 0.03%. Also, results of the under-5 model indicates that the health expenditure only has the right sign after including corruption index although it was not significant. The life expectancy model with the introduction of the governance variable showed that health expenditure has a positive significant relationship with life expectancy. It indicates that life expectancy would rise by 0.02% if health expenditures increase by 1%. They noted that there could be reverse causation between health expenditure and these health statuses and they can equally be determined jointly. Hence, to handle the issue of reverse causation, in addition to the OLS, these authors performed the Two-Stage-Least Square and they concluded that public health expenditure has a positive relationship with life expectancy but has negative relationship with infant and under-5 mortality rate.

Lv and Zhu (2014) Examined the relationship on the between per capita health care expenditure and per capita GDP for forty African countries grouped into lower middle income countries using the Semiparametric panel data for the period 1995 to 2009. Aside the per capita GDP as independent variable, the study also include population aged 65 and infant mortality rate in the model. It was found that income is significant in explaining per capita health expenditure so it was concluded that health care is not a luxury but a necessity. It equally found a negative relationship between health expenditure and infant mortality rate while the effect of the population aged 65 is not significant in determining health care expenditure.

Kiymaz et al., (2006) investigated the long-run relationship between the per capita private, public, and total health care expenditure and per capita gross domestic product and population growth for Turkey and reveals that health care expenditure and per capita gross domestic product and population growth are countergrated, health care expenditure and per capita gross domestic product was found to have bivariate cointegration. They concluded that that health care expenditure is luxury given that the income elasticity is greater than 1 for turkey. They also found that unilateral causality runs from per capita GDP to health expenditure.

Elisabeta et al., (2015) adopted the cross-country and time series analysis to estimate the relationship between life expectancy and health expenditure for one hundred and seventy five
countries of the world grouped based on geographical region and income for the period 1995-2010. The fixed effect model was estimated for a group of one hundred and seventy-five countries to examine the impact of health expenditure on life expectancy for the period while the difference in the life expectancy and health expenditure for 1995 and 2010 was examined with the use of box plots.

Among the studies examined in this section while studies such as Newhouse (1977), Hitiris and Posnett (1992), Hitiris (1997), Jewel et al., (2003), Okunade and Murthy (2002), Kiymaz et al., (2006), Chakroun (2009), Baltagi and Moscone (2010), Mehrara et al., (2010) and Santiago et al., (2013) have focused on examining the relationship between health care expenditure and income in the OECD countries, Nyamwange (2012), Yaqub et al., (2012), Elmi and Sadeghi (2012), Bakare and Olubokun (2011), Aurangzeb (2003), Mehrara and Musai (2011), Boachie et al., (2014) focused on individual countries while others focused on a group of countries such as emerging economy, Indian states, developing countries, and Africa (Baldacci (2004; Rajagopal, 2016; Elmi and Sadeghi, 2012, Bedir, 2016; and Lv and Zhu, 2014) respectively. Equally noted from discourse on the relationship between health expenditure and income is that in line with Newhouse (1977), some studies have found the income elasticity of health expenditure to be 1 and above. These includes studies such as Hitiris and Posnett (1992), Hitiris (1997), Kiymaz et al., (2006), Mehrara et al., (2010), Santiago et al., (2013) who saw health expenditure as a luxury good implying that health expenditure responds faster to a percentage change in income. However, other studies have classified health care as necessary good. For example, studies such as Okunade and Murthy (2002), Chakroun (2009), Baltagi and Moscone (2010), Nyamwange (2012), Lv and Zhu (2014) found health expenditure to be necessary rather than luxury. This reveals that there is no consensus if health care expenditure is a necessary or luxury good.

expenditure and infant mortality rate, Boachie et al., (2014) indicate a positive relationship between these variables.

Clearly observed from this review is that at the time of this study, study investigating the relationship between health expenditure and per capita GDP or income has not being done for the ECOWAS countries. It is also observed that the methodology used in these studies are different. Though studies such as Kiymaz et al., (2006), Elmi and Sadeghi (2012), Lv and Zhu, 2014 focused on developing countries and Africa using panel approach, while Elmi and Sadeghi (2012), employed the cointegration approach within the framework of Vector Error Correction Model, Lv and Zhu, 2014 employ the fixed effect model. Hence, this study seeks to fill this gap by examining relationship between Health care expenditure and income for the ECOWAS countries following the panel cointegration approach within the framework panel fully modified OLS and panel dynamic OLS methods as well as Toda and Yamamoto causality test so as to add to the body of knowledge.

3.4 CONCLUSION

This chapter is centred on the discussion of literature (theoretical and empirical) underpinning the study. Theories such as the Wagner theory of expenditure, Keynesian expenditure theory, the neoclassical growth model and the endogenous growth model are discussed in this chapter alongside the empirical literature. However, the Wagner theory alongside the empirical literature of Boachie et al., (2014) was adopted to build the functional model for this study. Hence the literature reviewed justifies the necessary variables included in the model of this study so as to achieve the goal as well as the gap identified.

From the studies and literatures reviewed, it is observed that studies related to health expenditure and growth in the ECOWAS sub-region is quite necessary and will enhance the policy formulation of this region.
CHAPTER 4

RESEARCH METHODOLOGY

4.1 INTRODUCTION
This chapter focuses on vivid and explicit explanations of the methodology of the study. It captures the model specification based on both the theoretical and empirical literature underpinning the study as examined in the preceding chapter. The chapter equally presents the data description/a priori expectation, and the modelling strategy employed to achieve the aim of the study. The panel cointegration approach is utilized as the modelling technique suitable for this study. Thus, the systemic or logical procedure embarked upon includes: descriptive statistics, visual investigation of the variables followed by the panel unit root test which will be performed with the Levin at el. and Im Persran unit root test. This will be immediately followed by panel test of cointegration to determine the long run relationship among the variables after which the Panel Fully Modified Ordinary Least Square (FMOLS) and Panel Dynamics Ordinary Least Square (DOLS) will be performed to generate the long run elasticity of the variables. After this, the Panel causality test is performed in order to determine the direction of causation.

4.2 MODEL SPECIFICATION
The Peacock and Wiseman (1967) version of the Wagner theory of increasing state expenditure forms the basis for the model specification of this current study. This version of the Wagner theory posits that increase in the per capita income, usually proxied as Gross Domestic Product (GDP) per capita, has the tendency in the long run to increase the share of public expenditure relative to income. This version of Wagner’s theory is usually empirically investigated by following the functional form stated as:

\[ GE = f (GDP) \]  

(4.1)

GE is total government expenditure and GDP is gross domestic Product. This version of the Wagner theory is normally linked to Peacock and Wiseman (1967) and Musgrave (1969).

To further expand and modify the above stated functional form of the Wagner theory, this study follows the empirical model of Nyamwange (2012) and Boachie et al., (2014) to build
the functional multivariate panel model of this study. Hence the functional model is expressed as below:

\[ \text{HEPC}_{it} = f(\text{GDPPC}_{it}, \text{LEXP}_{it}, \text{POPG}_{it}, \text{MORATE}_{it}) \]  
(4.2)

Where Health expenditure per capita (HEPC) is a function of Gross Domestic Product per capita (GDPPC), life expectancy (LEXP), population growth (POPG) and infant mortality rate (MORATE)

This model 4.2 is stated in logarithmic form as:

\[ \ln \text{HEPC}_{it} = \beta_0 + \beta_1 \ln \text{GDPPC}_{it} + \beta_2 \ln \text{LEXP}_{it} + \beta_3 \ln \text{POPG}_{it} + \beta_4 \ln \text{MORATE}_{it} + e \]  
(4.3)

Where \( i \) = individual units (countries) and \( t \) = time

\( \beta_0 \) = the intercept for equation

\( \beta_1 \) = the parameter estimates of GDPPC

\( \beta_2 \) = the parameter estimates of LEXP.

\( \beta_3 \) = the parameter estimates of POPG

\( \beta_4 \) = the parameter estimates of MORATE

4.3 DATA DESCRIPTION AND THE APRIORI EXPECTATION

The table below shows the variables included in the functional model of this study to be examined to achieve the main purpose of the study. It also indicates a brief description of the variable, the unit of measurement, data source as well as the apriori expectation or the expected relation between the dependent variable and the independent variables.
Table 4.1 Descriptions of data and expected relationship with the dependent variable

<table>
<thead>
<tr>
<th>VARIABLES</th>
<th>DESCRIPTION</th>
<th>UNIT OF MEASUREMENT</th>
<th>SOURCE</th>
<th>APRIORI EXPECTATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>LnHEPC</td>
<td>Per Capita Health Expenditure.</td>
<td>Annual Data measured in current U.S dollars</td>
<td>World Bank Development Indicators</td>
<td>+ (Positive)</td>
</tr>
<tr>
<td>LnGDPPC</td>
<td>Gross Domestic Product Per Capita</td>
<td>Annual data measured in current US dollars</td>
<td>World Bank Development Indicators</td>
<td>+ (Positive)</td>
</tr>
<tr>
<td>LnLEXP.</td>
<td>Life expectancy at birth,</td>
<td>Total (years)</td>
<td>World Bank Development Indicators</td>
<td>+ (Positive)</td>
</tr>
<tr>
<td>LnPOPG</td>
<td>Population growth</td>
<td>Annual percent</td>
<td>World Bank Development Indicators</td>
<td>+ (Positive)</td>
</tr>
<tr>
<td>LnMORATE</td>
<td>Mortality rate, infant</td>
<td>Per 1,000 live births</td>
<td>World Bank Development Indicators</td>
<td>- (Negative)</td>
</tr>
</tbody>
</table>

Source: Authors design based on literature

As stated in the table 4.1 above, equation (4.3) assumes a positive relationship exists between the dependent variable (HEPC) and GDPPC, LEXP AND POPG, while a negative relationship is expected to exist between HEPC and MORATE (infant mortality rate) in the ECOWAS countries as identified from the literature reviewed.

- **Per capita health expenditure**: This is the total health expenditure which is the sum of public and private health expenditures as a ratio of total population. This is spending on family planning, providing health services (preventive and corrective), nutrition activities, and emergency aid designated for health, but does not include provision of water and sanitation. This is expressed as a ratio of GDP.

- **GDP per capita**: this is gross domestic product divided by midyear population. GDP is the sum of gross value added by all resident producers in the economy plus any product taxes and minus any subsidies not included in the value of the products. It is calculated without making deductions for depreciation of fabricated assets or for
depletion and degradation of natural resources. This variable has been found to account for the divergence in the growth and level of healthcare expenditure in literatures. Badi et al. (2010) found that income and health expenditure are positively related in the long run.

- **Life expectancy** is referred to as the expected average number of years a group of people or a new born child will live given the prevailing death rate since a decline in the rate of mortality would mean that life expectancy is low compared to the actual life span. It is used to compare the mean number of years a specific group or cohort born in a particular year would live if the rate of death remains the same in the future (Chiang, 1984). This variable is one of the most-used health indicators since it captures the quality of life of a particular economy as well as presenting the death rate at all age cohorts. Improved access to good health services, among others, is the driving force for gains in life expectancy. According to Show et al, 2002; Cutler et al., 2006; Poças and Soukiazis, 2010), life expectancy and mortality indicator are good proxies that can be used to measure health outcome or status of the people of a particular economy. Jen et al., (2010) observed that the higher the life expectancy, the more healthy the people.

- **Population growth**: Annual population growth rate for year t is the exponential rate of growth of midyear population from year t-1 to t, expressed as a percentage. Population is based on the de facto definition of population, which counts all residents regardless of legal status or citizenship (World Bank, 2013). The growth of the population will cause expenditure on health to increase because population is sensitive to the rate of senility which occurs as a result of adverse and severe illnesses and diseases. A rapid rise in the growth of the population will exert pressure on the healthcare resource. This is even more severe in the lower income countries such the ECOWAS countries. Thus, a positive relationship is expected to exist between health and the population growth.

- **Infant mortality rate**: MORATE as used in this study refers to the number of infants that die before reaching one year of age, per 1,000 live births in a given year. Reducing the rate of infant mortality is one of the targets of the Millennium Development Goals (MDGs) (UN, 2000). West Africa has a higher under-five of mortality rate of about 132, compared to other African regions with only 84, in 2011. Increased Health expenditure has be found to reduce the infant mortality rate. Anand
and Ravallion (1993); Filmer and Pritchett, (1999); and Gupta, et al. (1999) showed that when health expenditure increases, the infant mortality rate will fall. In the same vein, Novignon, et al. (2012) found that a rise in health expenditure will cause infant mortality for 44 sub-Saharan African nations to fall by about 3 per 1000 live births for the period 1995-2010.

4.4 MODELLING TECHNIQUES

This study is based on the panel data analysis to examine the relationship between the income and health expenditure in the ECOWAS countries for the period 1995-2014. Panel data is referred to as the combination of time series and cross-section data. As noted by Baltagi (2008), panel data involves pooling cross section observations over some time periods. Hence, the study uses the pool model approach to obtain estimates of all the parameters.

This modelling technique is the most appropriate for this because as Gujarati (2003) noted, combining cross-section with time series will improve both the quantity and quality of data in a manner that will not be achieved if only one of these two dimensions is used. Also, the choice of this approach is made on the basis that it provides the opportunity of avoiding the problem of degrees of freedom since pooling allows for the number of observation to increase, as observed by Gogas et al., (2014).

Baltagi (2008) states that panel data is capable of accounting explicitly for heterogeneity in the individuals or subjects by making room for individual-specific variable. Thus, panel data, unlike cross-section and time series studies, is capable of avoiding bias estimates since it controls for heterogeneity of the individual units in the panel. He also noted that with panel data, effects that are not easily detected with time series or cross-sectional data can be identified and measured.

Following the panel cointegration test, we are able to overcome the problem of spurious regression. If two time series are stationary it means that they have constant variance and means. If they are not stationary at level, then there is a need to run the cointegration test so as to examine if the variables have constant co-variance. By doing this it is possible to overcome estimating nonsensical regression.

The Eviews statistical software package will be used in running the analysis so as to obtain the estimates and to ascertain whether or not the Hypothesis holds. Results obtained will
enable us to say specifically which of the variables included in the model is related to the dependent variable.

The logical steps involved in this technique include: Panel descriptive statistics, Panel stationary test, panel cointegration test, panel fully modified ordinary least square (FMOL), dynamic ordinary least square (DOLS) and lastly; panel Granger causality test.

4.4.1 Descriptive statistics

At this stage the time series property of the variables will be examined before the econometric investigation of the relationship between the dependent variable (HEPC) and the exogenous variables (determinants) is conducted. Here, the descriptive statistics of the variables are presented.

This is done so that the summary of the data in quantitative form can be provided. The descriptive statistics provide information on the mean, standard deviation, median, skewness and kurtosis of the data. When conducting econometric investigation, descriptive statistics is seen as a good start as observed by Mahadeva & Robinson (2004).

4.4.2 Stationarity Investigation

Performing a stationarity test is very important since most macroeconomic variables are non-stationary. It is important because it will avoid running spurious regression and help to make appropriate forecasts based on the results obtained. To run a regression analysis on non-stationary series will results in spurious or nonsensical regression producing invalid coefficients and goodness of fit. According to Gujarati and Porter (2009), a spurious regression will result in high $R^2$, highly significant F-statistic and t-statistics. Thus, to avoid this problem, the series ought to be stationary. The test for stationarity will start with the visual inspection and there-after the panel unit root will be carried out.

4.4.2.1 Visual Inspection for Stationarity

The visual inspection for stationarity involves generating the graphical representation of the individual series which displays the trend of the series. This test will serve as the preliminary test of stationarity. The main idea behind this test is that it can enable one to access quickly the trend of the series (Rachev et al., 2010). That is, it is easy to view how the series moves around the mean and the covariance. There is however, need for a proper unit root test because the visual test of stationarity cannot display the level at which the series is stationary, either at first difference or at second difference.
4.4.2.2 Panel Unit Root Test

Levin and Lin (1992) built the foundation for the panel unit root testing after which other suggestions on panel unit root tests were developed by Im et al. (2003), Maddala and Wu (1999) and Islam (1995). The usage of cross-sectional data was expanded in the analysis of panel data by Islam (1995) but Quah (1992, 1994), Evans and Karras (1996), Bernard and Jones (1996) and Im et al. (2003) developed the procedure for testing panel unit root. These tests are virtually the same except for the restrictions and assumptions made. While Evans and Karras (1996) account for different autoregressive coefficients across the units (countries), Bernard and Jones (1996) only account for individual effects but Quah (1992) does not consider fixed effects.

This study however employs two of the first generation panel unit root tests: the Levin et al., (2002) and IPS (Im et al., 2003) tests to determine if the variables are stationary or not. Although these tests both allow for heterogeneity of the intercept among members of the panel, LLC assumes a homogenous slope coefficient for all the units in the panel while IPS, also known as the heterogeneity panel unit root test, allows for heterogeneity in the slope coefficients.

4.4.2.2.1 The Levin- Lin-Chu (LLC) Unit Root Test

The test is one of the first generation panel unit root tests which assumes a common slope coefficient for the units in the panel. According to Levin et al. (2002), the test for individual unit root is low in power as against the alternative hypotheses hence they proposed a more powerful panel unit root test which suggests that $\rho_i$ are the same and negative. The test begins by estimating a different Augumented Dickey- Fuller (ADF) for the individual cross-section. The ADF equation below is performed for each cross section.

$$\Delta y_{it} = \rho_i y_{i,t-1} + \sum_{i=1}^{\rho_i} \varphi_{i,t} \Delta y_{i,t-L} + \alpha_i d_{it} + \epsilon_{it} \quad (4.4)$$

With $d_{it}$ showing the vector of deterministic variable and $\alpha_i$ is the corresponding vector of the coefficients for the model. $\rho_i = 0$ implies that the $y$ process has a unit root for individual i, while $\rho_i < 0$ indicates that the process is stationary around the deterministic part. Hence, the null and alternative Hypotheses are stated as below:

Null Hypothesis: $H0$: $\rho_i = 0$, individual series has unit root

Alternative Hypothesis: $H1$: $\rho_i < 0$, individual series has no unit root (series is stationary)
In this test for stationarity, the \( \rho_i \) is allowed to vary across the cross-section. The procedure is followed immediately with estimation of two other auxiliary regressions stated as below:

\[
\Delta y_{it} \text{ on } \Delta y_{i,t-L} \text{ and dit to obtain the residuals } \hat{e}_{it} \text{ and } y_{i,t-1} \text{ on } \Delta y_{i,t-L} \text{ and dit to obtain residuals } \tilde{\nu}_{i,t-1}.
\]

The next step in performing this test involves standardizing the residual so as to account for different variance across the cross-section or individual by performing

\[
\hat{e}_{it} = \hat{e}_{it} / \hat{\sigma} \hat{e}_i \tag{4.5}
\]

\[
\tilde{\nu}_{i,t-1} = \hat{\nu}_{i,t-1} / \sigma \hat{\nu}_i \tag{4.6},
\]

The ADF standard error is denoted by \( \sigma \hat{\nu}_i \)

The final operation in this test is to compute the pooled OLS regression. We compute the panel t-statistics.

\[
\hat{\epsilon}_{it} = \rho \hat{\nu}_{i,t-1} + \hat{\epsilon}_{it} \tag{4.7}
\]

The necessary asymptotic and sufficient requirement for this test is that:

\[
\sqrt{NT / T} \rightarrow 0, \ NT / T \rightarrow 0 \text{ and } NT / T \rightarrow \kappa \text{ respectively. NT stressed that } N \text{ (the cross-sectional dimension) is monotonically an increasing function of } T \text{ (the time dimension). The authors suggest that this test is appropriate for a panel of moderate size where } N \text{ is between 10 and 250 and } T \text{ is between 25 and 250. To them the test is adequate for the large } T \text{ and that standard procedure for a panel may not be powerful enough for this size of panel. This test however, is not without its shortcomings. Its shortcomings are the assumption that cross-sections are independent of each other and also the restrictive assumptions that all cross-sections are non-stationary (have unit root) or stationary (have no unit root).}

**Guiding Rule:**

To make a decision on whether or not the series is stationary, the computed p-value is compared with the absolute p-value at 10%, 5% and 1% significance level. Hence, when the computed p-value is more than any of the absolute p-values, we fail to reject the null hypothesis of non-stationary and reject the alternative hypothesis. However, for this study decision will be made at the 5% significant level and if the p-value of the statistics is more than the 5% level, we fail to reject the null hypothesis but if the p-value is less than 5% significant level we reject the null hypothesis and accept the alternative hypothesis and conclude that the series is stationary.
4.4.2.2 The Im, Pesaran, and Shin unit root test (IPS test)

The study also employs the IPS (Im et al., 2003) panel unit root test to investigate if the variables in the model are stationary or not. This test (IPS) is also known as the heterogeneity panel unit root test. It is less restrictive unlike the LLC test stated above. Hence, it allows for heterogeneity of the intercept among members of the panel as well as the slope coefficients. Hoang and McNown (2006) noted that this test combines the individual unit root tests to form the panel specific results. The test is more or less the performance of the augmented Dickey–Fuller (1979) test for individual cross-section data but individual cross-sections are assumed to be independent, thus, the null on the panel is examined using the combination of the large sample distribution of the t-statistics. The IPS is also chosen for this study since studies done by O’Connell (1998), Maddala and Wu (1999), Im et al. (2003) and Jonsson (2005) found that the LLC unit root test has low power compared to the IPS unit root test. The IPS test proposed the average of the individual ADF statistics when $\varepsilon_{it}$ is serially correlated across cross-sectional unit. The starting point of the IPS is the same as that of LLC stated in eq (4) above and duplicated as equation (4.8) below

$$\Delta y_{it} = \rho_i y_{it-1} + \sum_{l=1}^{\rho_i} \varphi_{il} \Delta y_{it-l} + \alpha_i d_{it} + \varepsilon_{it} \quad (4.8)$$

Null Hypothesis: H0: $\rho_i = 0$: individual series has unit root for all cross-sections

However, the alternative hypothesis is such that it permits some cross-sections to contain unit root.

$$H_1: \begin{cases} 
\rho_i < 0 \text{ for } i = 1,2,\ldots,N_1 \\
\rho_i = 0 \text{ for } i = N_1 + 1,\ldots,N
\end{cases} \quad (4.9)$$

The fractional cross-section series that do not contain unit root (stationary) are to be more than zero but less than or equal to one. That is, $\lim_{N \to \infty} (N_1 / N) = \delta$, but $0 < \delta \leq 1$.

The above condition is necessary as it allows for panel unit root to be consistent. Thus, the IPS t-statistics is defined as the mean of the individual ADF statistics stated below:

$$\bar{t} = \frac{1}{N} \sum_{i=1}^{N} t_{\rho_i} \quad .$$

From this equation, $t_{\rho_i}$ the ADF is the t-statistics for $H_0: \rho_i = 0$ for all individual cross-section series (i) in equation

$$\bar{t} = \frac{1}{N} \sum_{i=0}^{N} t_{\rho_i} \quad .$$

When the $\rho_i$ is not zero for the cross-section IPS indicates that $\bar{t}$ will have an asymptotic N (0,1) distribution in the general case.
To make a decision on whether or not the series is stationary, the computed p-value is compared with the absolute p-value at 10%, 5% and 1% significance level. Hence, when the computed p-value is more than any of the absolute p-values, we fail to reject the null hypothesis of non-stationary and reject the alternative hypothesis. However, for this study decision will be made at the 5% significant level and if the p-value of the statistics is more than the 5% level, we fail to reject the null hypothesis but if the p-value is less than 5% significant level we reject the null hypothesis and accept the alternative hypothesis and conclude that the series is stationary.

4.4.3 COINTEGRATION TEST

Another step undertaken in this study to further examine series that are integrated in panel data, apart from the unit root test explained above is the cointegration test. According to Engle and Granger (1987) cointegration among variables can occur if the individual series are integrated of the same order. By implication, if there exists a linear combination between two or more I (1) variables such that this combination is integrated of zero [I (0)], then these series are said to be cointegrated. This implies that there is a long run relationship among the variables. Cointegrations techniques are employed to confirm the long run equilibrium relationship among series that are integrated of order one.

However, there are two basic approaches in panel cointegration analysis. These are the maximum-likelihood approach and the residual-based approach. This study will follow the residual-based approach. Thus, the Pedroni test of cointegration and the Kao test of cointegration are utilized. These residual based tests of cointegration assume only one cointegrating vector just as in Engle and Granger (1987). The motivation for these tests is because they overcome the problem of low power associated with a small sample in standard cointegration test if there is large cross-section dimension (Örsal, 2009)

4.4.3.1 Pedroni Test of Cointegration

This study employs the Pedroni panel cointegration test (1999, 2004) to affirm the cointegration of the variables. This version of the Pedroni test of cointegration is an extension of the Pedroni (1995) residual-based panel cointegration tests to accommodate a procedure of multiple regressors. It suggests seven residual based tests of cointegration to test the null hypothesis that there is no cointegration. Out of these tests, four are within dimension based and they are calculated by adding the numerator and the denominator across the number of
cross-section individually while the remaining between-dimension-based statistics are calculated by dividing the numerator and denominator before adding up over the number of cross-section. The four within-dimension-based statistics are: panel-$v$, panel-$p$, semi-parametric panel-$t$ and parametric panel-$t$ and the three between-dimension-based statistics are group-$p$, semi-parametric group-$t$ and parametric group-$t$. This Pedroni (1999, 2004) panel cointegration test starts by computing the residuals of the cointegrating regression hypothesized,

$$y_{i,t} = \delta_0 i + \delta_1 i, t + x_i,t \beta_i + \epsilon_i,t,$$  

(4.10)

$i = 1..., N; t = 1..., T$, where $N$ is the number of individuals in the panel and $T$ is the number of observations over time. $\delta_0 i$ and $\delta_1 i, t$ represents the individual specific intercept and the trend parameter which can vary over cross-section. The yit and the $K$-dimensional vector of exogenous variables $x_i,t = x_{i,t-1} + v_i,t$ are both assumed to be integrated of order one [I(1)]. The error process $w_t = (e_t, v't)$ is also assumed to be independently distributed. The cointegrating vector $\beta_i = (\beta_1 i... \beta_K i)'$ can also vary across sections.

As stated earlier, the Pedroni test suggests seven cointegration statistics which are categorised into two. The first consists of four within-dimension based statistics and out of these statistics three are non-parametric corrections while the fourth is parametric based on the ADF-test. The second category contains three tests of which one is parametric based on ADF-test and two are non-parametric corrections.

The null hypothesis of this test is that there is no cointegration among the variables. To test this null hypothesis in a model which allows for some measure of heterogeneity, some tests were suggested by Pedroni (2000, 2004).

These tests are specified as below:

1. Panel $v$-Statistic:

$$T^2 N^\frac{3}{2} Z_{\hat{v},N,T} \equiv T^2 N^\frac{3}{2} (\sum_{t=1}^{T} \sum_{i=1}^{N} \hat{L}^{-1}_{11i} \hat{\epsilon}_{i,t-1}^2)^{-1}$$  

(4.11)

2. Panel$\rho$-Statistic:

$$T\sqrt{N} Z_{\hat{\rho},N,T} \equiv T\sqrt{N} (\sum_{t=1}^{T} \sum_{i=1}^{N} \hat{L}^{-1}_{11i} \hat{\epsilon}_{i,t-1}^2)^{-1} 1 \sum_{t=1}^{T} \sum_{i=1}^{N} \hat{L}^{-1}_{11i} (\hat{\epsilon}_{i,t-1} \Delta \hat{\epsilon}_{i,t} - \hat{\lambda}_i)$$  

(4.12)

3. Panel $t$- Statistic (non-parametric):

$$Z_{t,N,T} \equiv (\hat{\sigma}_{t,N}^2 \sum_{i=1}^{N} \sum_{t=1}^{T} \hat{L}^{-1}_{11i} \hat{\epsilon}_{i,t-1}^2)^{-\frac{1}{2}} \sum_{t=1}^{T} \sum_{i=1}^{N} \hat{L}^{-1}_{11i} (\hat{\epsilon}_{i,t-1} \Delta \hat{\epsilon}_{i,t} - \hat{\lambda}_i)$$  

(4.13)

4. Panel $t$- Statistic (parametric):
5. Group $\rho$-Statistic:

$$T N^{-\frac{1}{2}} \tilde{Z}_{\rho_N,T-1} \equiv TN^{-\frac{1}{2}} \sum_{l=1}^{N} (\sum_{t=1}^{T} \hat{e}_{l,t-1}^{2})^{-1} \sum_{t=1}^{T} \left( \hat{e}_{l,t-1} \Delta \hat{e}_{l,t} - \lambda_{l} \right)$$

(4.15)

6. Group $t$-Statistic (non-parametric):

$$N^{-\frac{1}{2}} \tilde{Z}_{t_N,T} \equiv N^{-\frac{1}{2}} \sum_{l=1}^{N} \left( \sum_{t=1}^{T} \hat{e}_{l,t-1}^{2} \right)^{-\frac{1}{2}} \sum_{t=1}^{T} \left( \hat{e}_{l,t-1} \Delta \hat{e}_{l,t} - \lambda_{l} \right)$$

(4.16)

7. Group $t$-Statistic (parametric):

$$N^{-\frac{1}{2}} \tilde{Z}_{s_N,T} \equiv N^{-\frac{1}{2}} \sum_{l=1}^{N} \left( \sum_{t=1}^{T} \hat{e}_{l,t-1}^{2} \right)^{-\frac{1}{2}} \sum_{t=1}^{T} \hat{e}_{l,t-1}^{*} \Delta \hat{e}_{l,t}^{*}$$

(4.17)

Wherein,

$$\hat{\lambda}_{i} = \frac{1}{T} \sum_{s=1}^{g_{i}} \left( 1 - \frac{S}{K_{i} + 1} \right) \sum_{t=s+1}^{T} \hat{\mu}_{i,t-s}, \quad \hat{\lambda}_{N,T} = \frac{1}{T} \sum_{i=1}^{N} \hat{\mu}_{i,T}, \quad \tilde{\sigma}_{i}^{2} = \tilde{s}_{i}^{2} + 2 \hat{\lambda}_{i}, \quad \tilde{\sigma}_{N,T}^{2}$$

But the residuals $\hat{\mu}_{i,t}, \hat{\mu}_{i,t}^{*}$ and $\hat{\eta}_{i,t}$ are obtained from the regressions stated as follow:

$$\hat{e}_{i,t} = \hat{y}_{i} \hat{e}_{i,t-1} + \hat{\mu}_{i,t}, \quad \hat{e}_{i,t}^{*} = \hat{y}_{i} \hat{e}_{i,t-1}^{*} + \sum_{g=1}^{g_{i}} \hat{y}_{i,g} \Delta \hat{e}_{i,t-g}^{*} + \hat{\alpha}_{i,t}^{*} \Delta y_{i,t} = \sum_{m=1}^{M} \hat{b}_{m} \Delta x_{m,i,t} + \hat{\eta}_{i,t}$$

Decision will be made on 5% significance level. Hence if the p-value of the statistics is more than 5% level, we fail to reject the null hypothesis but if the p-value is less than the significant level, we reject the null and accept the alternative hypothesis. Also, if the majority of these test confirms that there is cointegration then we can accept the alternative and conclude there is cointegration against the null of no cointegration.

This test of co-integration is relevant for this study since the study is concerned with multiple regressors. Pedroni test of cointegration lends explanation to the null hypothesis of no
cointegration in heterogenous and homogenous panel of multivariate relationship. This will help to avoid the problem of spurious regression.

### 4.4.3.2 Kao Test of Cointegration

The second cointegration test employed in this study is the Kao test of cointegration. This test extended the Engle and Granger (1987) residual-based test of cointegration in time series to the analysis of panel data. Both the Pedroni (2000, 2004) and Kao (1999) cointegration tests are residual-based. As a residual-based cointegration test, conclusion on whether or not the series are cointegrated is made if the residual of two series that are integrated of order one, that is two I(1) series is found to be integrated of order zero [I(0)]. The main reason for this test is to further confirm the cointegration result of Pedroni. Although this test follows a similar approach as the Pedroni test, it differs with the specification of homogenous coefficients on the first stage regressors and the individual specific constant.

The Kao test is based on spurious LSDV least squares dummy variable panel regression equation with one single regressor. Hence, it examines the null hypothesis of no cointegration by estimating the equation below:

\[
y_{it} = \alpha_i + x_{it}\beta + e_{it}, \quad i = 1, \ldots, N; \quad t = 1, \ldots, T.
\]  

(4.18)

from this equation \(\beta\), which is slope coefficient, is assumed to be homogenous meaning that it is invariant across the cross-section but the intercept is heterogenous. \(e_{it}\) is integrated of order one [I(1)] as well as the \(y_{it}\) and \(x_{it}\).

Kao test applied both Dicky-Fuller (DF) and Augumented Dicky-Fuller tests. The DF test is examined with the equations stated as below:

\[
DF_p = \frac{\sqrt{NT(\rho-1)+3\sqrt{N}}}{\sqrt{10.2}}
\]  

(4.19)

\[
DF_t = \sqrt{1.25 \cdot t_p + 1.875N}
\]  

(4.20)

\[
DF_p^* = \frac{\sqrt{NT(\rho-1)+3\frac{N\sigma_p^2}{\sigma_{ep}^2}}}{\sqrt{3+\frac{36\sigma_p^4}{5\sigma_{ep}^4}}}
\]  

(4.21)
From the above equations, \( \hat{\sigma}_v^2 = \hat{\Sigma}_{yy} - \hat{\Sigma}_{yx}\hat{\Sigma}_{xx}^{-1}, \) \( \hat{\sigma}_{ov}^2 = \hat{\Omega}_{yy} - \hat{\Omega}_{yx}\hat{\Omega}_{xx}^{-1} \) while \( \hat{\Sigma} \) is estimator of long run covariance \( \xi_{it} = (\Delta y_{it}, \Delta x_{it})', \) and \( \hat{\Omega} \) is the estimator of contemporaneous covariance of \( \xi_{it} = (\Delta y_{it}, \Delta x_{it})'. \) \( DF_\rho \) and \( DF_t \) are centred on the strong exogeneity of the independent variables and error term, while \( DF_\rho^* \) and \( DF_t^* \) are based on the cointegration with an endogenous relationship amongst independent variables and error term.

The ADF test can be run from the following regression:

\[
\hat{e}_{it} = p\hat{e}_{it-1} + \sum_{j=1}^{p} \delta_j \Delta \hat{e}_{i,t-j} + v_{it} \tag{4.23}
\]

The null hypothesis is that there is no cointegration, thus the ADF test statistics can be estimated as follows: \( ADF = \frac{t_{ADF} + \sqrt{N} \hat{\sigma}_v}{2\hat{\sigma}_{ov}} \sqrt{\frac{\hat{\sigma}_v^2 + 3\hat{\sigma}_v^2}{2\hat{\sigma}_{ov}^2 + 10\hat{\sigma}_{ov}}}, \) \( (4.24) \)

\( t_{ADF} \) is the t-statistic of \( \rho \) in the preceding equation. The asymptotic distributions of \( DF_\rho, \) \( DF_t, DF_\rho^*, DF_t^* \) and ADF converges to a standard normal distribution \( N(0,1) \) by sequential limit theory.

Upon the determination of the cointegration among the variables, the next step is to investigate the long run effect of the variables, to generate and examine the relationship between the independent variables (LnGDPPCit, LnLEXPit, LnPOPGTit, and INFIt) on the dependent variable (LnHEPC) on the long run.

4.5. RESIDUAL-BASED PANEL FULLY MODIFIED OLS (FMOLS) AND DYNAMIC OLS (DOLS)

The appropriate step taken after determining that there is cointegration among the variables is the estimation of the long run parameters. To effectively perform this task, the panel Dynamic OLS (DOLS) and panel fully modified OLS (PFMOLS) will be used to generate the
long run coefficients of the variables. This approach is appropriate following the argument put forward by Kao and Chiang (2000) that these estimators account for endogeneity, heteroscedasticity and serial correlation properties of the residuals in the standard pooled OLS which is usually found in the long run equilibrium. These residual-based estimators as specified by Pedroni (2002) and Kao and Chiang (2000) produce normally distributed coefficients and asymptotically unbiased estimates.

4.5.1 The Panel Dynamic OLS

The panel dynamics OLS is completely parametric in nature unlike the FMOLS. This estimation technique also account for endogeneity by adding leads and lags (Arize et al., 2000). Although leads and lags account for endogeneity, they reduce the degree of freedom and this is one of the pitfalls of the DOLS estimators. DOLS estimator is estimated from the equation below:

\[ y_{it} = \alpha_i + \beta_i x_{it} + \sum_{k=q} c_{ik} \Delta X_{it+k} + \mu_{it}; \quad t=1,...T \quad i=1...N \]  

(4.24)

From the above equation, \( \alpha_{it} \) represent the country specific effects and \( c_{ik} \) stands for the coefficients of leads and lags of the first difference of independent variables; \( \mu_{it} \) is the disturbance terms and this is expected to be integrated of order zero. That is, it should be I(0).

The DOLS parameter estimate according to Pedroni (2004) is given as below:

\[ \beta_{i,DOLS}^* = \left[ N^{-1} \sum_{i=1}^{N} \left( \sum_{t=1}^{T} Z_{it} Z_{it}^* \right) \left( \sum_{t=1}^{T} Z_{it} Y_{it}^* \right) \right] \]  

(4.25)

From the above equation \( Z_{it} = (x_{it} - \bar{x}_{it}, \Delta x_{it-k}, ..., \Delta x_{it+k}) \) is the 2(k+1)*1 vector of the independent variables.

4.5.2 The Panel Fully Modified OLS (FMOLS)

The work on panel FMOLS estimator can be traced back to the works of Phillips and Hasen (1999) and that of Pedroni (2000). This panel FMOLS is similar to the time series FMOLS of Phillips and Hasen (1990). This FMOLS is non-parametric in nature and corrects endogeneity and bias problems in the OLS. As a non-parametric structure, this estimator accounts for any likely correlation between the first differences of the regressors and the disturbance term. It also takes cognizance of the existence of a constant term in order to correct for the serial correlation problem. This panel estimator can be explained with the equation below:
\[ \beta_{i,FM}^{*} = \left[ \sum_{i=1}^{N} \left( x_{it} - x_{i} \right) \right]^{-1} \left[ \sum_{i=1}^{T} x_{it} (x_{it} - x_{i}) y_{it}^{*} - T \hat{\Delta}^{*}_{ieu} \right] \]  \hspace{1cm} (4.26)

Where \( \hat{\Delta}^{*}_{ieu} = \Omega_{ie}^{-1/2} \hat{\Delta}_{ie} u + \hat{\Omega}_{ieu} \).\( \epsilon^{-1/2} \)

The variable \( y_{it} \) is transformed as \( y_{it}^{*} = (x_{it} - \bar{y}_{t}) - \frac{L_{21}}{L_{21}} \Delta x_{it} \) to account for the endogeneity problem in the series.

Since there is also the tendency of serial correlation among the series, the FMOLS is built so as to correct for serial correlation as stated below:

\[ \hat{\Delta}_{ieu}^{*} = (\hat{\Delta}_{ieu}, \hat{\Delta}_{ie})(\Omega_{ie}^{-1}) = \hat{\Delta}_{ie} - \hat{\Delta}_{ie} \hat{\Omega}_{ie}^{-1} \Omega_{ieu} \].  \hspace{1cm} (4.27)

Hence, the long run covariance matrix is explained below as proposed by Kao and Chiang (2002):

\[ \Sigma_{j=0}^{\infty}E \left( w_{ij} w_{ij}' \right) = \Sigma + \Gamma + \Gamma \sqrt{\Omega_{u}} \]

\[ = \left[ \begin{array}{cc} \Omega_{u} & \Omega_{u,e} \\ \Omega_{e,u} & \Omega_{e} \end{array} \right] \]

This FMOLS approach is equally appropriate for this model because it allows for variables of different integrations orders (Adu, 2012). Thus, it allows both I(0) and I(1) to be examined in one single model and since this situation may be applicable to this study as we proceed, decision on the long run relationship between the dependent and the independent will be made based on the outcome of the this technique.

### 4.6 PANEL CAUSALITY TEST.

A way of applying ad hoc distributed lag models is to show the direction of causality among economic relationships or among variables which are causal in nature. This test is very important in determining the direction of causality. That is, which variable causes the other to move or change if we have prior knowledge that these variables are related.

Two approaches for addressing the issue of indeterminate causality are: (i) theorizing that two variables are determined simultaneously and (ii) performing the Granger causality test. Granger causality test is not a test of theoretical causality. According to Granger (1969),
granger causality, also known as precedence, is the case or circumstance whereby a variable predictably and persistently changes prior to change in the other variable. Hence, if lagged values of a variable, say $y_{it}$, can be used to foretell the changes in another variable, say $x_{it}$, then it can be said that $y_{it}$ granger causes $x_{it}$. The power of one variable to predict another variable is what is referred to as causality, as stated by Asteriou and Hall (2011).

To examine the causal relationship between variables, the Toda and Yamamot (1995) causality approach will be followed. The procedure requires that an augmented vector autoregressive (VAR ($p + \text{dmax}$) is first estimated to generate a Wald statistic that is an asymptotic distribution ($\chi^2$) because this procedure is robust to cointegration and integration properties of the process (Zapata & Rambaldi, 1997). The Toda and Yamamoto (1995) approach involves two basic steps. In the first step, appropriate lag length is determined with the help of any of the lag length criteria (Akaike Information Criterion (AIC), Hannan-Quinn (HQ), Final Prediction Error (FPE) and Schwarz Information Criterion (SC)) and the second involves determining the maximum integration order.

The Toda and Yamamoto (1995) test of causality is appropriate for this study since it has the capacity to overcome the drawbacks of the granger causality as identified by Gujarati (1995) to include specification problems and the number of lags, and nonstationary problems (Huang, Kao et al., 2004). Also, according to Wolde-Rufael (2005), Toda and Yamamoto causality is approriate integrated of order one (I(1)) or I(0) or I(2) and whether or not the series are cointegrated to overcome the drawback of granger causality stated here. This test of causality employs MWALD (modified Wald) test to place restriction on the parameters of the VAR ($p$) where $p$ is lag length.

Thus to apply the Toda and Yamamoto (1995) causality test, this study adopts the panel VAR of Emirmahmutoglu and Kose (2011) specified in 4.11:

$$z_{i,t} = \theta_i + \forall_{i1}z_{i,t-1} + \ldots + \forall_{ipi}z_{i,t-pi} + \varepsilon_{i,t} \quad i=1,2,\ldots,N, \quad t=1,2,\ldots,T$$

From 4.11, the individual cross section is represented as $i$ while $t$ denotes the time period. $\theta_i$ shows the k dimensional fixed effect vector while $\forall_{i1}, \ldots, \forall_{ipi}$ are matrices of the parameters (k*k) varying across the individual cross sections. The column vector represented as $\theta_i$ which is assumed to be identically and independently distributed across the units (cross sections) with $E(u_i, t)= 0$ and $V(u_i, t)=\Sigma u_i$ is positive covariance matrices. The lag order $pi$ varies across the units and it is assumed to be known or estimated with the lag length selection criterions (Lutkepohl, 2005).
The Null and alternative hypothesis are specified as below:

H0: \( \forall_{12,ij} = 0 \) for all \( i=1, 2, 3, \ldots N, j = 1,2,3,\ldots p \), that is Economic growth does not granger-cause health expenditure, and the alternative hypothesis is

H1: Economic growth does granger-cause health expenditure.

The Toda and Yamamoto (1995) version of causality test for the health expenditure-growth model is performed by estimating the models 4.12 and 4.13:

\[
\begin{align*}
HEPC_{it} &= \theta_i^{HEPC} + \sum_{j=1}^{p+dmax} \varphi_{11,ij} HEPC_{it-j} + \sum_{j=1}^{p+dmax} \varphi_{12,ij} GDPPC_{it-j} + \epsilon_{i,t}^{HEPC} \quad (4.29) \\
GDPPC_{it} &= \theta_i^{GDPPC} + \sum_{j=1}^{p+dmax} \varphi_{21,ij} GDPPC_{it-j} + \sum_{j=1}^{p+dmax} \varphi_{22,ij} HEPC + \epsilon_{i,t}^{GDPPC} \quad (4.30)
\end{align*}
\]

From these equations, lnHEPC and lnGDPPC are measures for health expenditure and economic growth respectively for the ECOWAS countries. \( i = 1, 2, 3, \ldots N \) represents cross-section while \( \epsilon_{i,t} \) are error terms, the lag length is captured by \( p \), and \( dmax \) represents the maximum order of integration. 4.29 examines causality from GDPPC to HEPC while 4.30 shows causality from HEPC to GDPPC.

The likely results of this test are either unidirectional, no causality or bidirectional.

The decision to accept or reject the null hypothesis is based on the probability value of the Wald-test. Hence, the rule is that, if p-value of the F-statistics is less than 5%, we reject the null hypothesis and accept the alternative hypothesis which would mean that economic growth does granger-causes health expenditure. However, if the p-value is more than 5% we fail to reject the null hypothesis meaning that economic growth does not granger cause health expenditure.

The causality test will be performed since the cointegration test only shows that there is causality in the series but does not indicate the direction of causality.
4.8 DIAGNOSTIC CHECK

This study use the fully modified OLS and Dynamic OLS which accounts for endogeneity and bias problems in the OLS. The estimators accounts for any likely correlation between the first differences of the regressors and the disturbance term. It also takes cognizance of the existence of a constant term in order to correct for the serial correlation problem. (Arize et al., 2000). Therefore, only the normality test will be performed in this study.

Normality is another assumption of the classical regression model. By this assumption, the residual is normally distributed. This test tests the null hypothesis that the residual is normally distributed with the skewness being zero and the excess kurtosis being zero. The
decision rule here is that if the probability value of the Jarque-Bera is significant then we conclude that the model is normally distributed.

The normality is performed to show if residual are normally distributed. Decision will be made based on the probability of the Jarque-Bera statistics. Thus, if the P-value of the Jarque-Bera statistics is more than 5%, it the null will be accepted meaning that residual is normally distributed but if it is less than 5%, we reject the null and accept the alternative hypothesis and it will be concluded that the residual is not normally distributed. The null and alternative are stated as below:

The null hypothesis is $H_0$: residual are normally distributed
Alternative hypothesis is $H_1$: Residual are not normally distributed

### 4.9 CONCLUSION

Chapter four focused on explicit explanations of the methodology of the study. It captured the model specification based on both the theoretical and empirical literature underpinning the study as examined in the preceding chapter. The chapter also presented the a priori expectation, data description/source and the modelling strategy employed to achieve the aim of the study. The Panel cointegration approach is utilized as the modelling technique suitable for this study. Thus, the systemic or logical procedure embarked upon includes: descriptive statistics, visual investigation of the variables followed by the unit root test which will be performed with the Levin et al. and Im Persran unit root tests. This will be immediately followed by a test of cointegration to determine the long run relationship among the variables after which the Panel FMOLS and DOLS will be carried out to identify the long run effect of the independent variables on the dependent variable. The panel causality test follows after these.
CHAPTER 5

PRESENTATION OF RESULTS AND DISCUSSION OF EMPIRICAL FINDINGS

5.1 INTRODUCTION

This chapter focuses on the presentation and discussion of results obtained from the logical steps presented in the previous chapter. In chapter four, the logical estimation techniques employed for this study were presented. Here, the results of the various tests are obtained with the use of Eviews 9.5 statistical software package. They are presented and discussed in detail.

5.2 DESCRIPTIVE STATISTICS

In this section, the descriptive statistics of the variables used in the model of this study are presented before proceeding with the econometric analysis. The variables were observed from 1995 – 2014 across 15 ECOWAS countries. Table 5.1 presents the descriptive statistics whereby the values of mean, median, standard deviation, skewness, the kurtosis and the Jarque-Bera statistics are observed. For the purpose of analysis, the point of interest here is the value of skewness and kurtoses because they help in observing how normally distributed our data are.
From table 5.1, we observed that, LNHEPPC, LNGDPPC are positively skewed while LNLEXP, POPG and LNMORATE are negatively skewed. This implies that the series here are not symmetrical. It was also found that the series are not normally distributed given that the probability of the Jarque-Bera statistics for all the variables are less than 5% significance level. The kurtosis shows the flatness or peakedness of the distribution and from the table, the kurtosis is from 2.742864 to 11.89283. Field (2009) is of the opinion that the test for skewness and kurtosis should be done with large samples as they may be significant though skew and kurtosis are not far from normal. Having observed that the series are not normally distributed, it is necessary to perform other tests to examine the economic property of the variables. Hence, we proceed with the stationarity tests.
5.3 STATIONARITY INVESTIGATION

This section is focused on examining the data for stationarity. This is done because running a regression analysis on non-stationary series will result in spurious or nonsensical regression so that the coefficients and goodness of fit are invalid. According to Gujarati and Porter (2009), a spurious regression will result in high R^2 with F-statistic and t-statistics highly significant. Thus, to avoid this problem, the series ought to be stationary. The test for stationarity begins with the visual inspection and it is immediately followed with the panel unit root test as presented below:

5.3.1 VISUAL INSPECTION FOR STATIONARITY

In this section, the graphs of the variables at levels and at first difference are presented for easy examination of the series for stationarity. Therefore, figure 5.1 and 5.2 are the graphical representations of variables at level and first difference respectively.

Fig 5.1 Graphical Representation of Variables at Level

The figure below indicates that both HEPC and GDPPC are non-stationary at level since they all trend upward, while the trend of the remaining variables are clear from the graph. It does not clearly show if they are stationary at level. As shown in the graphs, the trend of the variable for each country is depicted by different colours.
Figure 5.2 Graphical Representations of Variables at First Difference

The figure shows that only HEPC and GDPPC became stationary after first difference, that is, they are I (1) variables. However, LEXP, MORATE and POPG do not show stationarity clearly at the first difference. Hence, our variables have different orders of integration. To further confirm the stationarity of the variables, the panel unit root test is performed and presented in the next section.
5.3.2 PANEL UNIT ROOT TEST

The panel unit root test presented in the table 5.3.2 shows the results obtained from the Levin, Lin & Chu (LLC) test and the IM, Pesaran and Shin (IPS) test of all the variables included in the model of this study.

**Table 5.2 Panel Unit Root Test Result**

<table>
<thead>
<tr>
<th>Methods</th>
<th>HEPC</th>
<th>GDPPC</th>
<th>LEXP</th>
<th>MORATE</th>
<th>POPGT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Level</td>
<td>Level</td>
<td>Level</td>
<td>Level</td>
<td>Level</td>
</tr>
<tr>
<td></td>
<td>Difference</td>
<td>Difference</td>
<td>Difference</td>
<td>Difference</td>
<td>Difference</td>
</tr>
<tr>
<td></td>
<td>Statistics</td>
<td>Statistics</td>
<td>Statistics</td>
<td>Statistics</td>
<td>Statistics</td>
</tr>
<tr>
<td></td>
<td>P-value</td>
<td>P-value</td>
<td>P-value</td>
<td>P-value</td>
<td>P-value</td>
</tr>
<tr>
<td>Levin, Lin &amp; Chu (LLC)</td>
<td>-1.55136</td>
<td>0.8963</td>
<td>-33.0173</td>
<td>-13.0748</td>
<td>-6.48555</td>
</tr>
<tr>
<td></td>
<td>0.0604</td>
<td>0.815</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>IM, Pesaran and Shin W-Stat (IPS)</td>
<td>-0.83511</td>
<td>3.85324</td>
<td>-68.0177</td>
<td>-6.48555</td>
<td>0.8000</td>
</tr>
<tr>
<td></td>
<td>0.2018</td>
<td>0.9999</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td></td>
<td>-2.99338</td>
<td>-4.00311</td>
<td>-26.7737</td>
<td>-0.00612</td>
<td>-0.00061</td>
</tr>
<tr>
<td></td>
<td>0.0014</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.4976</td>
<td>0.4976</td>
</tr>
</tbody>
</table>

**Conclusion**

- I (1) indicates the variable is integrated of order 1.
- I (0) indicates the variable is integrated of order 0.
From table 5.2, it is observed that at the 5% significance level, HEPC and GDPPC are stationary after first difference while LEXP, MORATE and POPG are stationary at level. Therefore, it is concluded that the series are a mixture of both I(1)s and I(0)s. Sims (1988), Pagan and Wickens (1989), are of the view that to avoid misspecification of a model, if the dependent variable is integrated of order one then there must be at least one variable integrated of order one among the independent variables. Based on these results, we can proceed with the test of cointegration by employing the Pedroni (2000, 2004) and Kao (1999) cointegration tests. With these tests, both I(1) and I(0) can be examined for long run equilibrium relationship.

### 5.4 Cointegration Test

Upon discovering the order of integration of the variables of the model, the next appropriate step is to examine the variables for long run equilibrium relationship. Hence, the study employed the Pedroni test of cointegration and Kao test of cointegration to accomplish the task of cointegration investigation. These tests both follow the Engle-Granger approach wherein the residual generated from regressing I(1) variables is tested for stationarity. Thus, the Pedroni and Kao test of cointegration results are presented below in section 5.3.1 and 5.3.2 respectively.

#### 5.4.1 Pedroni Test of Cointegration

The Pedroni test of cointegration presents eleven statistics which include Panel v-Statistic, Panel rho-Statistic, Panel PP-Statistic Panel ADF-Statistic and Four Weighted statistics which are in the first category of within –dimension while the remaining three statistics; Group rho-Statistic, Group PP-Statistic and Group ADF-Statistic are found in the second category, which is known as the between- dimension.

As observed in the Table 5.3 from the Within-Dimension, Panel PP-Statistic and Panel ADF-Statistic are statistically significant at the 5% significance level. Also, the weighted statistics of Panel PP-Statistic and Panel ADF-Statistic are significant at the 5% level while Panel v-
Statistic and Panel rho-Statistic are not significant at the 5% level. From the Between-Dimension, both Group PP-Statistic and Group ADF-Statistic are significant at the 5% level while Group rho-Statistic is not significant. Based on this interpretation, we found that there is cointegration among the variables since six out of the eleven statistics are statistically significant at 5% level. Hence, we conclude that there is a long run relationship among our variables.

**TABLE 5.3 Pedroni Cointegration Result**

<table>
<thead>
<tr>
<th></th>
<th>Weighted</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Statistic</td>
<td>Prob.</td>
<td>Statistic</td>
<td>Prob.</td>
</tr>
<tr>
<td>Panel v-Statistic</td>
<td>-0.34787</td>
<td>0.6360</td>
<td>-2.72743</td>
<td>0.9968</td>
</tr>
<tr>
<td>Panel rho-Statistic</td>
<td>0.942498</td>
<td>0.8270</td>
<td>0.294970</td>
<td>0.6160</td>
</tr>
<tr>
<td>Panel PP-Statistic</td>
<td>-4.79227</td>
<td>0.0000</td>
<td>-11.2476</td>
<td>0.0000</td>
</tr>
<tr>
<td>Panel ADF-Statistic</td>
<td>-4.89055</td>
<td>0.0000</td>
<td>-8.01715</td>
<td>0.0000</td>
</tr>
</tbody>
</table>

Alternative hypothesis: individual AR coefs. (between-dimension)

<table>
<thead>
<tr>
<th></th>
<th>Statistic</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group rho-Statistic</td>
<td>2.255625</td>
<td>0.9880</td>
</tr>
<tr>
<td>Group PP-Statistic</td>
<td>-17.9798</td>
<td>0.0000</td>
</tr>
<tr>
<td>Group ADF-Statistic</td>
<td>-6.92981</td>
<td>0.0000</td>
</tr>
</tbody>
</table>

**5.4.2 Kao Test of Cointegration**

**TABLE 5.4 Kao Cointegration Result**

<table>
<thead>
<tr>
<th>Method</th>
<th>t-Statistic</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADF</td>
<td>-7.49053</td>
<td>0.0000</td>
</tr>
</tbody>
</table>
The table 5.4 presents the outcome of the Kao cointegration test result. The table contains three columns comprising the statistic method, t-statistic and the probability in the first second and third columns respectively. The Kao ADF-statistic as presented in the table is -7.49053 and the prob-value is 0.0000 which indicates significance at the 5% level. This shows that our variables are cointegrated. That is, there is a long run relationship among the variables. Hence, by implication we can reject the null hypothesis of no cointegration and accept the alternative hypothesis concluding the existence of cointegration among variables of this study.

5.5 RESIDUAL-BASED PANEL FULLY MODIFIED OLS (FMOLS) AND DYNAMIC OLS (DOLS)

From the above cointegration results presented we observed that the variables of this study have a long run equilibrium relationship. Therefore, to generate and examine the impact of the independent variables (LnGDPPC, LnLEXP, LnPOPG and LnMORATE) on the dependent variable (LnHEPC) in the long run, the study employs Panel Fully Modified Ordinary Least Square (FUMOLS) and the Panel Dynamic Ordinary Least Square (DOLS) estimation techniques. It is worthy of note at this point that although Stock and Watson (1993) proved via Monte-Carlo simulation that the DOLS estimators were superior over the FMOLS estimators, consensus has not been reached by researchers regarding which of the two is superior as observed by Tintin (2009). This study will present the results of both estimation techniques for the purpose of emphasis. However, in a situation of divergent outcome since our variables are a mixture of different orders of integration, the FMOL result will be adopted because this technique is appropriate for handling variables with different orders of integration (Adu, 2012, Boachie et al., 2014).
Table 5.5A Panel Dynamic Ordinary Least Square Results (DOLS)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Std. Error</th>
<th>t-Statistic</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNGDPPC</td>
<td>0.535667</td>
<td>0.187115</td>
<td>2.862777</td>
<td>0.0059</td>
</tr>
<tr>
<td>LNLEXP</td>
<td>3.013964</td>
<td>2.621209</td>
<td>1.149837</td>
<td>0.2551</td>
</tr>
<tr>
<td>LNPOPG</td>
<td>-1.65312</td>
<td>0.601337</td>
<td>-2.74907</td>
<td>0.008</td>
</tr>
<tr>
<td>LNMORATE</td>
<td>-0.25349</td>
<td>0.859887</td>
<td>-0.2948</td>
<td>0.7692</td>
</tr>
</tbody>
</table>

| R-squared   | 0.996915    |            |             |        |
| Adjusted R-squared | 0.986009  |            |             |        |

TABLE 5.5B Panel Fully Modified Ordinary Least Square (FMOLS)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Std. Error</th>
<th>t-Statistic</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNGDPPCUS</td>
<td>0.595291</td>
<td>0.06192</td>
<td>9.613938</td>
<td>0.0000</td>
</tr>
<tr>
<td>LNLEXP</td>
<td>0.908033</td>
<td>0.706212</td>
<td>1.285779</td>
<td>0.1996</td>
</tr>
<tr>
<td>LNPOPGT</td>
<td>0.05042</td>
<td>0.118793</td>
<td>0.424434</td>
<td>0.6716</td>
</tr>
<tr>
<td>LNMORATE</td>
<td>-0.672543</td>
<td>0.219244</td>
<td>-3.06756</td>
<td>0.0024</td>
</tr>
</tbody>
</table>

| R-squared   | 0.924283    |            |             |        |
| Adjusted R-squared | 0.919159  |            |             |        |

In Table 5.5A and Table 5.5B, the long run coefficients of estimated model, standard error, t-statistics and p-values of the parameters of the model generated from the DOLS and FMOLS method are presented.

The outcome of this method is not so different from that of the FMOLS presented above. It can be seen that the coefficients of our parameters of LnGDPPC, LnLEXP and LNMORATE
have the same sign as in FMOLS and they are in line with the a priori expectation while LnPOPG is negative negating the theoretical postulation in DOLS, but it is positive in the FMOLS.

It can be observed from Table 5.5A that LnGDPPC is statistically significant while LnLEXP is insignificant as they are in the FMOLS model. However, unlike in the FMOLS model LnPOPG is found to be negative and significant while LnMORATE is found be insignificant

The Adjusted R-squares from the DOLS estimates depicts that about 0.98% change in the per capita Health expenditure of the region is explained by the changes in the independent variables included in the model of this study while the value of the Adjusted R-squared from the FMOLS estimates indicates that changes in the independent variable accounts for about 0.92% variation in the dependent variable. Overall, the values of the Adjusted R-squares indicate that the estimated model is a good fit.

Since this study adopts the FMOLS estimate as appropriate, the detailed explanation of the result presented in Table 5.5 B is stated here based on the FMOLS estimate.

Looking at the coefficient, p-value or the t-statistic, we can conclude that LnGDPPC is positive and statistically significant at the 5% significant level since the p-value is less than 0.05. This result confirms the finding of Elmi and Sadeghi (2012), Nyamwange (2012), Bedir (2016) and Boachie et al., (2014). The coefficient indicates that a 1% increase in the variables LnGDPPC will cause LnHEPC to increase by 0.59%. This implies that income of the nations can positively influence the level of health expenditure on the long run.

The coefficients of the variables, LnLEXP and LnPOPG are equally consistent with the findings of Nyamwange (2012) and Boachie et al., (2014) since they are both positive and statistically insignificant at 5% level since their p-values are higher than 0.05. The results indicates that a 1% increase in the variables LnLEXP and LnPOPG will cause LnHEPC to increase by 0.90% and 0.05% respectively

It is also observed from Table 5.5B that LnMORATE is negative and statistically significant at the 5% significant level since the p-value less than 0.05. The coefficient of LnMORATE shows that a 1% rise in LnMORATE will cause LnHEPC to fall by approximately 0.67%. This result is consistent with the findings of Rajagopal (2016), Lv and Zhu (2014) and Yaqub, et al. (2012) where an inverse relationship is seen to exist between health expenditure, and infant mortality rate and under-5 mortality rate respectively.
The outcome of this model is robust and supports the cointegration result obtained earlier by reinforcing the fact that the variables have a long run equilibrium relationship.

5.6 LAG LENGTH SELECTION CRITERION

The appropriate lag length for estimating the VAR is selected based on the information criterion in Table 5.6. The lag length selected based on the information provided by the various criteria is 3 and since the maximum integration order is 1, restriction is placed on the fourth lag, as suggested by Toda and Yamamoto (1995).

Table 5.7 Summary of Lag Length Selection Criterion

<table>
<thead>
<tr>
<th>Lag</th>
<th>LogL</th>
<th>LR</th>
<th>FPE</th>
<th>AIC</th>
<th>SC</th>
<th>HQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2827.367</td>
<td>3130.827</td>
<td>6.37E-17</td>
<td>-23.10306</td>
<td>-22.3054</td>
<td>-22.7817</td>
</tr>
<tr>
<td>2</td>
<td>3659.639</td>
<td>1553.574</td>
<td>7.64E-20</td>
<td>-29.83032</td>
<td>-28.6701</td>
<td>-29.3628</td>
</tr>
<tr>
<td>3</td>
<td>3958.551</td>
<td>545.5148*</td>
<td>7.80e-21*</td>
<td>-32.11292*</td>
<td>30.59015*</td>
<td>31.49936*</td>
</tr>
</tbody>
</table>

* indicates lag order selected by the criterion

LR: sequential modified LR test statistic (each test at 5% level)

FPE: Final prediction error

AIC: Akaike information criterion

SC: Schwarz information criterion

HQ: Hannan-Quinn information criterion
5.7 PANEL CAUSALITY TEST

Table 5.8 shows the panel causality test and from the table we can observe that at 5% significant level we can reject the null hypothesis that GDPPC does not Granger-cause HEPC and also that HEPC does not Granger-cause GDPPC. Hence, it is concluded that causality runs in both direction and this result confirms the finding of Elmi and Sadeghi (2012) and Bedir (2016). Also, there is no causality from any of the other independent variables (LEXP, POPG, and MORATE) to HEPC in the ECOWAS region. This implies that GDPPC is a very crucial determinant of the level of growth of per capita health expenditure; hence there is a need to improve the level of growth in the region.

Table 5.8 Summary of Panel Causality Test

<table>
<thead>
<tr>
<th>Panel Granger Causality Tests</th>
<th>Obs</th>
<th>F-Statistic</th>
<th>Prob.</th>
<th>Granger causality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample: 1995 2014</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lags: 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Null Hypothesis:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNGDPPC does not Granger-Cause</td>
<td>240</td>
<td>3.29429</td>
<td>0.0119</td>
<td>bidirectional causality</td>
</tr>
<tr>
<td>LNHEPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNHEPPC does not Granger-Cause</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNGDPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNLEXP does not Granger-Cause</td>
<td>240</td>
<td>2.77075</td>
<td>0.028</td>
<td></td>
</tr>
<tr>
<td>LNHEPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNLEXP does not Granger-Cause</td>
<td>240</td>
<td>7.39907</td>
<td>1.00E-05</td>
<td></td>
</tr>
<tr>
<td>LNHEPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNLEXP does not Granger-Cause</td>
<td>240</td>
<td>1.7777</td>
<td>0.1341</td>
<td>no causality</td>
</tr>
<tr>
<td>LNHEPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNLEXP does not Granger-Cause</td>
<td>240</td>
<td>1.43943</td>
<td>0.2217</td>
<td></td>
</tr>
<tr>
<td>LNPOPG does not Granger-Cause</td>
<td>240</td>
<td>6.31682</td>
<td>8.00E-05</td>
<td>no causality</td>
</tr>
<tr>
<td>LNHEPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNPOPG does not Granger-Cause</td>
<td>240</td>
<td>1.08462</td>
<td>0.3648</td>
<td></td>
</tr>
<tr>
<td>LNMORATE does not Granger-Cause</td>
<td>240</td>
<td>0.6839</td>
<td>0.6038</td>
<td>no causality</td>
</tr>
<tr>
<td>LNHEPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNMORATE does not Granger-Cause</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LNLEXP does not Granger-Cause</td>
<td>240</td>
<td>3.88017</td>
<td>0.0045</td>
<td>bidirectional causality</td>
</tr>
<tr>
<td>LNGDPPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Granger-Cause</td>
<td>2.77623</td>
<td>0.0278</td>
<td></td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------------------</td>
<td>---------</td>
<td>--------</td>
<td></td>
</tr>
<tr>
<td>LNGDPPC does not Granger-Cause LNLEXP</td>
<td></td>
<td>240</td>
<td>1.44935</td>
<td>0.2186</td>
</tr>
</tbody>
</table>

Unidirectional causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>3.18613</th>
<th>0.0142</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNGDPPC does not Granger-Cause LNPOPG</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>1.89593</th>
<th>0.112</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNGDPPC does not Granger-Cause LNMORATE</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>19.9043</th>
<th>4.00E-14</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNPOPG does not Granger-Cause LNLEXP</td>
<td></td>
<td>240</td>
<td></td>
</tr>
</tbody>
</table>

Unidirectional causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>6.10011</th>
<th>0.0001</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNLEXP does not Granger-Cause LNPOPG</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Unidirectional causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>1.12166</th>
<th>0.347</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNMORATE does not Granger-Cause LNLEXP</td>
<td></td>
<td>240</td>
<td></td>
</tr>
</tbody>
</table>

Unidirectional causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>4.04548</th>
<th>0.0034</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNLEXP does not Granger-Cause LNMORATE</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Unidirectional causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>1.73992</th>
<th>0.142</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNMORATE does not Granger-Cause LNPOPG</td>
<td></td>
<td>240</td>
<td></td>
</tr>
</tbody>
</table>

No causality

<table>
<thead>
<tr>
<th></th>
<th>Granger-Cause</th>
<th>2.22285</th>
<th>0.0673</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNPOPG does not Granger-Cause LNMORATE</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No causality
5.8 NORMALITY CHECK

The normality check from the DOLs and FMOLs model estimated are presented in fig 5.3a and fig 5.3b respectively. These tests showed that the residual is not normally distributed given that the pvalue of the Jarque-Bera statistics in both cases is less than 5%.

5.9 CONCLUSION.

This chapter focused on the presentation and discussion of results obtained from the logical steps presented in chapter four. The specified model was estimated and it was found that all the variables included in the model have the expected signs. The descriptive statistic was presented followed by visual inspection which indicates that only HEPC and GDPPC are
stationary at first difference while the other variable was not clear. Based on the outcome of the visual inspection, panel unit root tests were carried out and from both tests, it was discovered that only HEPC and GDPPC are stationary at first difference while the rest are stationary at level. Since the order of integration is mixed, the study proceeded with the cointegration where both Pedroni and Kao tests were performed and they both established that the variables are cointegrated. The next step taken was to run the FMOLS and DOLS to generate the long run coefficients of the variables. The estimates of these models are slightly different; hence the study adopted the estimates of FMOLS since the model had been found appropriate for handling variables of mixed order of integration. The direction of causation between the variables was estimated since they are found to be integrated but it was first estimated using a panel Vector Autoregressive Model (PVAR) and the appropriate lag length based on the lag length criterion was determined and finally the causality test was performed. The results were all obtained through the use of Eviews 9.05 and were presented in tables.
CHAPTER 6

SUMMARY, CONCLUSION AND POLICY RECOMMENDATION

6.1 SUMMARY AND CONCLUSIONS OF THE STUDY

The main purpose of this study was to empirically examine the relationship between health expenditure and economic growth of the ECOWAS countries from the period 1995 to 2014. The study was also set out to examine the direction of causation between health expenditure and economic growth (income) as well as highlight the importance of health expenditure to growth of an economy. To accomplish the above stated goals, the study was structured into five major chapters and the sixth chapter is the final chapter; it captures the summary, conclusion and the policy recommendation.

Chapter one laid the foundation for the study whereby focus was on the introduction to the study, the problem statement, the research objective, the research hypothesis and significance of the study.

The study proceeded to Chapter two which handled the overview of health issues in the ECOWAS sub-region, examined the expenditures on healthcare, the growth of the region as well as the importance of health expenditure on economies in general. It was found that the region has been challenged by some diseases and has set up an institution to address these health issues in member countries. Also observed from this chapter is that all countries have experienced fluctuations in health expenditure for the period under investigation. Although there was a rise in the total expenditure for almost all countries from 2006, a fall was experienced in 2009, after which it began to rise but this rise is quite slow. This fall in health expenditure could lend explanation to the fall in growth experienced in virtually all countries though the fall is more severe in some countries, such as Nigeria which witnessed a sharp fall in 2009 compared to others. It can also be seen from the graph that after 2009, although the per capita GDP increased for almost all countries, the rise has been slow.

To gain direction on the variables appropriate for this study, chapter three was centred on the discussion of literature (theoretical and empirical) underpinning the study. Theories such as the Wagner theory of expenditure, Keynesian expenditure theory, the neoclassical growth model and the endogenous growth model were discussed in this chapter alongside the empirical literature. However, the versions of Wagner’s theory of expenditure as presented by
Peacock and Wiseman (1969) alongside the empirical literature of Boachie et al., (2014) were adopted to build the functional model of this study. Hence the literature reviewed justifies the necessary variables included in the model of this study as it was observed that increased income, Life expectancy and population growth will lead to increased health expenditure while increased mortality rate will lead to a fall in health expenditure.

Chapter four focuses on explicit explanations of the methodology of the study. It captured the model specification based on both the theoretical and empirical literature underpinning the study as examined in the preceding chapter. The chapter also presents the a priori expectation, data description/source and the modelling strategy employed to achieve the aim of the study. The Panel cointegration approach was utilized as the modelling techniques suitable for this study. Thus, the systemic or logical procedure embarked upon includes: descriptive statistics, visual investigation of the variables followed by the unit root test which was performed with the Levin at el. and Im Persran unit root test. This was immediately followed by tests of cointegration to determine the long run relationship among the variables after which the Panel FMOLS and DOLS were carried out to identify the long run effect of the independent variables on the dependent variable after which the panel causality test followed.

Having set out the modelling strategy in chapter four, in chapter five, the focus was on the estimation, presentation and discussion of results obtained from the logical steps presented. The specified model was estimated and it was found that all the variables included in the model had the expected signs. Also, the descriptive statistic was presented. It showed that LNHEPPC, LNGDPPC are positively skewed while LNLEXP, POPG and LN MORATE are negatively skewed. This implies that the series here are not symmetrical. It was also found that the series are not normally distributed given that the probability of the Jarque-Bera statistics for all the variables are less than 5% significance level. The kurtosis shows the flatness or peakedness of the distribution and from the table, the kurtosis is from 2.742864 to 11.89283. Field (2009) is of the opinion that the test for skewness and kurtosis should be done with large sample as they may be significant though skew and kurtosis are not far from normal.

The graphs of the variables indicates that only HEPC and GDPPC are stationary at first difference while the other variable was not clear hence the panel unit root test was carried out to confirm the stationarity of the variables and from results of both tests, it was discovered that only HEPC and GDPPC are stationary at first difference while LnLEXP, LnPOPG and
LnMORATE are stationary at level. Since the order of integration is mixed, the study proceeded with the cointegration where both Pedroni and Kao tests were performed and they both established that the variables are cointegrated.

The DOLS and FMOLS estimation were used to generate the long run estimates. However the outcome of the FMOLS seems suitable for this study since our variables have different orders of integration and it had been found appropriate for handling variables of mixed order of integration. The results of this model indicates that all the variables included in this model are in line with a priori expectation, although LnGDPPC and LnMORATE are statistically significant at 5% significance level while LnLEXP and LnPOPG are insignificant at the 5% significant level. These results indicate that a 1% increase in the variables LnGDPPC, LnLEXP and LnPOPG will cause LnHEPC to increase by 0.59%, 0.90% and 0.05% respectively while a 1% rise in LnMORATE will cause LnHEPC to fall by approximately 0.67%.

The study also examined the direction of causation between the variables since they are found to be integrated and cointegrated. However, to successfully do this, the Toda and Yamamoto (1995) causality test was applied. Thus, the study first estimated a panel Vector Autoregressive Model (PVAR) and determined the appropriate lag length based on the lag length criterion and finally a causality test. The outcome indicates that there is bidirectional causality between health expenditure and GDP per capita for the ECOWAS region while no causality was observed from any of the other independent variables of this model.

Based on the results obtained, the null hypotheses specified for this study can be rejected at 5% significance level. This means that, economic growth of the ECOWAS countries has a positive relationship with health expenditure. Also, rejecting the null hypothesis that economic growth does not Granger-cause health expenditure implies that economic growth actually Granger-causes health expenditure in the ECOWAS.

The study found from literature as well as the causality test that health expenditure also determines the growth of an economy. This implies that an increase in health expenditure would lead to an increase in economic growth of nations.

6.2 POLICY RECOMMENDATIONS

The per capita GDP has been found to be positive and statistically significant in relation to health expenditure for the ECOWAS countries and, by implication, as the per capita GDP increases, potential spending on health increases. The coefficient of GDPPC is less than
unity; hence it means that health expenditure in this region is a necessity and not a luxury. This result is consistent with the findings of Bac and Le Pen (2002) and Freeman (2013), who are of the view that income elasticity of health expenditure less than unity would mean that health expenditure is a necessity. As a necessary good, it means healthcare is a normal good because the increase in income would cause a rise in the demand for healthcare though the rise in healthcare demand is less than the rise in income. The implication would be that to improve the health of the people, there is a need to stimulate the growth of these countries. Policies targeting at the growth of the economy would also stimulate investment in the health sector for these economies. Therefore, this variable should be given critical attention so as to achieve improvement in healthcare. The elasticity of the per capita GDP should be considered in the policy framework as a benchmark that will determine the least financial outlay of the per capita health expenditure, bearing in mind that health is a capital which would mean that investing more to improve it will result in an increase in income which will generally lead to growth of the economy.

The coefficient of the life expectancy showed that there is a positive relation with healthcare expenditure. Hence, the policy implication is that to improve the health of lives in this region there is a need to spend more on health care. This will be so because to achieve the gain of healthiness, there must be an increase in the investment in the health sector.

Though the relationship of population growth was not statistically significant, it indicates that policies that will increase the growth of population, will lead to increased investment in the health sector. The governments of this region must make every effort to control the rate of population growth because an increase in the growth of the population will definitely lead more spending on healthcare.

The coefficient of Mortality rate (infant) is statistically significant and negative which would mean that a rise in the rate of infant mortality would significantly decrease healthcare expenditure. However, since there is an inverse relationship between these variables, increased health spending would lead to decline in infant mortality rate in the ECOWAS region. This outcome is consistent with findings of Rajagopal (2016). These results indicate that critical attention must be given to these variables especially the level of income, so as improve the level of health expenditure in the ECOWAS region.
6.3 LIMITATION OF THE STUDY

This study only examined the relationship between health expenditure and economic growth for the period 1995-2014 due to the unavailability of data for some countries in the region up to 2016. Also, it was not possible to include some of the variables identified in literature that influence health expenditure such as physicians per 100,000 populations among others due to unavailability of data. Hence this study is not exhaustive meaning that it also gives insight into areas of further research.


Indicators, A.H.O. 2010. Monitoring the building blocks of health systems.


McIntyre, D. & Gilson, L. Equitable health care financing and poverty challenges in the African context.


UNAIDS, 2016. 'Prevention Gap Report'.


WHO (World Health Organization) 2005. World Health Development Indicators. Washington, DC.


WHO, 2007. Global Tuberculosis Database online, WHO.


World Bank, 2004. Those countries having an index equal to 3.5 of the “Country Policy and Institutional Assessment Index” are those that would obtain these benefits on average

