UNIVERSITY OF CAPE COAST

STRUCTURE AND CAUSES OF MORTALITY IN THE KASSENA–NANKANA DISTRICT OF UPPER EAST REGION OF GHANA: 1995–1999

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2009

DECLARATION

Candidate's Declaration

I hereby declare that this thesis is the result of my own original work and that no part of it has been presented for another degree in this university or elsewhere.

Candidate's Signature:..... Date:.....

Name: Joshua Amo–Adjei

Supervisors' Declaration

We hereby declare that the preparation and presentation of this thesis were supervised in accordance with the guidelines on supervision of thesis laid down by the University of Cape Coast.

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ABSTRACT

Since 1662 demographers have been interested in mortality with the discourse centred around how mortality movements had impacted on population characteristics such as growth. Later discussions have aimed at spotting variations within and between groups and corresponding social and biological distal and proximate determinants. This study contributes to the discussion on morbidity and mortality by analysing data from a rural district in the Upper East Region of Ghana in time, space, season and socio–demographic characteristics.

The study adopted Omran's (1971) epidemiological transition theory as a framework, and used longitudinal data between 1995 and 1999 from Navrongo Demographic Surveillance System site. Analyses of structure and causes of death were done at the population level.

Results showed gradual shift in causes of death from communicable, maternal, prenatal and nutritional related diseases to non–communicable diseases such as cardiovascular disease within the period. There was also a general decline in mortality, with the southern zone, the most rural reporting the highest in almost all the cause–specific and age–specific mortalities. The findings suggest that Omran's model cannot have a strict universal application, as this study area, though relatively homogeneous, some notable spatial variations in the number of deaths and the causes thereof were observed. It is suggested that health needs must be assessed at more disaggregate levels since disease burdens differ substantially by time, space and socio–demographic characteristics.

ACKNOWLEDGEMENTS

My first appreciation goes to Prof. K. Awusabo–Asare (Principal Supervisor) for being a father and mentor, who continues to motivate me to diligently seek for knowledge. My Co–Supervisor, Dr. Kwabena Barima Antwi also deserves a special commendation. Indeed, words are not enough to express my gratitude to them.

I would also like to convey my profound gratefulness to Dr. Abraham Hodgson (Director) and Dr. Cornelius Debpuur (Head, Population Unit) and Mr. Martin Adjuik Alhassan (Data Manager), all of Navrongo Health Research Centre for making the centre's Demographic and Surveillance System data readily available to me at no cost. My gratitude also goes to William and Flora Hewlett Foundation and the INDEPTH Network for their financial assistance in carrying out this work. I would again want to express gratitude to my siblings Mr. Stephen Amoako, Mr. Emmanuel Amo–Adjei, Mr. Samuel Amo–Adjei and Miss. Hannah Fosuah Amo for their inspiration and encouragements as well as their immense financial support all these years.

I am also grateful to Dr. Akwasi Kumi–Kyereme, Mr. Eugene Kufour Maafo Darteh and Mr Augustine Tanle of the Department of Population and Health, Mr. Emmanuel Mensah Abeashi of the Department of Geography and Regional Planning and Dr. Ahmed Adu–Oppong of the School of Medical Sciences, UCC. Miss Mavis Osei Effah provided me with much emotional support and she can never be forgotten for her goodness to my academic life. Nonetheless, I wholly remain responsible for any weakness in this material.

DEDICATION

To my parents, Papa Amo and Maame Amoakoaa

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LIST OF ACRONYMS

ALRI	Acute Lower Respiratory Infection
AOCD	All other communicable diseases
AONCD	All other non–communicable diseases
CDC	Centres for Disease Control and Prevention
CHERG	Child Health Epidemiology and Reference Group
CHFP	Community Health Family Planning
CKI	Community Key Informant
CNS	Central Nervous System
COD	Cause of Death
CRB	Compound Registration Book
DSS	Demographic Surveillance System
GBD 2000	Global Burden of Disease 2000
GSS	Ghana Statistical Service
HRS	Household Registration System
ICD+10	International Classification of Diseases 10^{th} Version
INDEPTH	International Network for Demographic Evaluation of developing countries Population and Their Health
KND	Kassena–Nankana District
MDG	Millennium Development Goals
NDSS	Navrongo Demographic Surveillance System
NHRC	Navrongo Health Research Centre
UN	United Nations
VAST	Vitamin–A Supplementation Trial
WHO	World Health Organisation

YLD	Years of life with disability
YLL	Years of life lost

CHAPTER ONE

INTRODUCTION

Background to the study

From ancient to contemporary times, one way that national, regional (state) and local governments have measured their successes is how human lives have been prolonged. Historically, good governance had been assessed on rate of population growth, defined as either an increase in fertility or reduction in mortality or both. Caldwell's observation is instructive:

> For most of human history, the chief explanation for mortality declines have been good government in the sense of strong governments that kept the peace, suppressed internal disorder and violence, avoided or mitigated famine, and attempted to mitigate the worst excesses of epidemics … early attempts to record mortality were aimed partly at providing an index of good government (Caldwell, 2001a: 25).

Observations from past and present analyses indicate a mix of directions: interrupted or uninterrupted declines and patterns of increases as well as stagnation in mortality. There is no record of either a unidirectional decline or increase, resulting in alternation among increased, stagnated and or decreased trends (Caldwell, 2001b).

Until the turn of the 20th Century, mortality dynamics were mainly influenced by infections. Predominant diseases included, but were not limited to, cholera, small pox, dengue fever, and tuberculosis. Chronic and man–made diseases such as cancers are quite recent (Caldwell, 2001b). The transition from infectious diseases to chronic diseases in developed countries was not by chance or natural providence; rather, they resulted from deliberate efforts to overcome infections, even though, some of the approaches engaged might have been indirect (Caldwell, 2001b).

Currently, almost all countries throughout the world have seen some changes in diseases and mortality characteristics. During the 1960s, researchers speculated a convergence of mortality in developing countries before the end of the 20th Century, regardless of prevailing socio–economic conditions (Stolnitz, 1965 cited in Adetunji & Bos, 2006). In fact, public health experts and advocates in the 1960s and early 1970s were, without cautious optimism, arguing that the fight against infectious diseases had ended. Sanders Fuhrer, Johnson and Riddle (2008) quoted US Surgeon General, William H. Stewart as confidently stating that, the time had come "to close the book on infectious diseases", and instead, attention be given to chronic diseases like cancer and cardiovascular diseases. Almost five decades on, these predictions have been true for only developed countries. Even though these sentiments and predictions have failed to be the reality, quite clearly, they reflected the realities of the time.

Countries in the developing world show a clear contrast to developed countries in terms of pace of progress in disease patterns and trends. For the greater part of human history, Africa has been characterised by extremely high

mortality levels and fatal infectious diseases. Even so, this phenomenon is changing. Adetunji and Bos (2006) observe that one of the remarkable achievements of the 20th Century Africa was the continuous improvements in health, which was reflected in mortality patterns, trends, differentials and levels.

Life expectancy in the early 20th Century was, for instance, less than thirty (30) years, the level of Europe one–hundred and fifty to two hundred years earlier. From 1950–55, average life expectancy in most developing countries was between 35–45 years, far behind developed countries which was around 65–70 years. In 1960–69, the worldwide life expectancy at birth was 52.5 years. The highest recorded by world a region was in North America (70.1 years), while Africa recorded the lowest (42.4 years). In 2004, the average for Africa had slightly improved to 45.9 years while that of North America had increased to 73.0 years. Among sub–Saharan African regions, the highest life expectancy at birth is in South Africa (47.7 years). West Africa recorded 46.3 years and the lowest was Middle Africa; 43.4 years (United Nations, 2005). The pattern had slightly changed by 2008, with Africa's life expectancy increasing to 51 years for both sexes (World Health Organisation [WHO], 2008).

Notwithstanding this slow pace of change in the 1990s, Africa, on the average, is making progress, though not substantial and consistent as expected. Since the 1960s, the rate of change in expectation of life has been estimated to be about 3.5 years. Some researchers have argued that some regions on the continent reached their highest ever–recorded health improvement in 1990. Undoubtedly, the onset of HIV/AIDS in Africa has eroded much of the

improvements that were expected in life expectancy at birth (Adetunji & Bos, 2006). HIV/AIDS in particular, and other emerging and re–emerging diseases that unfolded on the African continent and other developing countries rendered Stolnitz's (1965) cited in Adetunji and Bos (2006) prediction void. The seeming progress was curtailed by an abrupt divergence from the predicted convergence, except in few countries such as Sri–Lanka, which continued to record notable progress in mortality inclinations (Adetunji & Bos, 2006).

These pictures about morbidity and mortality patterns in advanced and developing countries present considerable interest, and sometimes, controversy among demographic and public health researchers, especially, the disease specifics. Global data for 2003 for low–income countries indicated five of the ten major causes of death and disease burdens to be in Group I as (communicable, maternal, peri–natal and nutritional conditions) cause of death and disability. In developed countries, nine of the ten major causes of death were in groups two (non–communicable diseases) and three (injuries). The highest burden of disease in high–income countries was uni–polar depressive disorders (9.0%). Road traffic accident was the lowest (2.6%). The situation in developing countries was different. Peri–natal conditions topped the list (7.0%) with malaria and tuberculosis being the lowest (2.5% each). In terms of mortality, ischemic heart disease caused 12.1 percent of deaths in low and middle–income countries, while the same condition was highest in high–income countries (17.0%). The lowest for both regions was malaria, 1.8 percent each (WHO, 2004).

Different segments of populations also present other revelations. Out of the total 57 million global deaths for 2003, 20 percent were among children under five and of these, 99 percent occurred in developing countries, with over 40 percent in sub–Saharan Africa. Demographers have made several attempts over the years to adduce convincing reasons to explain the documented differences in diseases and deaths in developing and developed countries. For instance, Razzell (1974) argued that inoculation against smallpox in the 18th Century and improved hygiene as well as the use of detergents and washable cotton clothes (public health) in Europe were the key factors in improvements of mortality. McKeown's (1976) thesis on mortality transition in England, placed much emphasis on nutrition, necessitated by improved incomes. Landers (1992) also linked the differential patterns to biological and climatic influences in diseases and deaths transitions.

Most of these linkages have frequently been used in discussions on health transitions. Based on such suppositions, some place little premium on changing living conditions (distal) and at the extremes, discount such variables. Even among authors who are aligned to proximate determinants, there are distinctions in opinions as per mortality dynamics. Caldwell (2001b) attempts to strike a balance between distal and proximate determinants and argue that absolute or relative contribution of any single variable remains an illusion to both biological and social epidemiologists. Meanwhile, Krieger (2008) proposed that the use of the proximate–distal lexicon should be stopped as the two concepts obscure the connection between levels, pathways and power in understanding causal mechanisms in diseases and their outcomes. Nevertheless, whatever the case could be, some of these efforts have resulted

in past and present trends and patterns of health observed across world regions.

Another controversial discourse on what is important to be studied in epidemiological studies relates to the morbidity–mortality interfaces. While some authors believe that only mortality should be described and analysed, others support combining morbidity and mortality. Ruzicka and Kane (1990) cited in Johansson (1991, p.41) favour studying mortality alone to understand the true state of a country's health. Their point of view is that: "… since rising morbidity and declining mortality seem so fundamentally inconsistent with one another, … measures of mortality are and remain the best indicators of a population's 'real' health status, and thus the best measurable indicator of morbidity levels" (Ruzicka & Kane, 1990, cited in Johansson, 1991; p, 41). Ruzicka and Kane's (1990) conception might have arisen from the reversibility of some morbid conditions. Some people recover from diseases without necessarily consulting a physician. Consequently, not all cases will be captured by a health system. However, for policy intervention purposes, Johansson (1991) states that:

> This solution is very unsatisfactory because it sabotages the systematic study of morbidity data. In policy–related terms, it leaves open the possibility that the rise of morbidity is indeed illusory, and … generous health benefits and disability programs should be cut back in the developed countries, and not extended to people who live in developing ones (p, 41).

Holland and Breeze (1988) cited in Nolte and McKee (2004, p. 47) also note that mortality alone does not provide a conclusive index of actual

health performance of any specific jurisdiction. Considering mortality only then presupposes that those who seek care for pain relief and quality of life are neglected in measuring real changes in a population's health status. Holland and Breeze (1988) state that "while careful examination of mortality from specific causes can provide information on the outcome and effectiveness of health services, mortality is not always an appropriate indicator" (cited in Nolte & McKee, 2004, p.5).

Mortality data are also sensitive indicators of differences within a national population in the degree of progress towards modern conditions, thereby helping to identify target groups for special health and development programmes. They can also be very useful in evaluating the successes of programmes already instituted. Mortality levels are related to other variables of social concern, such as labour productivity, fertility, and require attention because of those relationships. In evaluating how successful a certain area has been in modernizing its health conditions, it is necessary to compare the situation in an area to achievements that have been recorded elsewhere (United Nations [UN], 1986).

Nonetheless, valid as these differing opinions may be, the choice of one over the other (morbidity or mortality) or the combination of the two needs to be evaluated in the light of availability of quality data. In African countries where these data needs are notoriously unreliable, the task becomes more daunting. However, past and current mortality studies aid in understanding future demographic issues. The tracking of cholera fatalities by Snow (1854) to contaminated pipe water in England is recognised as a landmark epidemiological study. His conclusion was based on mapping of

cholera incidence on spatial principles based on mortality data and concluded premised on who felt sick, which did not, and where and how the sick and healthy lived. Nine years later, William Budd made use of Snow's approach to study typhoid in 1859 in Taw Valley (UK). Through such spatial studies, their contributions were seminal in population health (Barry, 2005). These issues have informed the need to track mortality trends, patterns, differentials and levels that have evolved in the Kassena–Nankana District in the Upper East Region of Ghana, from 1995–1999.

Current epidemiological profile of Ghana

A review of data on Ghana by Adams and Boerma (2006) specified malaria to be the highest cause of death (15%) as well as the highest cause of years of life lived with disability (YLD) – 16 percent. The annual report of Ghana Health Service (2006) shows an increasing trend in non–communicable diseases; even though communicable diseases continue to be the greatest in burden of diseases. The report indicates that hypertension emerged as one of the commonest outpatient diagnosed diseases and was among the top ten (10) in all regions of the country among patients 15 years and above. Approximately 80 percent of deaths among children under–five years are attributed to four causes; malaria (33%), pneumonia (17%), conditions arising from peri–natal period (17%) and diarrhoea (14%). The first three causes of death (malaria, pneumonia and peri–natal) put together accounted for two–thirds (67%) of all deaths among children under–five years in Ghana. More than 80 percent of deaths among neonates are caused by three main conditions: sepsis (29%), birth asphyxia (26%) and prematurity (25%). In

Ghana, communicable diseases are still the major cause of disease burden. Overall, malaria is the leading cause of disease burden in all ages, accounting for 16 percent of all disease burdens in Ghana. Among adults aged 15 to 59 years, HIV/AIDS imposes the largest burden of YLD. Ischemic heart disease and stroke (cerebrovascular disease) are two of the four leading causes of disease burden in older age groups (60 years and above) (Adams & Boerma, 2006).

Problem statement

Epidemiological literature is replete with studies on the important linkages between health and development. This desire was re–echoed at the millennium summit in September 2000 when world leaders at the summit agreed to implement eight goals for the millennium. Out of the eight goals, three are directly about improving health. Goals four and five are specifically concerned with improving mortality (maternal and infant/child) while the sixth goal is towards morbidity change (reduce malaria incidence and reverse HIV/AIDS spread). The remainders have indirect impact on health (eradicate extreme poverty and hunger, achieve universal primary education, promote sex equality and empower women, ensure environmental sustainability, develop a global partnership for development) (Bygbjerg & Meyrowitsch, 2007).

In spite of these efforts, differences and variations in morbidity and mortality exist due to local conditions. Therefore, there is the need to analyse diseases and deaths by locality. Yet, the bulk of literature on diseases and their outcomes in Ghana have concentrated on the national level than smaller units

such as the Demographic and Health Surveys (Ghana Statistical Service (GSS), Noguchi Memorial Institute for Medical Research (NMIMR) & ORC Macro, 2004; Ghana Statistical Service, Ghana Health Service & ICF Macro, 2009). Others which considered disaggregate data have been more inclined towards maternal and infant mortality dynamics. (See for instance, Kiersten, Rutstein, & Govindasamy, 2005; Adams & Boerma, 2006). Rao, Lopez and Hemed, (2006) studied the trends and patterns in African epidemiological studies and observed that about 80 percent of studies were on maternal and infant health outcomes. However, such aggregate level analyses, whether within or between groups, could have high odds of missing very important details at sub–national or regional levels. Murray and Salomon (2002) have argued that a step towards appreciating the health of a given population is conscious and systematic documentation of developments in mortality as well as possible conditions that could have precipitated the observed transformations among populations.

Since 1993, the Navrongo Demographic Surveillance System (NDSS) has been collecting data on causes of death through verbal autopsy (VA) (see Chapter 3 on VA). The wealth of data available at the site, and the studies and papers emanating from NDSS are immense, though not proportional to the available data and more inclined towards the biomedical realms. On the basis of this, Baiden, Hodgson and Binka (2006) have stated that one of the problems of demographic surveillance sites has been few longitudinal analyses of the data acquired in these sites over the years.

This thesis uses part of the surveillance data from the NDSS to analyse mortality and its relationship with social, economic and demographic variables

in the Kassena–Nankana district from 1995–1999. It contributes to the wealth of knowledge in population health that has emerged from the site.

Objectives of the study

The main objective of this study was to examine the structure of all–cause mortality occurrences in Kassena–Nankana in the context of epidemiological transition theory. The specific objectives were to:

- Identify annual variations in mortality in Kassena–Nankana;
- Examine spatial patterns of mortality in the Kassena–Nankana from 1995–1999;
- Identify the links between mortality and demographic variables;
- Appraise seasonal and monthly dynamics of mortality in the Kassena–Nankana over the period of investigation (i.e. 1995–1999); and
- Make practical recommendations for dealing with, and reducing the burden of diseases common in the study area.

Hypotheses for the study

Contextualising the study within the epidemiologic triad (person, place and time), it is expected that there would not be differences in sex and age (demographic) patterns of mortality and the causes of deaths thereof. These premises are made because risk factors are likely to be influenced by age and sex, which are informed by social and biological aspects that influence health. Based on the foregoing, the following hypotheses were proposed for the study.

1. H_0: There is no significant relationship between places of residence mortality patterns.

H_1: There is a significant relationship between place of residence and mortality patterns.

2. H_0: There is no significant relationship between demographic variables (sex and age) and trends in mortality.

   H_1: There is a significant relationship between demographic variables (sex and age) and trends in mortality.

3. H_0: There is no significant relationship between deaths among males and females and monthly patterns.

   H_1: There is a significant relationship between deaths among males and females and monthly patterns.

4. H_0: There is no significant relationship between deaths among various age groups and month of death.

   H_1: There is a significant relationship between deaths among various age groups and month of death.

Rationale

Among other things, this work will contribute towards the understanding of quality of health care that prevails in KND through the concept of avoidable death (Nolte & McKee, 2004). This approach provides a framework for identifying health challenges in the area to provide some information on health in a rural setting in Ghana.

Spatial dynamics of events, especially mortality are critical for policy planning. Spaces are important fundamentals of epidemiologic inquiry since different ecological zones present their own health hazards. This study combined spatial elements with time to analyse mortality and diseases that

caused these deaths in the Kassena–Nankana District. This is very relevant as too often, studies on health disparities in Ghana are at regional or at best district level differences. This study assesses morbidity and mortality among clusters in a district.

Another relevance of this study is the analysis of mortality and the diseases that caused these deaths at the ecological level. This analysis tends to bring out the likely environmental and structural conditions that make some populations more vulnerable to ill health, useful for policymaking. With this macro conception in perspective, this study has appended the need for the changing paradigm shift in population health research that seeks to address population level issues rather than few individual needs.

Finally, viewed from the perspective of longitudinal studies, epidemiological investigations are highly limited in Ghana, as the present study has offered evidence of progress in achieving the health–related Millennium Development Goals, which has 1990 as the base period. The study also adds to the debate on the viability of epidemiological transition in explaining mortality trends in Ghana.

Organisation of the study

This thesis is organised in six chapters. The introductory Chapter one puts the study into perspective through introduction, problem statement, objectives and hypotheses. Other issues are rationale behind the research and how study has been organized. The next chapter discuses related literature with specific reference to psychosocial theory, social/political economy of health, ecosocial theory, the pathocenosis theory, the concept of avoidable

mortality and the epidemiological transition theory. Chapter three outlines study design, sources of data, description of the study area and NDSS data collection procedures and analytical techniques involved in the study. Chapter Four was devoted to presentation and discussion of results related to the overall structure of mortality in the district. Chapter Five outlines issues on diseases that caused the mortalities thereof. The last chapter ends the thesis with summary of findings, conclusions and recommendations as well as avenues for future research.

CHAPTER TWO

REVIEW OF RELATED LITERATURE

Introduction

Mortality changes reflect patterns in both biological and socio–cultural influences and are therefore a valuable source of information on many important aspects of population adjustments. Longitudinal cause–specific death records are critical resources for providing information on the evolution of a group's ability to respond to environmental changes. In many historically acknowledged populations, a shift can be identified in the principal causes of death from infectious diseases and other exogenous (external) causes to degenerative diseases and diseases related to modern lifestyles (Muñoz–Tudurí, Garci´a–moro, & Walker 2006).

Contemporary compositions on mortality adaptations require an understanding of how transitions have occurred over time and several theories, models and hypothesis have emerged in explaining these events. Complementary to these theories is a concept that attempts to explain the extent to which the processes engaged are yielding expected results or otherwise, that is, avoidable mortality. A basic assumption underlying this proposition is that given an effective health system, certain diseases should not occur first and second, even if they occur, mortality should be avoidable (Nolte & McKee, 2004). Corollary to these models of transition that depict the

dynamics proposed by theories of disease distribution are the epidemiological transition and pathocenosis theories. During the mid–20th Century, there was ardent revolution for building holistic models for explaining why some people felt sick while others did not. These models were primarily based on socio–biomedical frameworks which brought to the fore the discipline of social epidemiology (Krieger, 2001a).

Apparently, social epidemiology arose out of the social movements of the 1950/60s for social justice in all spheres of human existence, of which health is at the core. Social epidemiology has, since its emergence, dealt with three main theories: psychosocial, social production of disease/political economy of health and ecosocial theory, which put together are referred to as 'theories of disease distribution' (Krieger, 2000). Changes in health status at any point in time may be a function of psychosocial, social production of disease/political economy of health and or ecosocial theory, an upshot of mortality.

The thrust of this chapter is to discuss some of the perspectives on mortality transitions. Among them are the psychosocial, social production of diseases/political economy of diseases, ecosocial, pathocenosis and the epidemiological transition theories.

Psychosocial theory

Krieger (2001a) summarised the psychosocial perspective as "a direction of attention from endogenous biological responses to human interactions and focuses on stressors and stressed people in need of psychosocial resources" (p.670). This theory emerged because of the failure of

prior theories and models, such as the bio–medical individualism, miasma and germ theories to explain the complexities of why some people, even though exposed to disease causing germs, do not become infected and why not all infected persons become sick. The core of these theories sought to prioritise biological determinants over origins. The psychosocial theory was, therefore, an expansion of simple etiological framework of agent to host–agent–environment conception (Krieger, 2001a). Cassel (1976) who initiated this theory identified an intricate link between physical and psychological vulnerability and health patterns. Cassel argued that to understand disease and outcome distributions, researchers have a responsibility to seek explanatory variables of susceptibility. Thus:

> The question facing epidemiological inquiry then is: are there categories of or classes of environmental factors that are capable of changing human resistance in important ways and making subsets of people more or less susceptible to these ubiquitous agents in our environment? (1976, p. 108).

According to Cassel (1976), health–modifying factors of host–agent–environment variables in developed countries are not likely to include malnutrition, healthcare accessibility (cost, distance) and sanitation. This is because developed countries have higher incomes that enable them to invest in human capital development and public health. Factors that are more probable may be embedded in what Cassel (1976) terms as social environment, informed by human–to–human interaction. This also arises due to more individualistic lifestyles that accompany modernisation. On the other side,

nutrition, public health and its accessibility would predominate in developing countries.

The main theme of Cassel's argument is how social environment modifies host susceptibility. Some significant psychosocial factors may include dominance hierarchies, social disorganisation, and marginal status in society. This then explains why certain social groups have different disease burdens (Cassel, 1976).

This view shifted attention from specific aetiology to generalised susceptibility and Cassel (1976) argued that the most "feasible and promising interventions to reduce health will be to improve and strengthen social supports rather than reduce exposure to stressors" (p.121) alone. However, he acknowledged that a community's disease burden is dependent on prior exposures as well.

Martikainen, Bartleyb and Lahelmac (2002) in a further extension of the concept were concerned about the levels in which psychosocial variables operated. According to them, research in understanding diseases and their consequences should consider micro–, meso– and macro–level intervening factors. Their point of view was that "psychosocial explanations of health are essentially viewed here as processes that cannot be fully captured by single measures at one level, but require due attention to macro and micro (individual) level factors as well" (p.1092). Some of the common factors are social networks, supports, and work–family pressures.

This perspective is criticised as too diffused since it does not specify the environments, whether natural or social, or interaction, which is responsible for the occurrence (Krieger, 2001a). Similarly, it downplays

biological factors which Coelho (1997) found to be very important in demographic and epidemiological transitions in the New Worlds from 1500–1800s. Coelho's (1997) argument was that transition in the New Worlds was largely influenced by genetic (biological) factors, which made some populations more resistant to tropical diseases.

Social production of disease/political economy of health

Social production of disease/political economy of health theory emerged in the 1960s to 1990s and it is premised on upstream–downstream (proximal–distal divide) synergies, which are considered to be the broad determinants of diseases and deaths (McKinlay & Marceau, 2000). The main hypothesis of this theory is that decisions made or imposed on citizenry by political heads have a direct impact on health as well as spatial distribution of diseases and death. This theory is a critique on the victim–blaming lifestyle assumptions, which give more precedence to individual's will power to choose more responsible healthy lifestyle (Doyal & Pennel, 1979). This means that, communities that experience the so–called lifestyle causes of death by implication will not benefit from country–level policies that are targeted towards population health. Implicitly, the political economy concept may be inferred to as an attempt by socialists to counter the capitalists' preoccupation of prioritising capital accumulation over basic welfare.

The focus of this perspective is to analyse economic and political determinants of deaths as well as structural variables (Turshen, 1989). For instance, the mode of production, either, through capitalism whereby a few stay rich and healthy (improved life expectancy) and the majority lurches in

vulnerable surroundings or the socialist approach of ensuring equitable distribution of resources has implications for patterns of mortality (Townsend, 1993). Thus, when, where and who benefits from public health investments are directly or indirectly determined by political considerations. Consequently, 'behind the scenes' politicking and voting patterns could influence decisions on health. Underlying this viewpoint is the extent to which others benefit from specific policies and at the expense of others. Advocates for this theory, for example, are concerned about exposure to occupational hazards, commercialization of health services (example, "cash and carry" health provision), military spending and regulation or deregulation of corporations (Krieger, 2001a).

Link and Phelan (1996) believe that economic and political institutions create and reinforce fundamental inequalities in health. Using a pooled data for the West–Africa Sub–Region, Bour (2008) concluded that political stability for example was very significant in charting the course of mortality distribution, since countries in Sub–Saharan Africa that had experienced political instability were affected most by high maternal and infant mortalities. This view is also located in the structure–agency debate, which started in the last quarter of the 20th Century and which discusses the interplay between individual and institutional factors (Giddens, 1984). Initial analyses under this perspective focused on health gaps within countries. Contemporary discourse, however, centres on international multivariate variables, such as rising income disparities and structural adjustment programmes imposed by Britton Wood Institutions (World Bank and IMF) and their impact on health outcomes (Wilkinson, 1996). Analysis in the late 20th and 21st centuries focuses on

social differences such as race, sex and sexuality as determinants of within group differences (Krieger, 2001a). Other recent forms of analyses arise from environmental justice movements, which draw attention to the effects of indiscriminate disposal of toxic waste in poor countries and communities (Kim, Millen, Irwin, & Gershman, 2000).

Ecosocial theory

Ecosocial epidemiological framework seeks to integrate social and biological reasoning in a dynamic, historical and ecological perspective to develop new insights into determinants and distribution of social inequalities in health. The central question that ecosocial theorists ask is: "who and what is responsible for population patterns of health, disease, and wellbeing, as manifested in present, past, and changing social inequalities in health" (Krieger, 2001b: p.694). In an attempt to further enhance understanding of the theory, Krieger (2005) added that, "clues to current and changing population patterns of health, including social disparities in health, are to be found chiefly in the dynamic social, material, and ecological contexts into which we are born, develop, interact, and endeavour to live meaningful lives" (p.350).

Earlier, Krieger (2001a) identified the following as the basis of ecosocial theory: embodiment, pathways and pathways of embodiment, accountability and agency. Embodiment encompasses how individuals integrate biological and social materials in their surroundings, from conception to death. This view presupposes that biological dispositions of health cannot be understood without the knowledge of history and individual and societal ways of living.

The second component of ecosocial framework is the pathway which influences embodiment. These pathway variables may re–enforce or de–link the direction of embodiment. Some micro and macro variables involved are power, patterns of production, distribution, consumption, evolutionary history, ecological inter–linkages, individual background characteristics as well as the trajectories (routes) of social and biological development (Krieger, 2001a; 2005).

Thirdly, these routes yield successive synergies between exposure, susceptibility and resistance, which are expressed in pathways of embodiment. Individual factors operate at multiple levels (individual, neighbourhood, regional, national and inter–national) and in multiple domains (example: home, and work, school, church) over multiple scales of time and space.

Another aspect of ecosocial theory is accountability and agency. This reflects the extent to which national, regional, district and or community institutions, either private or public, see their responsibilities in affecting health. Within such responsibilities, epidemiologists and other scientists, for example, will try to explain social inequalities in health.

According to the ecosocial framework, adequate epidemiological explanation is able to account for both persisting and changing distributions of diseases and deaths, including social inequalities in health, across time and space. And to aid conceptualisation, "ecosocial theory uses a visual metaphor of an evolving bush of life intertwined with the scaffolding of society that different core social groups daily reinforce or seek to alter" (Krieger 2001a: p.697).

In sum, ecosocial theory considers how population health is generated by social conditions, in combination with biological processes at every spatio–temporal scale: sub–cellular to global or nanoseconds to decades (Krieger, 1994; 2001a; 2005). A major weakness of the model is that it attempts to portray health outcomes as only plausible when there is an equal mix of social and biological courses. Nonetheless, Krieger's (1994; 2001a; 2005) consistent emphasis on social trajectories were more inclined towards man–made chronic diseases such as stroke and myocardial infarction.

The concept of avoidable mortality

Rutstein and colleagues first developed the concept of avoidable disability and death in the 1970s (Nolte & McKee, 2004). Rutstein and colleagues created a list of conditions from which death should not occur in the presence of timely and effective medical care. Avoidable mortality or premature mortality provides a comparative measure of public health and medical care, which identifies differences in health care quality or health status across regions, social class and time (Nolte & McKee, 2004). Some of the conditions leading to death are amenable to health care. This is defined as "as those (conditions) from which it is reasonable to expect death to be averted even after the condition has developed" (p.15).

On the other hand, "preventable conditions typically include those for which there are effective means of preventing the condition from occurring" (Nolte & McKee, 2004: p.16). This concept provides a distinction between causes amenable to health care and causes amenable to health policy. Other authors have set the boundaries between conditions that are avoidable due to

primary or due to secondary prevention. Primary prevention includes aspects such as immunisation, safety policies and healthy behaviour. Conditions that can be prevented by secondary prevention comprise screening programmes as well as better medical treatment (Simonato, Ballard, Bellini, & Winkelmann, 1998; Forster, 1996). Simonato et al. (1998) split secondary prevention into secondary and tertiary prevention. Secondary prevention is based upon early detection and treatment whereas improved treatment and medical care determine tertiary prevention.

Studies on avoidable mortality have some problems. The first is the coding practices in causes of death since not all countries strictly follow the international diseases classification system. A second limitation is the lack of clear distinction between avoidable and non–avoidable causes of death and the setting of age limits at which deaths could be described as avoidable. Rutstein and colleagues (1977) cited in Nolte and Mckee (2004) set the age threshold of avoidability at 65 years, beyond which deaths are not an indictment on the system. Mackenbach, Looman, Kunst, Habbema and van der Maas (1988) cited in Nolte and McKee (2004) however, reviewed the age point limit by Rutstein and others to 75 years. The attempt to discount age raises ethical issues. This is because some people in certain ages are considered unimportant and therefore "appropriate" for death to transpire among people in such age groups.

Differential avoidable mortality may be caused by differences in socio–economic status, by age or place and, by medical care supply (Kunst, Looman, & Mackenbach, 1988). Although a strong relationship between avoidable causes of death with health system indicators is expected, studies

have shown that the association could be weak and inconsistent. According to Kunst, Looman and Mackenbach (1988), socio–economic indicators explain variation better than the use of avoidable concept.

Pathocenosis theory

In a paper entitled, "Diseases at the dawn of Western Civilization" Grmek (1983) referred to in Vallin (2005) attempted to model a concept that could explain etiologic changes of specific diseases, beginning from the first human being to current days using longitudinal mortality data. This approach was presumably based on a review of past epidemiologic studies by pioneers such as William Farr, John Snow and Robert Koch and focused primarily on specific diseases. This led Grmek to contend that, medical epidemiologists have approached the study of diseases and death one at a time, as well as how specific diseases have evolved (Vallin, 2005). Grmek (1969) cited in Vallin (2005) argued for application of synthetic methods to aid understanding of epidemiological dynamics of places. It involved three postulates. The first postulate is that "pathological conditions within a given population, at one time and in one territory, form a set, which is known as pathocenosis". Secondly, "beyond specific endogenous and ecological (exogenous) factors, the frequency and distribution of each case depends on the frequency and distribution of all other cases". The final supposition contends that, "pathocenosis tends towards a state of equilibrium" (Vallin, 2005, p.281).

According to Grmek in Vallin (2005), pathocenosis' stability may be influenced by interactive relationships among diseases. These kinds of relationships may be one of the following: symbiosis, antagonism and/or

indifference. Symbiosis may be due to "etiological links at different levels; genetic, ecological and social" (Vallin, 2005: p. 281) and could be experienced at the same period. Antagonism, unlike symbiosis, arises when two different diseases struggle for supremacy in one organism. The dominating disease then functions as an antibody to the previous. For example, the antagonistic relationship between leprosy and tuberculosis has been observed in some studies indicating that leprosy receded in some Western countries when tuberculosis started to spread. Indifference is the state in which no discernible relationship can be identified among diseases regimes, whether the interface is symbiotic or antagonistic.

Basing arguments on this theory, Biraben (1999) quoted in Vallin (2005) identified eleven pathocenoses from 8650 BC to early 20th Century which gives an impression of evolutions characterized by different infectious diseases regimes. This concept promotes detailed study of causes of deaths, which were/are peculiar to specific times. However, pathocenosis may lead to possible neglect of 'other' important diseases occurring at the same time due to its focus on one disease at a time.

Epidemiological transition theory

This theory was proposed by Omran (1971) to explain mortality as part of the demographic transition. The focus of the theory is the complex dynamics of diseases and deaths on one side, and the links between demographic and socioeconomic determinants and consequences on the other. Omran (1971) acknowledged that, though the theory was intended for academic exercise to clarify the link between health and disease and their

social, economic and political determinants of the modernisation era, it "is aimed as well, at shedding light on the tenacious population problems of less developed countries" (p.732). Omran's assertion may be attributed to the fact that policy makers during his era turned much of their attention towards fertility control, as a means of reducing social and economic pressures. Nonetheless, Omran (1971) believed that long–run successes that were made in fertility transition in Western countries could not have been possible without an adjacent mortality transition.

The theory, as proposed by Omran (1971), was captured under five thematic areas, namely, mortality and population dynamics; modifications in mortality and disease patterns; relative risks of mortality by age and sex; interacting transition variables and basic models of the transition. The first assumption of the theory is that diseases and the deaths thereof are important determining factors, whether populations will grow in exponential or cyclical directions. Omran states that:

> The clearest indication of mortality's dominant role in population dynamics is implicit in theories of population cycles. The cyclic rises and falls in population size that have been observed in animal and pre–modern human populations reflect sequential phases of population growth and decline … these cyclic movements must ultimately be accounted for in terms of the range of variation in fertility and mortality (p, 733).

In Omran's conception, the patterns observed in human populations in pre–historic times paralleled patterns identified in animal populations in pre–

historic times. Mortality influences on population growth depicted cyclic movements, which were implicitly related to theories of population cycles. Omran further contended that even though pre–modern data on fertility and mortality are scanty, observed variations in fertility and mortality do allow probabilistic statements to be made about pre–historic times.

The second proposition argues that in the course of the transition, pandemics of infectious diseases are gradually displaced by degenerative and man–made diseases as primary cause of death. This stage depicts three distinct but successive stages. The first is the age of pestilence and famine. This era shows high and fluctuating mortality, thereby precluding sustained population growth. In this stage, the average life expectancy at birth is low and wavers between twenty and forty years. The second, the age of receding pandemics, occurs when mortality declines progressively and the rate of decline accelerates as epidemic peaks become less frequent or disappear. Average life expectancy at birth increases steadily from about 30 to about 50 years and population growth begins to portray an exponential curve. The age of degenerative and man–made diseases, his final stage, is when mortality continues to decline and eventually approaches stability at a relatively low level. The average life expectancy at birth rises gradually until it exceeds 50 years. It is during this stage that fertility becomes a crucial factor in population growth.

Omran (1971) argued that, in the second stage, deaths are caused predominantly by the Malthusian "positive checks": epidemics, famines and wars. Determinants of mortality during this stage are eco–biologic, socioeconomic as well as public health interventions. An evidence of the

ecobiologic variable is the introduction of vaccines. Variolation against smallpox, practiced in China as early as the 10th Century was an early form of vaccination whereby matter from the scabs of previous victims was introduced into the bodies of healthy people to provide anti–bodies. Jenner introduced vaccination at the end of the 18th century, but wide scale research on vaccines depended on the germ theory of disease and did not occur until a century later (Centres for Disease Control, CDC, 1999). According to Centres for Disease Control and Prevention [CDC] (1999), tuberculosis and yellow fever vaccines introduced in 1927 and 1964 respectively were influential in reducing these two diseases. CDC (1999) mentions some milestone vaccines since late 19th century as including those for rabies (1885), plague (1897), diphtheria (1923), pertussis (1926), tuberculosis (1927), tetanus (1927), yellow fever (1935), polio (1955 and 1962), measles (1964), mumps (1967), rubella (1970), and hepatitis B (1981).

The mortality consequences of these diseases were high, but the best available historical data suggest that, in the now–rich countries, immediately prior to introduction of vaccines, direct mortalities from these diseases were relatively rare, except for tuberculosis. The greatest effect of vaccines on health was felt more on polio. In any case, elimination of these infections had indirect results on mortality as their decline made people more resistant to other diseases (Cutler & Miller, 2005). Meanwhile, the extent of these influences remains unknown. Figure 1 is a diagram of the theory based on proposition two.

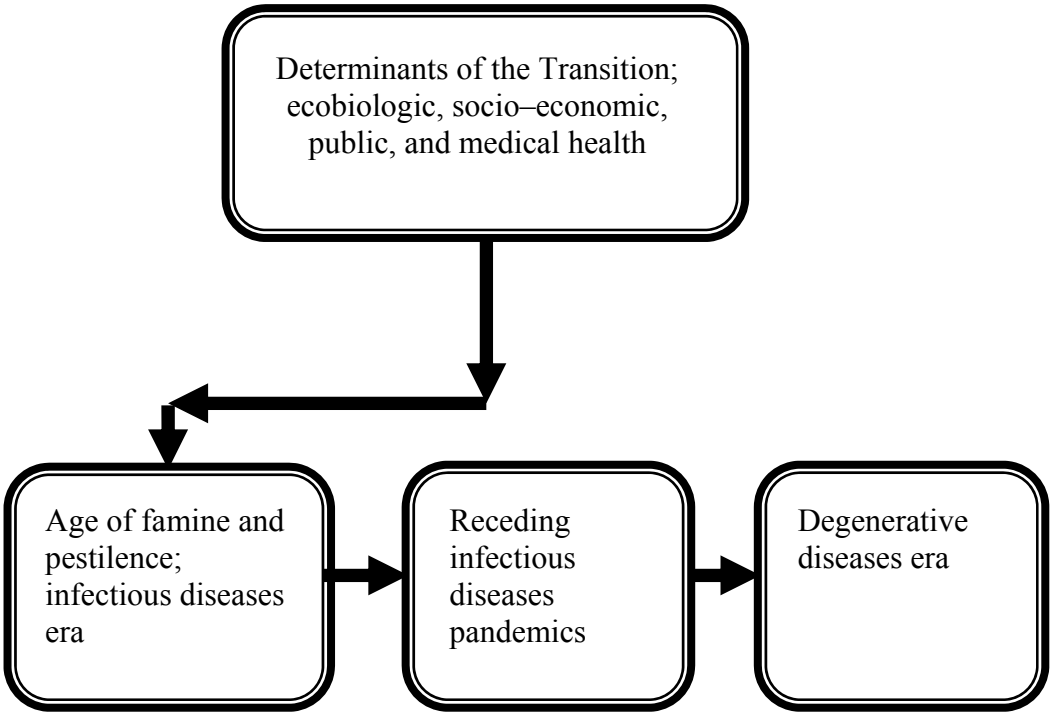

Figure 1: Conceptual stages of the epidemiological transition theory

Source: Omran, 1971

Some socio–economic factors that are recognised as playing important roles include education, nutrition and urbanisation. As regards education, social epidemiologists such as Davey–Smith et al., (1998) have indexed some factors accounting for this relationship. First, educated people acquire knowledge on health damaging behaviours. Second, the ability to optimise health services increases with education. Third, willingness to invest in human capital increases with rise in education. Again, education gives more access to benefit from new information and, finally, it promotes development of time preferences favourable to health maintenance. These advantages are believed to work through some proximate variables such as less smoking and drinking, living in clean environments, and several others.

Arguably, the nutritional hypothesis is the most controversial. The eighteenth century brought in its wake significant improvements in

agricultural yields. It is a basic scientific fact that, better–fed people resist most bacterial diseases, and recover more rapidly. McKeown (1976), a pioneer in population health research argued for the influence of nutrition in improved health. McKeown argued that: neither personal health care nor public health appeared to have had much impact prior to the 1900s. Using tuberculosis mortality data through the application of factorial analysis, he demonstrated declines in tuberculosis mortality as the single most important factor in mortality decline and went on to state that, about 80 of this fall took place before antibiotics were discovered. The same is true of other infectious diseases as well. However, some analysts such as Szreter (1988) disagree, especially regarding McKeown's neglect of public health.

Urbanisation too is another factor that was important during the second stage of the transition theory. At the initial stages of health development, urbanization was at least not favourable to morbidity change and mortality decline. The bulk of available data quite clearly indicate that mortality improvement was tarried by unsanitary conditions in sprouting cities between 1820s and 1870s (Cutler, Deaton & Lleras–Muney, 2006). These empirical studies have proven the link between urbanisation and mortality, for instance, in the Netherlands. Differing levels of life expectancy in the Netherlands have been more favourable in rural areas than in the most urbanised areas of the country, with mortality in urban areas being particularly high with respect to some cancers, arterial diseases and respiratory diseases, and other diseases linked to behaviour (Mackenbach & Kunst, 1994). Probably, this may be due to the less densely nature of human settlement, resulting in reduced rate of contamination (water, food), more ventilation, and less pollution associated

with industrialization. On the other side, availability of, and accessibility to various forms of public health services in urban areas is not in much doubt.

Cutler (2004), Cutler, Deaton and Lleras–Muney (2006) and Akachi and Canning (2008) give much recognition to public health at the beginning of the process and medicine, later in the transition. The public health argument is discussed under two sub–levels; micro and macro. Macro public health involves large–scale public work projects: filtering and chlorinating water supplies, building sanitation systems, draining swamps, pasteurizing milk and undertaking mass vaccination campaigns. Micro public health are changes made by individuals but encouraged by the public sector, including boiling bottles and milk, protecting food from insects, washing hands, ventilating rooms and keeping children's vaccinations up to date. Macro public health was always present to some extent. Bubonic plague victims are on record to have been quarantined in high–risk areas. Benjamin Latrobe built a water system in Philadelphia early in the 19th century, at least partly to reduce the disease burden (Cutler, Deaton & Lleras–Muney, 2006).

Fairchild and Oppenheimer (1998) adds that, 18^{th} and 19^{th} Century tuberculosis fatalities were contained to some extent by infirmaries and screening (public health intervention) programmes leading to reduction in fatalities. Akachi and Canning (2008) used the economic situation in sub–Saharan Africa to argue that, but for public health and medical interventions, infant and child mortality would have been higher in the sub–region than the current rate of 94/1000 live births (WHO, 2008). Binka, Ngom, Phillips, Adazu and Macleod (1999) found 17 percent reduction (γ=0.83) in all–cause mortality in children between 6 months and 4 years after an introduction of

permethrin impregnated bed nets in the Kassena–Nankana area (medical–public health process).

Omran (1971) concluded on the proposition that "medical and public health factors came into play late in the Western transition, but had an influence early in the accelerated and contemporary transitions" (p.741). Achievements made in the transition regarding the second proposition were caused more by eco–biologic and socio–economic variables. Transitions in African and other developing countries have been predicted to be necessitated more by medical and public health due to failing economic conditions (See for instance, Razzell & Spence, 2006). In as much as the evidence on this subject is intricate, it may be safe to assume that transitions in developed countries were predominantly influenced by socioeconomic factors and transition in developing countries would be propelled by medical and public health.

Omran (1971) also identified the relative risks by age and sex in the epidemiologic transition. His observation was that, the transition favoured children and women's health. For instance, there appeared to be substantial improvement in the health of women in the reproductive age, probably due to elimination of their vulnerability to infectious diseases. There was also improved survival among children, primarily due to improved sanitation, nutrition and standard of living (Omran, 1971; Razzell, 1974; Szreter, 1988).

In considering sex as a relative risk factor during the transition, Omran drew attention to the fact that:

> Females' risk of dying is less than that for males in the post–reproductive period at all life expectancy levels, but females have a higher probability of death during the adolescent and

> reproductive age intervals at low life expectancy levels. During the transition from infectious to degenerative disease predominance, women switch from a level of mortality in the reproductive years higher than that of men to a level more advantageous, such that the female's higher relative risk of death disappears at about the level of 50 years life expectancy and becomes lower than that of males thereafter (p. 742).

Historical and present data provides an insight into contributions that children and women's health play in reducing avoidable deaths.

According to Allotey and Gyapong (2005), even though "women and men may have similar rates of infection and mortality; there are often differences in their experience of the disease. This is relevant to the outcome, whether disability or death from the infection" (p.12). This argument is supported by empirical evidence. A study by Whiteford (1997) referred to in Allotey and Gyapong (2005) observes differential experiences of men and women in dengue fever. Women were found to be at a higher risk of being infected because of their higher participation in water storage, laundry and cleaning. Chagas disease or American trypanosomiasis, sometimes described as poverty–related disease, affects women and children more than men (Azogue, 1993 quoted in Allotey & Gyapong, 2005). As a result, a country's ability to reduce social, ecological and economic susceptibilities is critical in the transition process. Premised on this experience, countries that desire to hasten the completion of the transition cannot underrate the health of women as well as the health of children.

Omran (1971) envisaged that, the three preceding propositions are not accidental. They interact with other modifying variables to complete the transition. That is: "the shifts in health and disease patterns that characterize the epidemiologic transition are closely associated with the demographic and socioeconomic transitions that constitute the modernisation complex" (p 743). This decline leads to population growth because mortality reductions that accompany the demographic transition expand the "demographic gap".

The interactions between the epidemiologic and socio–economic transitions are no less complex. The epidemiologic transition is influenced, chiefly by socioeconomic improvements (as in now developed countries) as well as modern public health programmes (the situation in currently developing countries). Recognising disparities in determinants of ill health and death, Omran (1971) distinguished among the diversities in patterns, paces, determinants and consequences of change. According to him, these culminate into three distinct models: classical/western, accelerated and contemporary models.

The Western model depicts steady but consistent move from high death rates (above 30/1,000) to low mortality of about 10 per 1,000. This was concurrent to modernisation in Western European countries. These incidents were immediate to pestilence and famine era, which was characteristic of pre–modern and early periods. This slow pace gave way to fast declining pace at the turn of the early 20th Century. The unstable trends in mortality of pestilence and famine and receding pandemics ages were comparable to the classical model, with only time span being the main distinguishing feature. Thus, the accelerated model took less time to complete. This model is typical

of countries like Japan and Chile. The contemporary is still ongoing and is characteristic of most developing countries, especially. Yet, the rate of decline was unsteady in some countries that have gone through this model. Achievements in the last two models are, particularly ascribed to improvements in public and medical care that were rigorous after the World War II (Omran, 1971; Razzell, 1974).

In spite of the ascribed model to Africa, there are some doubts as to whether a completion of the model is possible, especially, with the emergence of HIV/AIDS. Kuate–Defo (2002) who holds a pessimists view on the subject offers three reasons why completion is unlikely. First, the slow pace of economic development is seen as posing a great hindrance to achieving an era dominated by chronic degenerative diseases in older ages. This is because most of the countries have not yet recovered from their economic shocks in late 1970s to early 1990s. Economic recovery programmes such as Structural Adjustment Programme (SAP) could not yield the expected benefits. The direction of health policies are more of vertical than broad base approach. There appears to be a relatively high substitution effects in morbidity and mortality since many are prevented from dying of "immunisable" diseases such as measles but only to suffer and die of other competing causes such as "non–immunisable" conditions like malaria or pneumonia. Again, there is a concern about the potential effects of drug resistance to conditions like malaria and tuberculosis, two common diseases in Africa.

In the course of the mid–1980s, researchers found out that contrary to Omran's (1971) proposition, mortality declines have no specific end. Omran (1971) predicted seventy–five years (75) as a point of convergence of life

expectancy for all countries. Later studies disproved this convergence criterion. Olshansky and Ault (1986) put forth a fourth stage, "age of delayed chronic diseases" which included rapidly declining mortality, mostly in advanced age and progressive shifts towards chronic diseases.

Rogers and Hackenberg (1987) agreed with Olshansky and Ault (1986) for a fourth stage dominated by chronic diseases but also added violent deaths (accidents, suicides and homicides) which were overlooked in Omran's thesis. Rogers and Hackenberg (1987) described this stage as a hybristic stage – a time when people feel invincible, man–made diseases are created as a result and are therefore unlikely to be cautious of life–threatening dangers, like drunk– driving.

Omran (1998) revised his 1971 model and argued for two possible additional stages to the initial model. In the revised model, Omran (1998) predicted life expectancy to rise until it reaches 80 to 85 years and then stabilised. Chronic diseases retreat as main causes of death, with the emergence of new diseases (HIV, Hepatitis B and C, Ebola, Hantaan virus) as well as re–emergence of old diseases (cholera, dengue, TB and flu–like infections). However, some researchers in the 21st Century have argued against setting limits to how long people can live (Vaupel, 2001; Carey & Judge, 2001). This is plausible as tracking of life expectancy in the developed countries since late 18th Century reveal consistent and progressive improvements, making such predictions doubtful.

Commenting on Omran's epidemiological transition theory, Mackenbach (1994) identified some challenges. Firstly, historical records are insightful on the contributions of specific diseases. Dating the beginning of the

transition to mid 19th Century cholera was, for instance, a pandemic disease. Others such as pneumonia and tuberculosis were endemic. Therefore, in delineating the theory from the broader demographic transition, cause–specific mortality fraction should not be overlooked. This will bring to bear the differential role of various diseases. Secondly, the designation of a stage in the second proposition as degenerative, corresponding to diseases in old ages became untenable few years after the labelling. For example, diseases like cancer, diabetes and hypertension, which were implicit in the degenerative designation, have been observed in younger populations as well. Again, the use of "man–made" diseases in the second stage of the theory attaches moral stigma to people who experience conditions in this category. Thus, victims of the so–called man–made diseases are likely to be stigmatised. Therefore, current thinking in population health prefers chronic to degenerative.

According to Meslé and Vallin (2004), the epidemiological transition comprises two stages and perhaps, a third. They acknowledged Omran's (1971) first stage during which the improvement in survival is due to the decline of infectious diseases and to the rise of chronic diseases. They contended that the second and last stages took place when the increase in life expectancy began to be sustained almost entirely by decline of cardiovascular diseases. This stage was termed as the cardiovascular revolution with life expectancy being pushed back endlessly. Meslé and Vallin (2004) have observed the possibility of some countries experiencing two stages concurrently, a situation that the WHO had described as a double burden of disease (WHO, 2000).

Some authors have cautioned against claims of unidirectional and uninterrupted progression, but instead, counter–transitions, which indicate re–emergence of infectious and parasitic diseases not necessarily leading to a fifth stage as suggested by some observers (Smallman–Raynor & Philips, 1999; Wolleswinkle–Van Den Bosch, Looman, Van Poppel, & Mackbenbach, 1997). For instance, Vallin (2005) criticises the theory as both too broad and narrow. It is too broad because the theory assumes universal application. That is, the assertion that all countries go through the same path of epidemiologic profile changes. In addition, it is narrow because, largely, the theory seems to argue that advances in health are more pronounced when infectious diseases decline. However, Vallin's description of the theory as too narrow needs further consideration. An examination of countries which have made significant gains in life expectancy and quality of life shows that they were successful due to sharp declines in infectious diseases which accounted for avoidable mortality.

In an earlier discourse, Kunitz (1991) argued in favour of generalising the theory but warned against overgeneralization and universalisation of results based on a US data. To Gaylin and Kates (1997), generalisation of the theory may only be limited by large differences in mortality trends in sub–populations. It is, therefore, important to particularize the theory to specific populations. As pointed out by Caldwell (2001b) "in truth, there are probably as many models as there are societies" (p.160). Similarly, Mensah (2008) citing Leeder, Raymond, Greenberg, Liu and Esson (2004) and Gaziano (2005) comments on the debate along the lines of Caldwell as:

> … countries and regions experience different stages of the transition at different times, and the speed with which they pass from one stage to the next varies widely, … within a particular country, various subgroups may undergo this transition at different rates depending on the speed at which economic development and industrialization occur and on the magnitude of changes in lifestyles and health behaviours (p. 837).

Inferring from the preceding discussions, two main limitations are adduced. First is Omran's assumption of unidirectionality that all societies go through the same stages even though some complete before others. Two, current improvements in life expectancy at birth has not followed Omran's (1971; 1998) predictions of maximum life expectancy of seventy years and the later limit of 80–85 years. Countries such as Japan have added twelve more years to the years predicted by Omran. Already, female life expectancy in Japan has crossed the 85 limit and France and Sweden will soon cross it (Vallin, 2007). In spite of this, Vallin (2007) says even though Omran was wrong in setting age limit, at least he did better than most demographers of the era who could not envisage the impending cardiovascular revolution. Noticeably:

> The main mistake of the Omran's epidemiologic transition theory related to it having led us to believe that any population, when modernizing, will go straightforwardly to that third age, without any interruption. Reality is more complex. The main shortcoming of the theory is to underestimate both the dynamics of infectious diseases, as if no new diseases could

> appear, and the link between the capacity of each population to improve its health status with its success in improving its economic, social, cultural and political capacities (Vallin, 2007: 2).

Vallin therefore proffers that the broader concept of Frenk, Bobadilla, Stern, Frejka and Loanza (1991) of health transition is more robust since it has the potential of capturing succeeding epidemiological transitions. In this sense, Omran's theory will only be one of the numerous "epidemiologic transitions".

Understandably, the existence of many models as there are societies helps us to arrive at equilibrium or a compromise between over– and under–generalizations. In spite of the several limitations that have arisen from the theory:

> The epidemiologic transition theory provides potentially powerful framework for the study of disease and mortality in populations, especially, for the study of historical and international variations. Although its primary purpose was to describe and explain the spectacular fall in mortality which has occurred in all currently industrialised countries, it can also be used to speculate on the likely consequences of future changes in mortality in countries which are lagging behind those which have already completed the epidemiologic transition: will a fall in infectious disease mortality in currently developing countries lead to a rise in chronic diseases and accidents (Mackenbach, 1994: p.330–331)?

Framework for the study

This study adopts Omran's (1971) epidemiological transition theory that has been discussed in the preceding section. With its five propositions and three stages (age of pestilence and famines, receding pandemics and the age of degenerative and man–made diseases), the model depicts a link between social, demographic and economic aspects on one hand and diseases and deaths on the other. The main theme of the theory is to identify stages of health development. This theory has been chosen over the others reviewed because: first, the theory contextualises diseases patterns in relation to space.

Another justification for the choice of the theory is its perceived semblance with the 2000 Global Burden of Disease (GBD). Omran (1971) grouped diseases experiences in the second assumption into pestilence, receding pandemics and degenerative diseases and Olshansky and Ault's (1986) addition of injuries. Based on current thinking, these have been indirectly refined into (a) communicable, maternal, pre–natal and nutritional deficiencies (Group I), (b) non–communicable (Group II) and (c) injuries (Group III). Besides its parallel with current evidence, the theory gives room for, first, speculative and a later empirical search for the determinants thereof. It also combines the stage as well as the determinants of health in communities. In addition, the model synchronises demographic and epidemiologic thinking in one framework. It is demographic because of the attention given to mortality rates and life expectancy and it is epidemiologic due the theory's emphasis on diseases that are concurrent with the mortality patterns. These potencies of the theory make it the most preferred for this thesis.

CHAPTER THREE

RESEARCH METHODOLOGY

Introduction

Using historical data in social science research has its own dynamics. The technique involves compiling and interrogating data which have already being collected. Such designs present challenges of translation and interpretation. One of such data sets is verbal autopsy which is derived from recollection of past events relating to deaths. Nonetheless, the approach provides another perspective for dealing with challenges of data availability on mortality in developing countries. This chapter deals with description of the study setting, research design and issues about demographic surveillance systems (DSS).

Setting of Kassena–Nankana District (KND)

The Navrongo DSS site is in the Kassena–Nankana District of the Upper East region of Ghana. The district lies between latitudes 10°30' and 11°00'N and longitudes 1°00' and 1°30'W and covers an area of 1675 km^2 along the Ghana–Burkina Faso border (Figure 2). It measures roughly 55 km by 50 km and has an altitude of 200–400m above sea level (Nyarko, Wontuo, Nazzar et al., 2002).

Located in the Guinea savannah belt, the district is typically Sahelian (hot and dry), with the vegetation consisting mostly of semi–arid grassland interspersed with short trees. The region has two main seasons, wet and dry. The wet season is from April to October, with the heaviest rainfall mainly occurring between June and October. The mean annual rainfall is 1365 mm, but the highest level is recorded in August. The dry season consists of the harmattan period (November–mid–February) and the dry hot season (mid–February–April). Monthly temperatures range from 20° to 40°C, with the mean minimum and maximum temperatures estimated at 22.8° and 34.4°C, respectively, for 1999 (Nyarko et al., 2002).

The population of Kassena–Nankana on 1 July 1999 was 136,955, which was slightly less than 1 percent of Ghana's population and about 15 percent of the total population of the Upper East region. The population in 2000 was 149,491 and the density was 84/km^2 as at 2000. The district is largely rural, with only 10 percent living in an urban setting (Navrongo township). The population comprises two distinct ethno–linguistic groups: the Kassena (49%) and the Nankani (46%). The Builsa and migrants belonging to other ethnic groups constitute the remainder (5%). The main languages spoken are Kassim and Nankam, with Builli spoken by most of the minority tribes. Despite the linguistic distinction, the population is in many respects a homogeneous group, with a common culture. The district has 10 paramount chiefdoms and has traditional forms of village organisation, leadership, and governance.

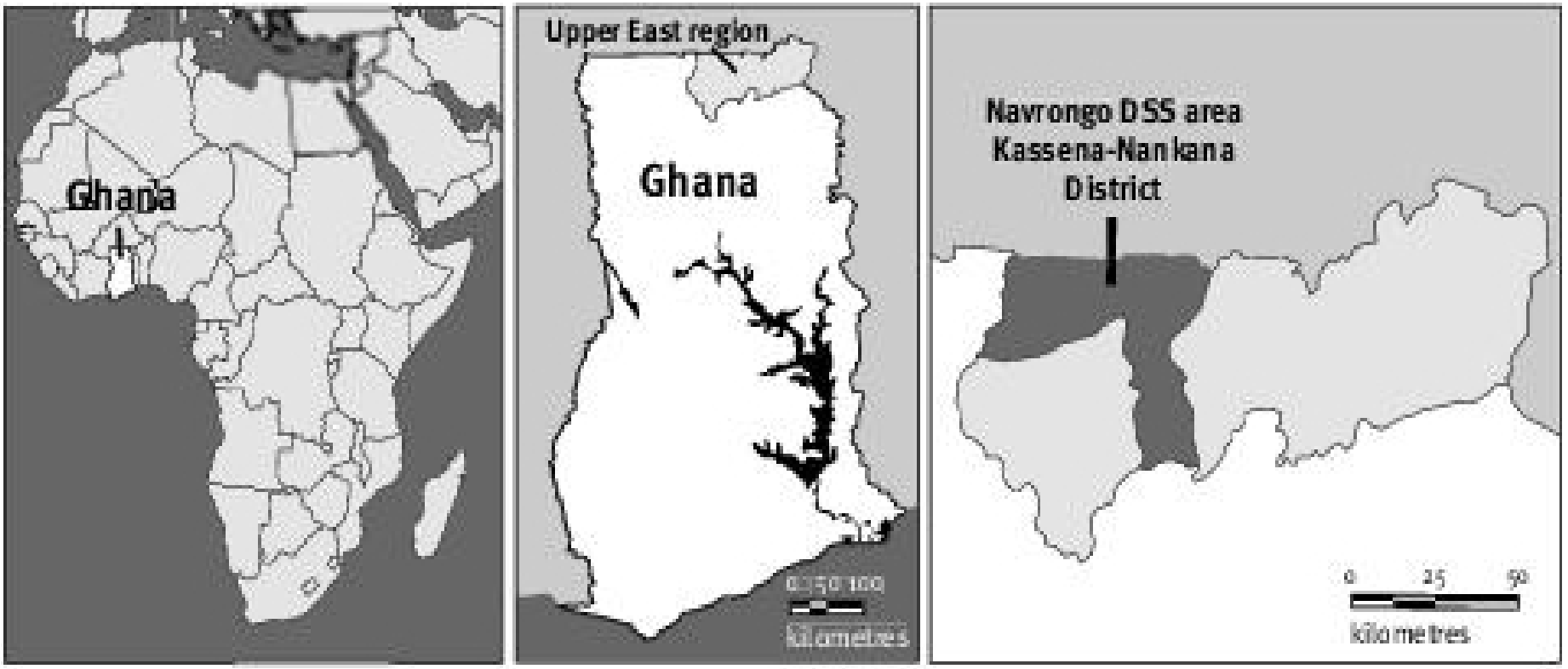

Figure 2: Study area

Source: Adopted from Nyarko et al., 2002

At both the village level and the family level, communities have a strong traditional social structure, which influences economic and social behaviour. Male dominance is strong, restraining the autonomy of women and limiting their health decisions. For example, curative and preventive health care may not be sought without the permission of the husband or, in his absence, the head of the compound (Binka, Morris, Ross, Arthur & Arteetey, 1994). Currently, about 33 percent of the people are Christians, 5 percent are Muslims, and the rest profess indigenous religion. However, the dominant traditional faith guides daily life, economic decisions, health beliefs, and practices including reliance on indigenous medicine (Debpuur, et al., 2002). In general, activities and communication systems such as road network, treated water and electricity in the district are minimal. Subsistence agriculture is the mainstay of the district's economy and this is complemented, to some extent, by retail trading. About 90 percent of the people are farmers. The major agricultural products are groundnuts, millet, guinea corn, rice, sorghum, sweet potatoes, beans, and tomatoes. Rearing of cattle, goats, sheep, pigs, and fowl, including guinea fowl, also forms part of the agricultural activities (Binka et al., 1994).

The district has 77 primary schools, 35 junior high schools, five senior high schools, one training college, and two vocational institutions. The district currently accommodates the Faculty of Applied Sciences of the University for Development Studies (Nyarko et al., 2002). About 89 percent of the houses in the district are mud huts with thatched roofs. The rest, which are built with cement blocks, are mostly found in the urban area. Almost two–thirds (65%) of the roofs are constructed with straw. Zinc sheets are used for the remaining

35 percent. The main sources of water in Kassena–Nankana are streams, wells, and boreholes. In a few urban houses, however, pipelines have been installed to provide treated water. Only 7 percent of the compounds have access to properly constructed toilet facilities. For those compounds with toilet facilities, two–thirds use either pan, or pit latrines or Kumasi ventilated improved pit (KVIP) and the rest use water closets (Binka et al., 1994).

The health facilities in the district are a hospital, four health centres, and four clinics. These static health–delivery points are complemented by community–based service delivery in all but the eastern part of the district, which serves as an experimental control cell through the demographic surveillance system. According to records from the district, predominant health challenges are malaria, gastroenteritis, and acute respiratory infections. Cerebrospinal meningitis is relatively common, with the peak season being March to April. Although improved delivery of family–planning services is one of the objectives of the Navrongo community–health and family–planning (CHFP) project, only 10 percent of married women in the district use such services (Nyarko et al., 2002).

Study design

Discussion of study design in epidemiology normally follows cause–effect paradigms. The cause–effect relationship can be deterministic or probabilistic. In a deterministic relationship, statement of "fact" can be made about a phenomenon being studied. Probabilistic, on the other hand indicates that a relationship could exists between the cause and the effect. The main preoccupation of epidemiological research is to provide evidence for

programme purposes (Canato, 2004). Epidemiological research designs are based on a number of observations in a study, directionality of exposure, data collection methods, timing of data collection, unit of observations and availability of subjects for study (Friis & Seller, 2004).

The main types of design in epidemiology are experimental, quasi–experimental and observational studies. Experimental are those studies that a researcher manipulates study subjects and thereby consigns factors to exposed and non–exposed groups. Examples of experimental are clinical trial (prophylactic and therapeutic) and intervention studies (controlled clinical and community interventions). Quasi–experimental involves indirect manipulation of study subjects. In quasi–experiment, participants or objects of study are not randomised. It is often described as natural experiment. Quasi–experimental studies are normally applied in evaluating health programmes after a distal factor has been introduced in a population (Friis & Seller, 2004).

Observational studies on the other hand do not involve manipulation or randomisation of study participants. Observational studies extensively rely on the use of measurement patterns of exposure on health outcomes (diseases and deaths). These analyses aid postulations about diseases aetiology for further studies through experimental or quasi–experimental designs (Friis & Sellers, 2004). Observational studies are either analytic or descriptive.

Analytic studies are concerned with the determinants of diseases and the reasons for relatively low or high frequency of disease in specific populations or their sub–groups. They are conducted to identify actual determinants of diseases. However, descriptive studies attempt to identify and characterise the amount and distribution of diseases (Friis & Sellers, 2004).

Some common types of observational descriptive models are case reports, case series and surveillance. Traditionally, descriptive studies focus on the epidemiologic triad: person, place and time that translates into answering the five basic "W" questions; who, what, where, when and sometimes, why certain observations. The "why" questions and answers provided in descriptive studies are more of probabilities or speculations and these speculations and probabilities become limitations of descriptive researches (Grimes & Schulz, 2002).

One approach is the use of longitudinal time–series data, which is information about a research unit across multiple times. Cnossen (1997) defines existing data as one collected by someone or an institution other than the current user or information collected for different purposes. It provides a cost–effective means of utilizing information available on a population.

This study used descriptive population level surveillance data. Surveillance, according to CDC (1986), and alluded to in Grimes and Schulz (2002, p.146), is the on–going systematic collection, analysis, and interpretation of health data essential for planning, implementation, and evaluation of public health practice, closely integrated with the timely dissemination of these data to those who need to know. Grimes and Schulz (2002) summarised this as monitoring of health events in a community.

There are two main categories of surveillance: passive and active. Passive surveillance is obtained through institutional systems such as death registers and active surveillance involves a continuous search for cases (Grimes & Schulz, 2002). Active surveillance systems usually have regular

field enumerators of health incidents, and this is what pertains at the Navrongo Demographic Surveillance System (NDSS).

The unit of analysis was at the population level and this was meant to situate the study at the broader societal level. Public health debates now tend to combine individual (proximate) to contextual factors. According to Susser and Susser (1996a, 1996b) public health epidemiology has already experienced three paradigm shifts. These paradigms are sanitary statistics (miasma theory, first half of 19th Century), infectious disease (germ theory, late 19th Century through first half of 20th century) and chronic disease epidemiology, which constituted black boxes in the latter part of the 20th Century. The current and the uncompleted era is eco–epidemiology. This era does analysis at different levels of communities with central focus on group level factors to design contextual interventions. This level helps to avoid victim–blaming public health policies and instead addresses contextual risk variables.

Background of the Navrongo Demographic Surveillance System (NDSS)

The NDSS evolved from an earlier study of Kassena–Nankana in 1989 by the Department of Community Health of the Kwame Nkrumah University of Science and Technology and the London School of Hygiene and Tropical Medicine, with support from the Ghana Ministry of Health and the UK Overseas Development Administration (now Department for International Development, DfID). The study, known as the Ghana vitamin–A supplementation trial (VAST), involved continuous demographic and health surveys of resident members of the study compounds with the aim of helping

to evaluate the effect of vitamin–A supplementation on children less than 5 years old. When VAST ended in 1992, Navrongo Health Research Centre (NHRC) was established to examine other health problems in the country and find practical solutions to them. The NHRC, thus, used and built on the VAST resources established in 1989. In 1993, the demographic surveillance system (DSS) was reorganised with respect to its coverage and content and formally set up as the NDSS to serve as a basis for assessing effects of insecticide–treated bed nets on mortality. The bed net study was concurrent with quasi–experimental designs on family planning and mortality in a project known as Community Health and Family Planning (CHFP). The NDSS started with a baseline census of the rural areas in the district in 1993. In the last quarter of 1995, activities were extended to Navrongo, the only urban area in the district. To qualify as a member of a compound, a person should have been resident in the compound for at least 3 months, except for a newborn baby whose mother is already a compound member. The initial DSS covered about 125 000 people, but with the addition of urban Navrongo, the population increased to almost 141 000 in 1995.

The baseline survey consisted of demographic background information, socioeconomic characteristics, compound possessions and the materials used in constructing buildings, vital events – births, deaths and marriages – and migration. This initial census was followed by compound visits at 90–day cycles to monitor demographic events. From 1993–2005 the 90–day cycle was used; from 2006–2008 the 120–day cycle was used. In 2009, the 180–day cycle was used. Since July 1993, the NDSS has become a longitudinal household–registration system (HRS) to support research on the

risk factors of health and fertility in an area typical of Ghana's rural savannah zone. The system routinely updates vital events (births, deaths, migration, marriages, and pregnancies) in almost all the compounds within the study area. When a death occurred in a compound, the compound was revisited to obtain information on the circumstances leading to the death. These verbal autopsies were and are conducted using different schedules for children and adults. In addition to the vital events, educational attainment and vaccination coverage within the population are annually monitored.

The field and data–processing operations of the NDSS are managed by a team comprising a demographer, two research assistants, two principal field supervisors, a data manager, and a data assistant. The team coordinates the activities of 26 fieldworkers and 12 field supervisors, who are responsible for field data collection, as well as the two filing clerks and three data–entry clerks who receive and process the field instruments. The fieldworkers are expected to visit and interview all compounds within their work area. The 12 field supervisors are responsible for conducting verbal autopsies (VAs) interviews, carrying out quality checks and resolving queries.

The field data collection and processing are mainly supported by funds from the Rockefeller Foundation, with technical assistance from the Population Council. Reports are compiled for the Ghana Ministry of Health/Ghana Health Services, which is the major consumer of the NDSS data. Lessons learned from the Navrongo CHFP project and the panel surveys have, for example, activated a process of extending the new health–delivery approach implemented in this district to the country as a whole, that is, Community Health Planning Services (CHPS). Other institutions that have and

continue to benefit from the Navrongo DSS database are the universities and other educational and research institutions (Nyarko et al., 2002).

Navrongo Demographic Surveillance System field procedures

For data collection purposes, Kassena–Nankana is divided into five zones: north, central, east, west and south (Figure 3). These are further subdivided into 21 sub–zones and 244 clusters. The clusters were not based on any scientific principles. However, the zones were based on geographic information systems. On average, nine contiguous clusters are assigned to each of the 26 fieldworkers to enhance fieldwork and reduce costs.

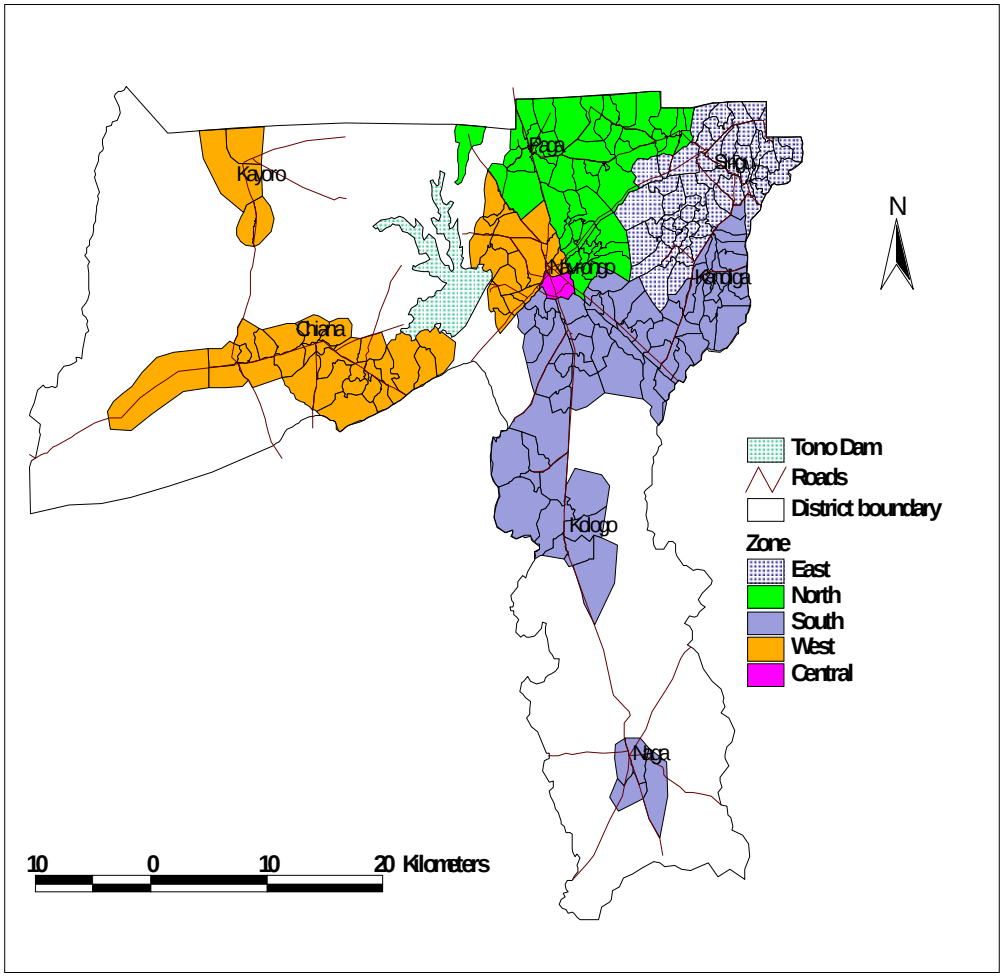

Figure 3: Kassena–Nankana District showing the zones
Source: NDSS data unit, 2009

To track the population, each fieldworker is expected to visit, and update demographic information on 15 compounds every day. The main data–collection instruments used for the routine recording and updating of vital events are compound–registration books (CRBs) and event forms. CRBs are field registers containing basic demographic information on all compounds in a cluster. Where a cluster has more than 99 compounds, an additional CRB is used. An event form is also filled out for each recorded event (Nyarko et al., 2002).

All vital demographic events occurring within the district are updated through regular visits to compounds. During these compound visits, new events are registered. Pregnancies recorded earlier are also monitored during these quarterly visits until the pregnancy outcome is observed. This is to help improve on birth and death reporting, particularly by capturing neonatal deaths. For every vital event that is recorded, detailed information is collected using the appropriate event–registration form. Apart from the event updates, the first quarter of each year is devoted to updating information on the educational attainment of those aged 6 years or more and the last quarter of the year is used to collect data on the vaccination status of children younger than 2 years (Nyarko et al., 2002).

Vital demographic events, including in– and out–migration, marriages, pregnancies, births, and deaths, are continuously monitored through quarterly updates. In addition to the routine collection of data by fieldworkers, the NDSS has recruited a number of voluntary community key informants (CKIs) to record all pregnancies, births, and child deaths that occur in their localities during the intervals between interviewer visits to compounds. Currently, 170

CKIs work within the district. Two field supervisors are assigned to visit the CKIs in their homes every two weeks to collect the information that they have gathered over the period. These data supplement what the NDSS fieldworkers collect during their regular visits to the compounds every 90 days (Nyarko et al., 2002).

Quality assurance is achieved through a variety of ways. First, there is re–interviewing of a 3 percent randomly selected compounds. This is done by a quality control supervisor. The second is re–interviewing some of the compounds already covered by the same fieldworker and results compared. Random review of CRBs and event forms are also conducted for inconsistencies, omissions, and observation of field interviews. Procedures employed at the office level include the assessment of the work progress of field staff at weekly meetings and retraining of interviewers at the end of each round of data collection for one week (Nyarko et al., 2002).

The NDSS also has a mechanism for pairing internal migrants, to avoid double counting and to minimize loss to follow ups. This process of pairing migrants is aided by issuing identity cards to all compound members. The identity cards are meant to improve the reporting of event dates and facilitate the linking of migrants to their previous records. To avoid familiarity with the respondents and forestall any attempts to manipulate data, the field staffs work in the same clusters for two consecutive rounds and they are changed. Improvement in event capture is also achieved through the voluntary activities of CKIs, who for a token fee, record births, deaths, and pregnancies in their communities during the interval between visits (Nyarko et al., 2002).

Navrongo Demographic Surveillance System data management

Every fortnight, each fieldworker submits all completed CRBs and event forms to the filing clerks. These records are then carefully documented and sent to the data–entry clerks, who update the database, using the HRS data–entry system. A data manager, a data assistant, and three data–entry clerks carry out data–processing operations of the NDSS. Each of these personnel has a different level of access to operate the database. A successful entry into the system allows data to be added, edited, or deleted. When CRBs and event forms are returned to the computer centre at the weekly zonal meetings for fieldworkers, it takes one or two days to have them sorted and distributed to the data–entry clerks. Data entry and validation take about one week (Nyarko et al., 2002).

The HRS system has built–in validation programmes, which help to maintain consistency in the database. Computer operations are organised to correspond to the interviewing cycle so that information, which fails the HRS logical checks, is printed with the relevant error message for field reconciliation. On the other hand, records that pass the logical tests are archived into the database. Thus, each round generates fully edited and cleaned data before a new cycle begins. The updated information is used (Nyarko et al., 2002).

Data acquisition, reorganisation and analysis

Data for the study was collected on 11,194 people who died between 01–01–1995 and 31–12–1999. Information on causes of mortality was obtained from caregivers, relatives, or a close neighbour of the deceased

through verbal autopsy (VA). Before the data extraction, one familiarisation visit was undertaken from 1st to 4th December 2008. This was done to obtain information about data available at the site and the processes involved in releasing data sets to applicants. The second visit, which included data extraction, took place from 13th to 17th April 2009. Data agreement between NHRC and the researcher was signed in the process (Appendix 10). After all the protocols on DSS data extraction had been observed, data for the study was released on 16th April 2009. The data set given had the following variables: deceased's identification number, cluster of resident, dates of birth and death, causes of death, marital status, sex, educational background, occupation and mid–year population of each cluster.

The data were then reorganised to suit the objectives of the thesis. First, the clusters were collapsed into zones; north, east central, west and south. This was done because, according to NDSS data manager, the clusters were not based on sound geographic principles. Equally, the clusters were many (244), and would make distinguishing the cluster characteristics difficult. Since clusters had been based on the prefixes of the zones, it was appropriate to merge same prefixes into one distinct zone. The original set was in string format that could not have allowed some descriptive and inferential analysis, such as, cross–tabulation and regression. Using Statistical Product and Service Solution (SPSS) version 16, the data were recorded into numeric to facilitate advanced analysis.

Similarly, individual ages were calculated based on dates of birth and death. In the verbal autopsy approach, different interview forms are used for peri–natal and neonatal deaths (deaths under 4 weeks), post–neonatal (beyond

4 weeks to 14 years) and adult mortality (15+) (MEASURE Evaluation & US Census Bureau, 2007). Accordingly, ages were grouped into ≤0 (peri–natal and neonatal), 1–4 (under–five mortality), 5–14 (late childhood), 15–44, 45–59 and ≥60. Further data mining was undertaken to extract month of death for seasonal decomposition.

DSS sites usually collect cause–of–death information through verbal autopsy (VA). WHO (2005) defines verbal autopsy as:

> A technique used to determine the cause of death by asking caregivers, friends or family members about signs and symptoms exhibited by the deceased in the period before death … usually done using a standardized questionnaire that collects details on signs, symptoms, complaints and any medical history or events … to describe the causes of death at the community level or population level where no, or only limited, vital registration is completed with medical certificates (p. 56).

Verbal autopsy questionnaire has a section for open narrative by the respondent and formatted checklist for symptoms. There are also questions about the health and status of mothers (in the case of peri–natal, neonatal, and child deaths), and questions that specifically relate to all women fifteen years or older (for maternal mortality). Additional information is collected about previously diagnosed conditions, medications used, health services used, place of death, and behavioural and environmental risk factors. The data collected is then evaluated by, at least, three independent physicians or trained health workers to ascertain the cause of death (Adams & Boerma, 2006). If two or all

three agree on a cause, the disease is assigned. There are instances where disagreements persist and unknown is entered in the data file. In cases where the cause cannot be ascertained it is designated as undetermined.

Attribution of cause of death normally is under the concept of underlying cause of death that is defined as "the disease or injury that initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence that produced the fatal injury" (WHO, 2005, p. 56). However, some surveillance sites do not strictly employ the underlying cause concept. In specific diseases endemic areas, the immediate cause is chosen if the condition showed similar symptoms of the endemic disease. The internationally accepted criterion for cause assignment is, however, the underlying concept. In cases where more than one cause is identified, for reasons of coding and reporting, the most probable cause is selected. World Health Organisation has rules and standards for ascertaining the underlying cause. Assigning a cause does not depend on the reviewers' opinion but on the standards set. This is to ensure that data from a site becomes comparable to others. Yet, some DSS sites have their own cause of death coding criteria (Adams & Boerma, 2006).

Besides being used for validating causes of death, VA helps to identify infectious diseases outbreak, risk factors of certain diseases as well as effectiveness of public health interventions (Pacqué–Margolis, Pacqué, Dukuly, Boateng, & Taylor 1990; Telishevka, Chenet, & McKee, 2001; Andraghetti et al., 2003; cited in Soleman, Chandramohan & Shibuya, 2006). Data from DSS sites do not allow extrapolation to other wider areas because they are not representative. Different DSS sites have their own criteria of

classifying diseases different from International Classification of Diseases (Adams & Boerma, 2006). This renders comparisons with other sites ineffective. Verbal autopsy may suffer from biases of coders at sites where the sites have specific stable endemic diseases.

Another weakness of VA is that description of symptoms may be impaired in cases where the recall period between event and interview is either too long or short. Both too long and too short time lag for VA interview can affect accuracy of information given. Too long impairs a respondent's ability to provide accurate symptoms before death occurred. Too short too may cause emotional stress and this can influence willingness and ability to give coherent and consistent account of symptoms that preceded death. The time lag between death and VA interview on the average varies from four weeks to six months (Soleman, Chandramohan & Shibuya 2006).

An argument against the procedure is its demand for more health resources by asking for more than two expert reviews. This is because countries that use the method already have a backlog of health personnel. The response to this, however, is the invaluable price of quality health data for health services planning. Even so, a recent study by Joshi et al. (2009) revealed that even in cases of single physician review, later anonymous validations in some DSS sites have proven statistically no significant differences.

The NDSS recorded fifty–eight (58) VA causes of death, including unknown/undetermined. However, NDSS uses a classification different from the International Classification of Diseases, 10^{th} Version (ICD+10) codes for causes of death. Consequently, the causes were re–classified unto the ICD+10

in the following categories: infectious and parasitic diseases, maternal conditions, malignant neoplasm (cancers) genito–urinary diseases, neuro–psychiatric disorders (central nervous system disorder), cardiovascular, respiratory diseases, respiratory infections and childhood clusters. There were also digestive diseases, prenatal conditions, intentional and unintentional injuries and nutritional deficiencies. Others were all other communicable diseases, all other non–communicable diseases and undetermined/unknown.

In putting the data into the ICD+10 codes, causes of death defined as "unspecified and "specified" communicable and non–communicable diseases were added to all other communicable and non–communicable diseases categories respectively. In ICD+10 coding, "lead" and "modifier" terminologies are applied. Lead is a descriptive concept applied to the condition why a person sought care or the real pathological condition. Modifiers are the anatomical context or site which could be several, for instance, malaria, malignant neoplasm. Therefore, in the description of cause of death, if the interviewee states a condition that has no modifier, the condition is prefixed with "un" or "not" specified (MEASURE & US Census Bureau, 2007). In this data, the unspecified conditions were added to the "leads" of similar cases or modifiers. For instance, unspecified acute febrile was taken as acute febrile, which was later merged into infectious and parasitic disease component. All "other communicable" and "non–communicable" categories consist diseases that rarely caused deaths, such as lymphatic filariasis. In another case where AIDS and Pulmonary TB had caused some deaths, it was taken as AIDS because of the "underlying" cause

concept. However, when a cause was autopsied only as TB, it was considered as such.

After these mergers, the data were put into the 2000 Global Burden of Disease (GBD) classification of diseases. These categories are Communicable, Maternal, Prenatal and Nutritional Deficiencies (Group I), Non–communicable Diseases (Group II) and Injuries (Group III). Besides these three broad categories, undetermined/unknown diseases were added to the adopted GBD system because the GBD assumes that all deaths would be autopsied and registered in appropriate death registry. Presumably, this was done without considering data constraints in developing countries. But given that data for this study was acquired from a system which uses the VA approach, with its inability to diagnose all causes of death, adding unknown was considered fitting.

Data analysis plan employed mostly descriptive methods and to some extent, some inferential statistics. Descriptive statistics were used to describe the characteristics of various parameters that the study was interested in measuring. These parameters included place of residence, occupation, sex, age, educational level, months and seasons (quarters) and ICD+10 classifications of causes of death. With respect to the seasonal and monthly patterns, the analysis followed a similar one by Kynast–Wolf, Hammer, Müller, Kouyate´ and Becher (2005).

Cross–tabulation was extensively used since a significant portion of the study was comparative. A non–parametric statistical tool, Pearson Chi–Square was employed to test some perceived significances in the hypotheses. The use of categorical Chi–Square tests was based on an expectation of

insignificant relationships stated in the hypotheses. This non–parametric technique was used because the variables considered were categorical: sex, month of death and grouped ages and were also assumed to be independent of mortality incidences. According to Norussis (1990), cross tabulations can be used to statistically test whether two categorical variables are independent or dependent. Pearson Chi–squared values and associated probability values (ρ–values) were used to ascertain the statistical significance of relationships. Additionally, simple linear regression was used to determine the kind of relationship between age (ratio level) and causes of death.

Limitations of the study

A major limitation of the study was the unavailability of denominators to calculate rates for the various socio–demographic variables that could have allowed reading differences into frequency of deaths observed among the various background characteristics. However, rates based on age and sex has been calculated elsewhere with these data (See for instance, Nyarko et al., 2002; as well as life tables for the area by INDEPTH Network, 2004). In the same way, prevalence of disease by age, sex and zones could not be measured. Data for the study did not contain morbidity incidences components. This also made it impossible to compare whether mortality or morbidity had decreased during the health transition, as argued by Johansson (1991). The study could not establish causalities; test statistics results only indicate plausible relationships. This occurred because of the unavailability of "proximal" data to complement the "distal" factors. This made the study primarily descriptive.

CHAPTER FOUR

MORTALITY STRUCTURE OF KASSENA–NANKANA DISTRICT, 1995–1999

Introduction

Until recently, information on adult mortality in developing countries has been scanty. This is attributed mainly to a gap or inadequacy of data that would allow objective and empirical assessment of mortality counts. Studies had, hitherto, focused more on childhood and maternal mortality because data on maternal and child health were/are relatively available than adult mortality data. Unlike adult mortality, child and infant mortality issues have received more attention. This may be due to the specific mention of childhood mortality phenomenon in the millennium development goals and also the fact that about 11 million under–five children die every year of preventable diseases. The concentration in developing countries is due partly to the fact that, out of these 11 million deaths, 10 million occur in developing countries and one–third of these infant deaths are recorded in sub–Saharan Africa. Furthermore, about two–thirds of these deaths are caused by virtually preventable diseases like acute lower respiratory infection, malaria and diarrhoea (Hill & Amouzou, 2006).

The emergence of HIV/AIDS has, in part, led to a "revolution" of studies on adult mortality (Bradshaw & Timaeus, 2006). Secondly, data for general mortality studies are gradually becoming available with the coming of

DSS sites in developing countries, particularly, Asia and Africa. Some of these sites have intentionally given priority to adult health, such as the Adult Morbidity and Mortality Project (AMMP) in Tanzania. Others too have targeted collecting complete and reliable data on the urban poor population. An example is Nairobi Urban Health and Demographic Surveillance System (NUHDSS). Similarly, some have focused on relatively rural districts, collecting data on both adult and child mortality. The Navrongo Demographic Surveillance System provides such type of data.

Through verbal autopsy, both adult and child deaths have been captured of all mortality cases since 1993 in the Kassena–Nankana district. This chapter deals with mortality structure of the district from 1995–1999. Among the issues addressed in the chapter are socio–demographic attributes of the dead person, crude death rate for each year and spatial as well as temporal dimensions.

Background characteristics of the dead

In population health, one very core indicator of quality of life is the age at which people die. It promotes the search for health hazards connected to age. According to Martelin (1994) mortality is a health outcome, exhibited in biological, functional and mental status of a person. Whilst some diseases occur primarily due to biological degeneration, others are acquired through behavioural risk factors regardless of age. Others too are zoonotic, transferred from animals and birds to humans, such as H1N1 influenza and rabies. In such circumstances, marital status, educational level and occupation may not be important determining factors. Background characteristics of people who died

in the five years are presented in the subsequent paragraphs. Nonetheless, differential meanings among the various background issues are unable to be imputed into the discussion due to the lack of required bases to calculate rates that would have brought out differences clearly. Consequently, the background features are only described in proportions. These are marital status, educational attainment and occupation at death.

The evidence on marital status and mortality is mixed and inconclusive. A section of the literature posits that people in marital relationship share lower morbidity and mortality than people who are not in unions. Two assumptions are adduced in the literature. First, marriage contributes to social integrative functioning. Another assumption is that, marriage benefits men more than women (Trovato & Lauris, 1989). As shown in Table 1, 67 percent persons of the dead were unmarried. This is understandable since as much as 30.7 percent of death occurred among those aged between 0–14 years and are legally not supposed to be married. This was followed by widows (20.9%), married (6.6%) and divorced (5.4%). Apart from the 30.7 percent who were outside the marriage market, the differences among the other marital groups raise some curiosity. The proportional differences in mortality by marital status may be associated with psychosocial (stress), material circumstances (livelihood support resources) and health behaviour (preventive) factors. All things being equal, widows are more likely to experience high death proportions as the death of a spouse could be a source of psychosocial (Cassel, 1976) as well as financial stress. Iwashyna and Christakis (2003) revealed that consumption of quality health care in USA was

minimal for widows compared to other groups. These could be some of the possible reasons for more deaths of widows than married.

The relatively low mortality proportion found among the married has been the subject of many studies. Poppel and Joung (2001) found an increased advantage for married men and women in mortality rates. Similarly, Manzoli Villari, Pirone and Boccia (2007) found a greater relative risk of death among unmarried (never married, widowed, separated/divorced) adults than married adults did. The results seem to be consistent with available evidence.

Table 1: Background characteristics of the dead

Variable	Frequency	Percent
Marital status		
Divorced /separated	600	5.4
Married	740	6.6
Unmarried	7514	67.1
Widowed	2340	20.9
Education		
None/NA	4459	39.8
Primary	660	5.9
Secondary	6075	54.3
Occupation		
Infant/Child/None	4459	39.8
Farmer	4907	43.8
Gov't/Privt.	199	1.8
Student	1210	10.8
Trader	419	3.7

Total of each Variable = 11194.

Source: NDSS Data Unit, 2009

From 1995–1999, out of the 11,194 deaths, 54.3 percent were among those with secondary education, 5.9 percent had primary education and 39.8 percent (Table 1) had attained not attained any level of education or were not

of school going age at the time of death. This description does not give any indication about between–group variation, as denominators for none, primary and secondary were unknown. One trend that has run through most of the studies on education–mortality relationship is their over–reliance on exogenous control group. Thus, education–health studies often use never attended school respondents or data to evaluate the existence or otherwise of a relationship. Regidor, Fuente, Calle, Navarro and Domínguez (2003) used between groups (never and ever attended school) approach and specified that mortality in never attended school was 4.7 and 3.7 times higher than men and women of higher educational level for respectively.

The effects of occupation on mortality have received much attention in demographic and public health (occupational epidemiology) literature. The 1960s and 70s critical social movements brought to public notice the extent of oppressions and hazards minority groups were exposed to in factories. Cutler, Deaton and Lleras–Muney (2006) believe that, among the socio–economic determinants of health, occupation is, presumably, the most researched in recent years. Despite the fact that there are no stringent rules applied to categorising occupations, the commonly used distinctions are manual and non–manual occupations. For this, like all other socio–economic variables, the evidences are not straightforward. Farmers among the dead between 1995 and 1999 were 43.8 percent. In a community where 90 percent of the people are farmers, it is not surprising to have more farmers dying than any other category of occupation. Students constituted 10.8 percent and traders amongst them were 3.7 percent. Government or private employees were 1.8 percent

(Table 1). The low proportion of government or private employees is because those within this category in the district are few.

Annual dynamics in structure of mortality

Crude death rate (CDR) is an aggregate index of number of mortality cases in a population expressed as a ratio of the size of that population, per a unit over time, often indicated per 1000 or 100,000. In this thesis, crude death per thousand has been used for this exploration. Even though demographers prefer specific rates, such as age–specific or sex–specific to delineate mortality burden of each sub–population, in circumstances where the required denominators are not available, CDR is considered appropriate as it gives the overall impression about a population's death structure.

Table 2 shows CDR for the period 1995–1999. The CDR of 18.4/1000 population in 1996 was the highest within the period. After 1996, CDR in the district showed a progressive decline to 16.9/1000 in 1997 and 14.7/1000 in 1999. Compared to CDR for Ghana in 1995 of 11.6 per 1000, and 14.7/1000 in 2000, those of the district are high. Current estimate for Ghana is around 8.5 per 1000. These figures suggest relatively high mortality rates in the district (www.uneca.org). The CDR then partially shows that Kassena–Nankana could be located in the third stage of Omran's theory. This interpretation doubtless, requires some caution as CDR is influenced by variables such as the age structure of a population and causes of death. This weakness of the CDR measure signifies that interpretation of results intending to measure health based on CDR need to hinge around circumspection. Nevertheless, CDR

provides a proxy indicator for further exploration to uncover patterns among various groupings within a population, such as age, sex or causes of death.

Table 2: Crude death rates from 1995 to 1999

Year of Observation	Total Population	Crude Mortality Rate (per 1000)
1995	125521	17.5
1996	137835	18.4
1997	137312	16.9
1998	135933	15.7
1999	136955	14.7

Source: NDSS Data Unit, 2009

Spatial trends and patterns of death

Cassel (1976) entreated epidemiologists to endeavour to understand whether there are environmental factors that made some groups of people more vulnerable to some diseases than others about four decades ago. Even though Cassel's concern was on the neglected social environment, his query nevertheless inspired a new quest to investigate social environmental risk factors of health alongside environmental risk factors. Spatial mortality counts have been a long cherished tradition in epidemiology. Snow's (1854) study on cholera in 17th Century Britain was typical of a spatial investigation. Current spatial analyses employ advance geographic information systems in mapping diseases patterns. This section presents CDR for the NDSS zones.

These zones are central, east, north, south and west. The zone with the lowest CDR for 1995 was the central zone (3.1 per 1000). The highest CDR for 1995 was marked in the western zone (17.9/1000). This trend of lowest

and the highest CDRs that were observed in 1995 between central and western were again repeated in 1996. However, instead of the anticipated decline, each of the zones recorded an increase over the previous year. Crude death rate for central zone increased by approximately threefold; that is, from 3.1/1000 to 10.7/1000 between 1995 and 1996. In reality however, this upward movement cannot be ascribed to any abnormality in health in the zone. From 1993 when the NHCR started collecting data on mortality in the district, Navrongo, the capital of the district was not part of the surveillance system. It became part of the system in the last quarter of 1995. This may partly account for the supposed abnormal increase.

In 1997, the northern zone (18.6/1000) reported the highest crude death rate with the central zone reporting the lowest. At the end of 1999, all the zones had experienced declining CDR, except central zone, which had recorded 0.3 more over the 9.2/1000 documented in 1998. As shown in Figure 4, the remaining zones consistently showed some marked improvement after 1996. In spite of the fact that the southern zone was second to the central in terms of CDR scores, the burden of mortality was high in the southern zone as 30 percent of all deaths occurred in the zone. Thus, there appears to be zonal differences. This brings to bear the earlier assertion that mortality structure analysis with CDR approach has an inherent potential of obscuring important details.

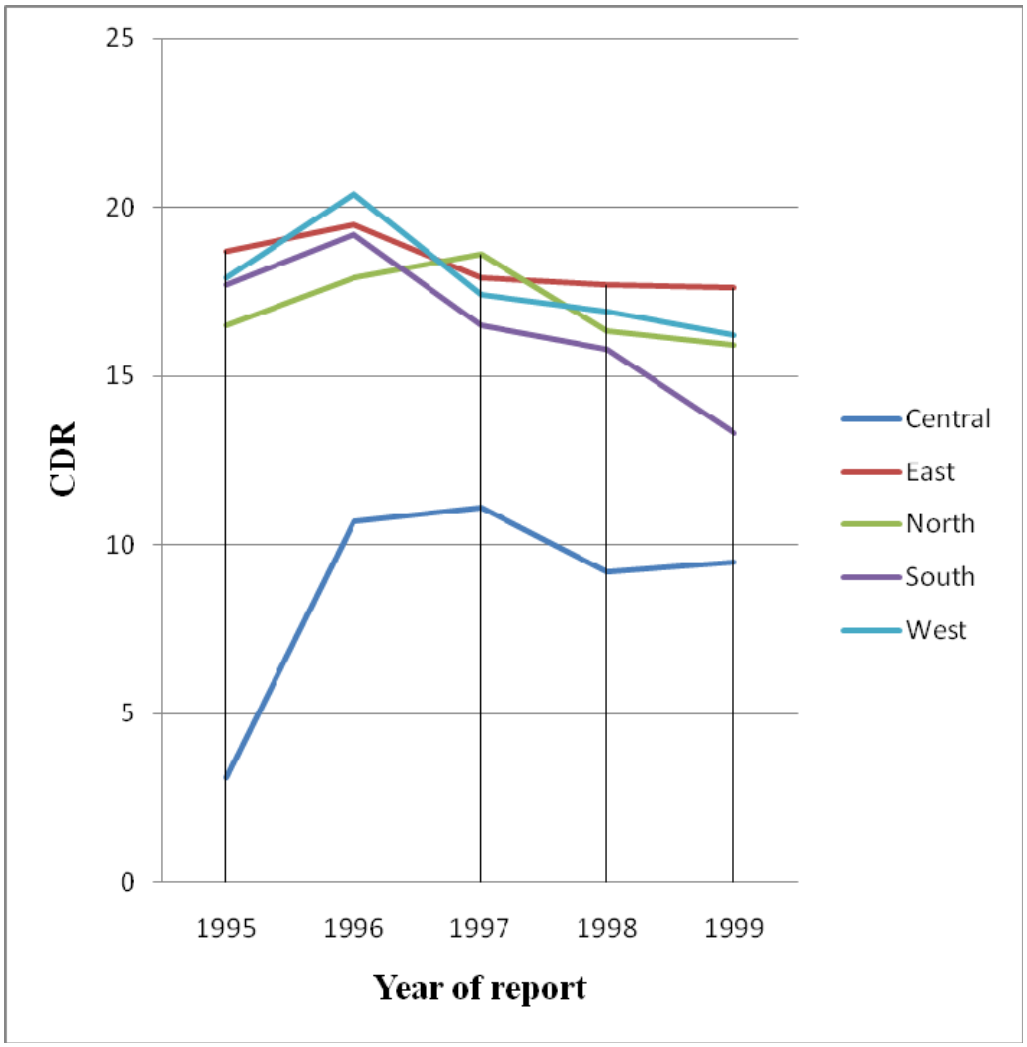

Figure 4: Annual crude death rates of the zones from 1995 to 1999

Source: NDSS Data Unit, 2009

To identify variations in deaths among the zones, the selectivity index was used. The selectivity index concept is based on the assumption that, for any event, the proportion must be equal to the proportion of the population base. During 1995, two zones, central (–0.008) and north (–0.014) recorded deaths below their shares of the population. In 1996, the selectivity effect was less than the population proportion in the central (–0.039) and north (–0.006) zones. Central (–0.033) and south (–0.008) were the zones that experienced deaths below the proportion of their populations in 1997. In 1998, only one

zone, central zone recorded deaths below its population proportion (–0.039). By 1999, three zones had documented selective indices of –0.033 (central), –0.011 (north) and –0.028 (south), while the east and the west had deaths higher than their share of the population (Table 3).

The results (Table 3) show disparities in the distribution of deaths. Central and northern zones consistently showed lower proportions of deaths than their population share throughout the study period while east, west and south reported higher proportions of deaths than their proportion of the population. Population proportion of an area has been identified as an important predictor of mortality. This is more noticeable in areas with certain endemic infectious diseases. For instance, the relatively devastating effects of recent outbreaks of respiratory related diseases in some Asian countries like China and Indonesia are cases in point. Salau, Galpin and Odimegwu (2006) have indeed strongly considered population density or proportion as an influential factor in infant deaths in some rural communities in Nigeria. This phenomenon occurs primarily because such high population concentration increases the rate of man–to–man infection.

Table 3: Selectivity indices of death by zone

Year	Zone	Proportion of death	Proportion of population	Selectivity Index
1995	Central	0.003	0.011	–0.008
	East	0.203	0.190	0.013
	North	0.230	0.244	–0.014
	South	0.328	0.325	0.003
	West	0.233	0.228	0.005
1996	Central	0.055	0.094	–0.039
	East	0.182	0.172	0.010
	North	0.219	0.225	–0.006
	South	0.309	0.297	0.012
	West	0.233	0.209	0.024
1997	Central	0.062	0.095	–0.033
	East	0.181	0.170	0.011
	North	0.249	0.225	0.024
	South	0.290	0.298	–0.008
	West	0.216	0.210	0.006
1998	Central	0.056	0.095	–0.039
	East	0.183	0.168	0.015
	North	0.233	0.226	0.007
	South	0.298	0.297	0.001
	West	0.228	0.212	0.016
1999	Central	0.061	0.094	–0.033
	East	0.190	0.168	0.022
	North	0.244	0.255	–0.011
	South	0.271	0.299	–0.028
	West	0.232	0.211	0.021

Source: NDSS Data Unit, 2009

Temporal (seasonal and monthly) patterns of death

Months of death have been grouped into four quarters. The least percentage of deaths occurred on the average between April and June (20.4%). October to December accounted for 28.9 percent of the total deaths and this was the highest. It appears that mortality in Kassena–Nankana follows a seasonal pattern, with peak mortality in October to December. These patterns are due, primarily to, environmental circumstances. As far as health is

concerned, the most favourable climate in Kassena–Nankana is April to June. This quarter marks the onset of the rainy season and this period is not characterised with intense rains to induce the influx of malaria parasites in an area that has been described as malaria holoendemic by Binka et al. (1994). Diarrhoea, which is also common during intense rainy seasons, is also not likely to be prevalent, as sanitary conditions would not have deteriorated. This same quarter again marks the end of the hot climate, during which time cerebrospinal meningitis and other infectious diseases would be receding. Figure 5 gives a pictorial view of the quarterly trends.

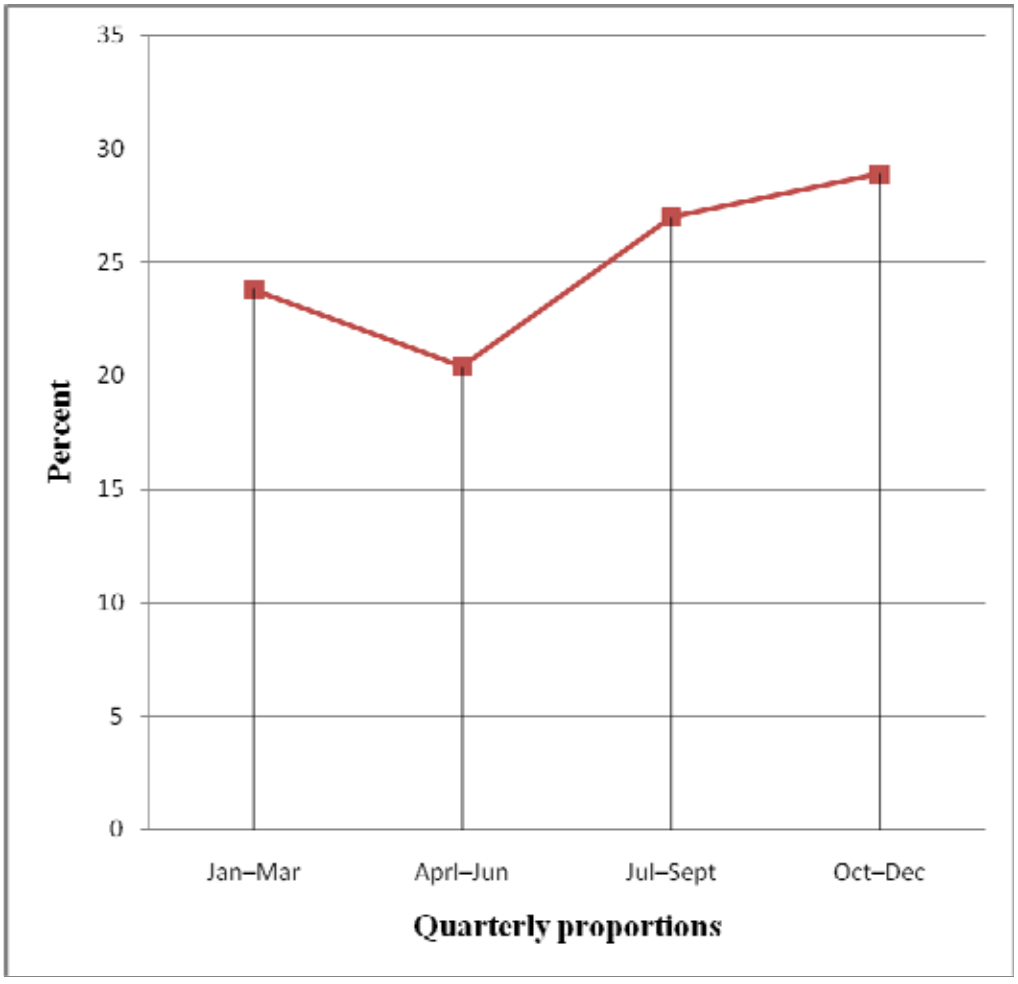

Figure 5: Quarterly mortality proportions from 1995 to 1999

Source: NDSS Data Unit, 2009

Hippocrates, the father of medicine advised: "whoever wishes to investigate medicine properly should proceed thus: in the first place consider the seasons of the year, and what effects each of them produces, for they are not at all alike, but differ much from themselves in regard to their changes" (Hippocrates 400BC, cited in Rau, 2007, p. 5–6). This suggestion by Hippocrates has prompted dozens of research works that consider seasons since then, with two major postulates emerging from these studies: biological context(s) and socio–cultural conditions. The biological model is associated with mortality arising from changing seasons. Socio–cultural factors are more of distal inputs such as loneliness and lack of employment. Gemmell, McLoone, Dickinson and Watt (2000) accept the biological reasoning but quickly add that seasons are able to sway mortality from "normal" levels because of low social protection against temperatures by some people, rather than temperature itself.

The northern part of Ghana in its entirety is characterised by two main seasons: hot and dry. The dry season begins in November and ends in March. April to October is the raining season. It is therefore important to assess how deaths occur within the seasons. As shown in Figure 6, November (11.6%) recorded the highest number of deaths in 1995. This was followed by October (10.6 percent). The lowest proportion of mortality in 1995 occurred in June (6.2%). In 1996, the highest deaths occurred in August and October, each recording 10.9 percent. The minimum deaths were reported in February (6.5%) and April (6.5%). That is, fewer deaths occurred in the first half of the year. The pattern of high and low deaths in the first and second halves varied in 1997. Peak mortality of 12.8 percent was observed in March in 1997 and

this was the highest recorded for any single month for the five–year period. October emerged as the month which registered the highest proportions in three out of the five years, with the total for the period being 10.8 percent and the lowest deaths overall being recorded in May (6.2%). The trends observed in sum suggest that what causes deaths in the district respond, directly or indirectly to the physical with some few exceptions, for instance food security. Typical of the study area, food is more available during the last quarter of the year, yet deaths were pretty high during this period while deaths were at the ebb during the food insecure quarters (April through June). This presents a paradox that may probably be explained by environmentally associated diseases such as malaria.

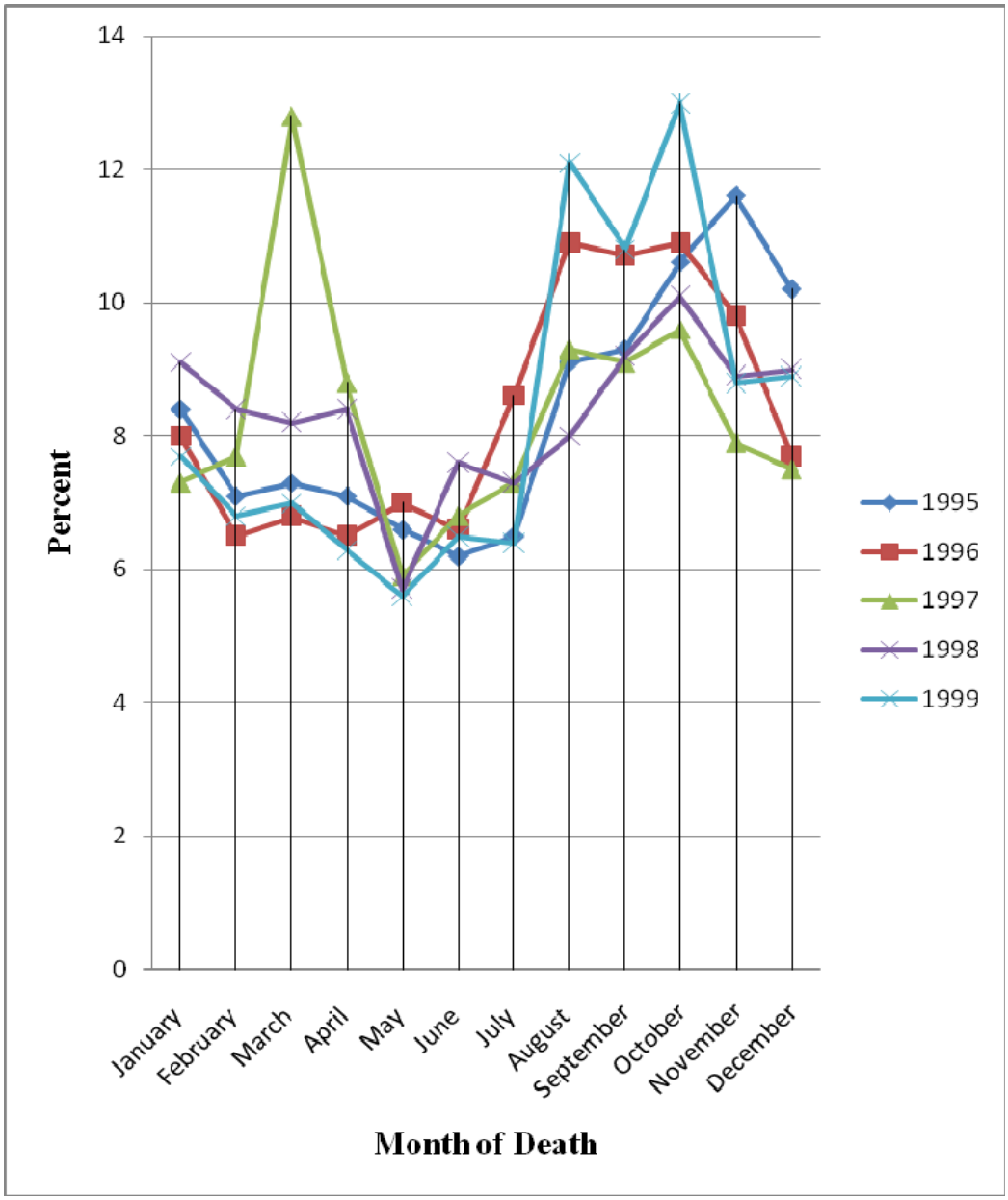

Figure 6: Monthly mortality trends

Source: NDSS Data Unit, 2009

According to Rau and Doblhammer (2003), age–seasonal mortality dynamics have received little attention both in demographic and epidemiologic literature. However, it is important to identify whether there are changes in deaths during certain seasons by age. Particularly, with the current changing climatic conditions, seasonal–age differences are crucial for intervention programmes.

Within Kassena–Nankana, neonatal and infant (less than one year) deaths were highest in the fourth quarter (33.8%) with the lowest in the second quarter (15.6%). A third of the deaths among 1–4 year olds occurred in the fourth quarter. For the ages 5–14 years, 36 percent occurred in the first quarter and for the 15–44 age groups, no single quarter clearly stood out, with the exception of the first quarter (27.2%). The fourth quarter, with 27 percent, had more deaths within 44–59 age groups, with the lowest being the 22.5 percent in the second quarter. More deaths were recorded in the third quarter (28.9%) for the adults 60 years or more. Throughout the period, the peak period for the age cluster 5–14 was January to April. Conversely, the trough for the other cohorts was virtually within the last five months of the year (Table 4). A Chi–square test of age with season gave χ^2=3.820 (Df. =5); ρ=0.000 at 0.05 alpha level, indicating a significant relationship between age and seasonal mortality. The reasons for these kinds of relationships are often detected by causes of death analysis. For example, respiratory diseases that are fatal in older adults and children could rise during the dry and cold and warm climates respectively. These are explored in detail in Chapter Five.

Table 4: Age–monthly mortality trends

	Age Intervals (%)							
	0	1–4	5–14	15–44	45–59	60+	Total	N
January	8.0	6.2	10.4	9.3	9.2	7.3	8.1	905
February	5.8	6.8	7.5	8.6	8.9	6.9	7.3	815
March	6.8	8.3	18.2	9.3	8.5	7.1	8.4	945
April	5.2	6.2	11.5	8.5	7.8	7.9	7.4	829
May	4.5	4.6	5.5	8.3	7.0	6.9	6.2	694
June	5.9	5.0	5.7	6.7	7.7	7.9	6.7	755
July	7.4	7.4	6.1	7.1	6.9	7.6	7.3	813
August	10.9	11.2	6.5	9.3	8.9	10.2	9.9	1106
September	11.7	10.7	6.4	7.1	8.1	11.2	9.8	1102
October	14.4	12.4	9.1	9.4	8.6	9.8	10.8	1209
November	11.4	12.5	6.5	7.6	8.2	8.8	9.4	1055
December	8.0	8.7	6.5	8.8	10.2	8.5	8.6	966
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	
N	2283	1570	704	1573	1873	3191		11194

Source: NDSS Data Unit, 2009

χ^2=3.820; ρ=0.000; α=0.05; Df.=5

Gemmell et al. (2000) have described seasonal mortality as a phenomenon where vulnerable people are “harvested” from a wider population. For instance, in Western countries, it is easier for one to die during winter than in summer and these may vary by sex. However, Rau and Doblhammer (2003) assert that these issues have not received much

consideration. For males and females, peak deaths were in October (10.8% each). Similarly, the lowest deaths occurred in May for both sexes as shown in Table 5. Based on χ^2 at 95% (ρ = 0.044), there was some statistical significance between sex and month of death, although the difference was not much.

Table 5: Sex–monthly mortality trends

Month of Death	Sex (%)			N
	Female	Male	Total	
January	7.5	8.6	8.1	905
February	7.4	7.2	7.3	815
March	8.0	8.8	8.4	945
April	7.1	7.7	7.4	829
May	6.6	5.8	6.2	694
June	7.0	6.5	6.7	755
July	7.9	6.7	7.3	813
August	10.1	9.7	9.9	1106
September	9.8	9.9	9.8	1102
October	10.8	10.8	10.8	1209
November	9.6	9.2	9.4	1055
December	8.1	9.1	8.6	966
Total	100.0	100.0	100.0	
N	5459	5735		11194

Source: NDSS Data Unit, 2009

χ^2=20.1: ρ=0.044: α=0.05: Df.=11

Demographic trends and patterns of death

Age is a major characteristic in any demographic analysis. Migration, fertility and mortality patterns are always assessed with respect to age. This component of the analysis explores variation by age from 1995 to 1999. Between 1995 and 1999, 28.5 percent of mortalities occurred among those aged 60 or more. The share for neonates and infants was 20.4 percent and those aged 5–14 accounted for 6.3 percent (Table 6).

The distribution of death amongst the age groups for 1995–99 did not show any discernible pattern (Table 6). Almost all age groups remained unchanged in their share of deaths, apart from 1996 when the proportions for those aged 1–4 increased from 12.5 percent in 1995 to 17 percent in 1997 and that of 45–49 years declined from 17.5 percent in 1995 to 16 percent in 1996. Deaths among those less than one year of age were the second highest in proportion from 1995 to 1999. Proportions among those aged 60+ decreased from 27 percent to 26.5 percent between 1995 and 1997 (Table 6).

As indicated in Table 6, the age group 60+ recorded the highest proportion of deaths throughout the period. In terms of changes, the proportion of deaths among those aged 15–44 years and 60+ years was a contrast to what was observed in that under–one year, 1–4 years, 5–14 years and 45–59 years. For instance, there was a sharp rise in the proportion of deaths among the 5–14 years, from 5.6 percent in 1996 to 9.1 percent in 1997 (close to 38.5%) but decreased to 5.5 percent in 1998 and further to 4.7 percent in 1999. For the 15–44 age groups, the proportion declined steadily from 14 percent in 1995 to 12 percent in 1996 but had risen to 17.4 percent by 1999. Deaths among those aged 60+ were 26.5 percent in 1997, increased to 30.8 percent and then to 32.4

percent in 1998 and 1999 respectively (Table 6). Mean age at death was 34.5. Equally, the modal age of death was recorded among those less than one year. Half of total of deaths tracked from 1995 to 1999 happened before age 37 years.

Table 6: Proportion of age–annual death patterns

	Year of Observation (%)						
Age Groups	1995	1996	1997	1998	1999	Total	N
0	22.6	22.2	19.5	19.5	17.7	20.4	2283
1–4	12.5	17.0	14.0	13.3	12.7	14.0	1570
5–14	6.4	5.6	9.1	5.5	4.7	6.3	704
15–44	14.0	12.6	13.6	13.8	16.7	14.1	1573
45–59	17.5	16.0	17.3	17.1	15.9	16.7	1873
60+	27.1	26.6	26.5	30.8	32.4	28.5	3191
Total	100.0	100.0	100.0	100.0	100.0	100.0	11194
N	2198	2532	2317	2137	2010	11194	

Source: NDSS Data Unit, 2009

The age group that showed consistent decline in proportions was neonates and infants (less than 1 year). This could be explained by the numerous health intervention programmes that have been undertaken in the district since 1989 such as the VAST project. On the other hand, the proportion among adults 60 years and above increased by 16.3 percent. However, one has to be cautious in interpreting the noticed trends to mean 'real' improvement in mortality or otherwise since the reported deaths could be complicated by age misreporting and lack of population of the district for

calculating age–specifc rates. Age misreporting has been found to be one major challenge in demographic data from Africa (Awusabo–Asare, 1990; Ntozi, Kabera, Ssekamatte–Ssebuliba, Mukiza–Gaperere & Kamateeka)

The data also provide evidence on sex differentials. In both social and bio–medical literature, it is generally accepted that risk factors in men and women vary due to variations in socio–economic conditions. This in turn influences mortality burdens for females and males differentially. As Allotey and Gyapong (2005) observed, much as women are more vulnerable to diseases related to household responsibilities, men are similarly at a disadvantage with respect to outdoor diseases which are varied by seasons and socio–economic circumstances. Besides socio–economic and other related issues, sex differences in years of life lost (YLL) could arise from biological factors, like sex hormones, X–chromosome inactivation and immunological response. These biological variables have been found to be present in collagen disease (Kazuto, 2006). For the 11,194 deaths that were registered, 5459 or 48.8 percent occurred among females and the rest (51.2%) were among males as shown in Table 7.

Further investigations were carried out to determine whether there was decline in either of the two. Out of the 2198 deaths that occurred in 1995, 51.0 percent were females. Nevertheless, from 1996 more males than females died (Table 7). The sex composition of the district in 2000 was 48.1 percent males as against 51.9 percent females. The literature reveals several postulates to explain these mortality differences in males and females, even though in most societies females outnumber males. Despite the fact that a number of studies have found out that women use health services accompanied by severe self–

assessment of illnesses more than men, women live longer than men of the same age do. Based on this, some argue that, in reality, women are healthier than men are but women are less stoical (Case & Paxson, 2004).

Table 7: Sex trends of mortality between 1995 and 1999

	Year of Observation (%)						
Sex	1995	1996	1997	1998	1999	Total	N
Female	51.0	49.3	48.0	47.1	48.2	48.8	5459
Male	49.0	50.7	52.0	52.9	51.8	51.2	5735
Total	100.0	100.0	100.0	100.0	100.0	100.0	
N	2198	2532	2317	2137	2010		11194

Source: NDSS Data Unit, 2009

In almost all countries of the world, females outlive males. In Ghana, there is about one year difference in life expectancy between men (58 years) and women (59 years). It has been established that, the gap between men and women is not linear for all ages. This argument is sustained by the fact that in early years of life, females have more advantage than males. This lead shrinks in middle years where the tide turns in favour of males, mainly due to the hazards women encounter during the reproductive years. In later years of life, the advantage of women re–occurs because of underlying social, biological and psychological determinants (Case & Paxson, 2004).

Based on the NDSS data, these issues were examined to establish whether these assumptions are applicable in the Kassena–Nankana as well. For mortality within the first eleven months, more males (50.4%) than females (49.6%) died. This pattern of more deaths among males than females also

occurred among the 1–4, 5–14 and 15–44 year groups (Figure 7). It was anticipated that more females than males would die in the 15–44 age group due to hazards involved in reproduction during these ages. However, this was not the case as 46.7 percent of all deaths occurred among females and 53.3 percent among males. Largely, the female population density within 45–49 years between 1995 and 1999 was higher than males. In the 2000 Population and Housing Census, females aged between 15 and 59 years in the district constituted 6.3 percent as against 5.2 percent of males.

Among those aged 60+, more females died than males: in 2000, females 60+ accounted for 4.5 percent of the population and males 4.4 percent. Statistical test of significance shows a significant relationship between sex and age at death at 95 percent confidence level (Chi–square test gave χ^2=21.93; ρ=0.001; α=0.05 with 5 degrees of freedom). Nevertheless, this brings into perspective the concept of natural selection, which argues perceptively that more dominant traits will remain over a generational course (Darwin, 1859). This notion presupposes that, all things being equal, more people in advanced ages should die for the next generation to succeed them. Figure 7 provides a visual impression of the variations between males and females for the under review (199–1999).

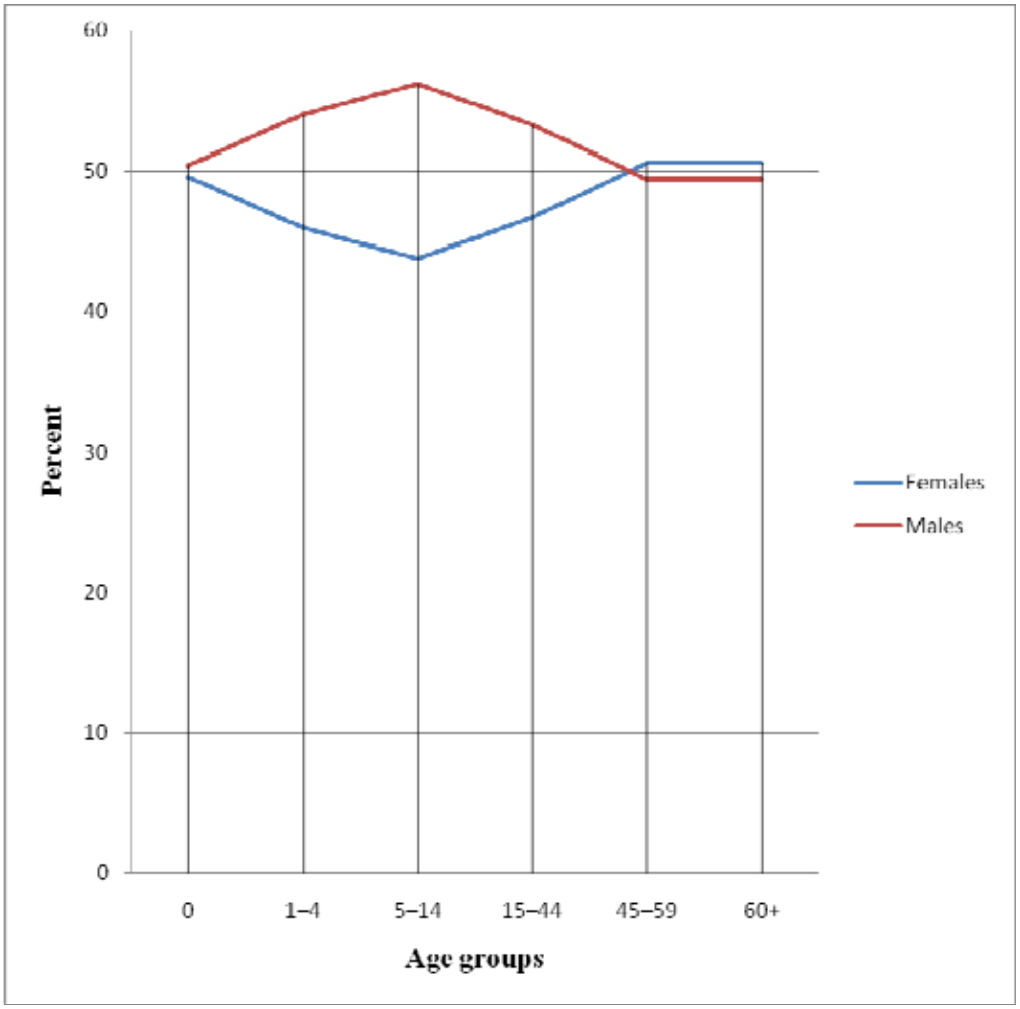

Figure 7: Sex and age at death

Source: NDSS Data Unit, 2009

Chi–Square Test: χ^2=21.93; ρ=0.001; α=0.05; Df=5

Summary

Basically, this chapter sought to identify spatial, demographic and temporal differences in deaths in the district. First crude death rates were found to be high in 1996 but declined considerably by 1999. Spatial differences in mortality burdens among the five zones showed that the eastern part of the district had higher proportion of deaths than any other area, followed by the southern zone. The spatial differences observed re–echo the argument for the need to consider epidemiologic research at the ecological

level (Susser & Susser, 1996b). Through this approach, it is believed that community health challenges would be made clearer.

The proportion of deaths among the 60 years and above was consistently higher than the other age groups, around 28.5 percent while deaths among the 5–14 years (6.3%) were the lowest. There were also sex differentials with the proportion of deaths among females being lower than that of males during the period. From 0–44 years, deaths among males outnumbered female. However, after 45 years, mortality tilted towards females. The implication of these observed patterns is that, health delivery policy interventions need to set the right priorities in relation to age and sex. The wholesale approach of assuming similar needs must be reconsidered to be in tune with realities on the ground.

A popular theme that has received attention in public health discourse is how seasons affect mortality structure. Seasons, measured in months (quarters) were examined with respect to sex and age. Besides 1997 where peak deaths were recorded in March (first quarter), in 1995–6, and 1998–9, the peaks were observed in the third and fourth quarters. The highest proportion of deaths among females and males occurred in October. These patterns give an indication of differences in seasonal deaths in developed countries and developing countries. Whereas the majority of deaths are recorded mostly during the winter in the north, in the tropics, the peak period is the wet season. In the tropical regions, deaths are mainly from infectious and parasitic diseases which peak in the rainy season. Principal among the reasons for this trend is that the rainy season provides suitable breeding grounds for insects such as mosquitoes and other organisms that induce conditions such as gastroenteritis.

The pattern indicates that seasonal influences on deaths are not age–selective. Whereas it is important to strive to reduce deaths throughout the year, it is imperative to consider specific seasons in order to tailor appropriate health improvement interventions.

CHAPTER FIVE

CAUSES OF DEATH IN THE KASSENA–NANKANA DISTRICT, 1995–1999

Introduction

Knowledge about causes of death helps to develop preventive measures and/or cure for them. Historically the study of diseases has led to the development of drugs and diagnostic procedures. The scientific quest to improve diagnoses of causes of death has considerably led to continuous precision. Before modern medicine and the antecedent pathological technologies, opinions of lay men were used extensively in describing diseases that had killed people. In 17th Century Britain, workers who were referred to as "death searchers" were engaged to enquire about deaths and the probable diseases that had caused these deaths and reports to local authorities. This method is a precursor of verbal autopsy (Garennea & Fauveau, 2006).

With developing countries such as Ghana which lack the capacity to undertake full diagnosis of deaths and to capture causes of deaths, opinions of lay persons have been used to document causes of death. This approach known as verbal autopsy, has been used to collect information on deaths in the KND since the early 1990s. Verbal autopsy (VA) as used was first named in a pioneer project in Narangwal (India) and later, Keneba (Gambia) (Garennea & Fauveau, 2006). This chapter deals with causes of death based on VA in KND from 1995 to 1999, using the ICD+10 categorisation of diseases.

Main causes of death

Diseases are caused by both distal and proximate factors: Distal factors are identified as socially predisposing while proximate are biologically endogenous variables. In general, distal variables are difficult to quantify. Consequently, their identifications are sometimes speculative. Proximate factors are more of biologically endogenous factors. To be able to identify distal factors variables however, sometimes, it is appropriate to give a brief knowledge of basic anatomic or etiologic shaping factors, especially, diseases of profound public health relevance.

Under ICD+10 coding of diseases, the broad categories applicable to the data for this analysis were (1) infectious and parasitic, (2) respiratory infections, (3) nutritional deficiencies, (4) digestive system disorders, (5) childhood clusters, (6) genito–urinary disorders, (7) maternal conditions, (8) peri–natal conditions and (9) malignant neoplasm. The rest were (10) miscellaneous communicable and (11) non–communicable diseases. For the remainder of this section, brief etiologic features of some diseases are provided to help in hypothesizing plausible risk factors.

From Table 8, malaria accounted for 19.1 percent of all deaths in the district from 1995 to 1999. Annually, malaria attacks 350–500 million people in the world. About 90 percent of these deaths occur in Africa (United Nations Children's Fund [UNICEF], 2008). Binka et al. (1994) have described Kassena–Nankana as a malaria holoendemic zone. It is therefore not surprising that malaria kills more people than any other disease. The proportion of deaths due to malaria in Kassena–Nankana district is consistent with national data, as observed by Adams and Boerma (2006).

Diarrhoea (11.4%) appeared as the second, followed by acute febrile (3.8%). Diarrhoea mortality is generally a manifestation of inadequate sanitation conditions. As of 2000, only 58.8 percent of residents in Kassena–Nankana had access to bore holes and pipe borne water and 84 percent had no toilet facility of any kind in the surveillance area (Ghana Statistical Service, 2000). This might explain the high incidence of diarrhoea as an important cause of death in the district.

Acute febrile is often diagnosed as typhoid fever. According to the WHO (1998), suspected typhoid case should indicate two major manifestations and at least one minor of the following clinical criteria: Major criteria are persistent fever for more than two days that fluctuates between low, high, and abdominal ache. The minor includes non–productive cough and less than 60 heartbeats per minute (bradycardia). Physical expressions include fever for more than three days with profuse night sweating, fatigue, and depression, plus one or more of the following: weight loss, headache, and joint pains (arthralgia). This is an infection typical of rural communities. Prevalence of febrile in livestock around residential areas is an important predictor of acute febrile illness. Afifi et al. (2005) have observed high incidence of acute febrile illnesses around the environs of Nile Delta. Other risk factors are consumption of untreated animal products such as cow milk. The occupational environment of Kassena–Nankana, namely, animal rearing, the dominant occupation in the district, crop farming and the practice of leaving animals to roam in streets and around compounds to some extent, explains the relatively high incidence of acute febrile as cause of death in the area (Table 8).

Table 8: Epidemiologic profile of Kassena–Nankana, 1995 to 1999 based on Group I causes of death

Causes of death	N	Percent
Group I: Communicable, maternal, prenatal and nutritional causes		
Infectious and parasitic		
Malaria	2137	19.1
Gastro–enteritis/Diarrhoea	1271	11.4
Acute febrile illness	422	3.8
Tuberculosis	230	2.1
Septicaemia	154	1.4
Meningitis	152	1.4
AIDS	85	0.8
Hepatitis	30	0.3
Respiratory Infections		
Acute lower respiratory infection	526	4.7
Pneumonia	452	4.0
Nutritional deficiencies		
Anaemia	367	3.3
Malnutrition	93	0.8
Maternal conditions		
Haemorrhage/Obstructed labour	67	0.6
Abortion	9	0.1
Peri–natal conditions		
Prematurity	155	1.4
Birth Injury	32	0.3
Neonatal jaundice	17	0.2
Childhood clusters		
Measles	148	1.3
All other communicable (AOCD)	1011	9.0
Total	7525	67.2

Source: NDSS Data Unit, 2009

Septicaemia, a bacterial infection of the blood (blood poisoning) kills about 30,000 people annually throughout the world, and mainly children (Hicks, 2007). In the district, septicaemia constituted 1.4 percent of total

deaths. Septicaemia may be due to a complication of an untreated infection of the lungs or kidneys that escapes into the blood stream or a bacteria flow into the blood stream due to burns, infected wounds and boils. High fever, violent shivering, faintness, cold and pale hands and feet, rapid and shallow breathing, restlessness and loss of consciousness are some usual indications of the infection (Hicks, 2007).

Meningitis accounted for 1.4 percent (Table 8). Meningitis is a swelling of the protective lining of the brain and spinal cord. Viruses, bacteria and microorganisms and sometimes, certain drugs are biological causes of the disease. Symptoms include headache, neck stiffness, fever, confusion, vomiting and sensitivity to light and irritation and drowsiness in children. Bacterial meningitis (meningococcal) has been identified as the most prevalent in northern Ghana (www.wikipedia.com). Risk of infection varies from age, immune system ability and accommodation arrangements. Pastoralists are also noted for high infection rate (www.neurology.health–cares.net). AIDS (0.08%) and Hepatitis (0.03%) were the remaining infectious and parasitic diseases from verbal autopsy in the Kassena–Nankana District.

Another disease group of public health importance in poverty endemic countries and communities are respiratory communicable diseases. Commonest of these are acute lower respiratory infection (ALRI) and pneumonia. Overall, acute lower respiratory contributed 8.7 percent (Table 8) deaths to total deaths recorded in the area between 1995 and 1999. This was made up of 4.0 percent pneumonia deaths with the rest (4.7%) representing ALRI in the study area. Mizgerd (2008) has noted that, globally, ALRI destroys more human lives than malaria, cancer and heart attacks. Pneumonia

and ALRI are infections of one or both lungs, by either bacteria or virus or fungus. Symptoms include high fever, shaking chills, unusual rapid breathing and coughs (Schiffman, undated). Risk dynamics of infection increases with age, (lower and advanced ages) smoking and alcohol use, nutritional status and several others. Within the family of respiratory diseases, chronic obstructive pulmonary disease (COPD) (0.1%) was also present, though at a lower prevalence rate. It is however, a non–communicable disease. This disease is known to be typical among men, primarily due to smoking.

Nutritional deficiency mortality parallels income deficiencies as measured in quality of food intake. Mortality arising from nutritional deficiencies (anaemia and malnutrition) contributed 4.1 percent (460) of deaths. Anaemia, primarily attributed to iron deficiency is prevalent in pregnant women and children. Among the causes are heavy blood loss because of menstruation, or parasite infections such as hookworms, ascaris, and schistosomiasis. Acute and chronic infections of malaria, cancer, tuberculosis, and HIV can also lower blood Hb concentrations (www.who.int). Malnutrition is partly due to vitamin–A deficiency as well as generally low nutritional food in–take. Other diseases which led to deaths in the Group I were prematurity (1.4%), measles (1.3%), maternal haemorrhage (0.6%), abortion (0.1%) and birth injuries (0.3). Miscellaneous communicable diseases that rarely caused deaths were altogether 9.0 percent.

Non–communicable diseases are mostly genetic, life style induced, and these include cardiovascular diseases, digestive system disorders, COPD, cancers, neuro–psychiatric disorders and renal diseases (urinal retention) as indicated in Table 9.

Mortality assigned to cardiovascular diseases was 2.0 percent of the total deaths. Digestive system disorders identified during the various verbal autopsy interviews were acute abdominal pains and liver cirrhosis. They formed 2.3 percent of deaths and the majority of these were mainly acute abdominal disorders (2.1%) and liver cirrhosis (0.2%). Abdominal pains are acute when the onset is sudden. It has no definite cause and it is usually a consequence of complications. For example, gastroenteritis or hydrocele (lymphatic filariasis) is prevalent in the area and mostly common in children and old adults (Gyapong, Gyapong, Weiss & Tanne, 2000). Granted that NDSS was strictly applying the underlying concept, reported acute abdominal disorders appear to be low. Other non–communicable diseases that killed people were neuro–psychiatric disorders (0.4%), renal diseases (0.9%) and lung, breast and cervix cancers (0.3%). All other non–communicable diseases mortality was 7.8 percent.

Deaths due to injuries have become a serious public health issue in the twenty–first Century. First ten causes of years of life lost (YLL) in 2003 in low–income countries indicated injuries to be the ninth (2.3%). World Health Organisation (2007) states that 9 percent (5 million) of global deaths are attributable to intentional and unintentional injuries, one of the fifteen major causes of deaths. The recognition of injuries as public health issue in the 20th Century is considered by CDC (1999) as an important achievement in population health, obviously due to its abrupt but devastating long run impact on DALYs. Unintentional injuries were 4.1 percent with intentional injuries accounting for 0.4 percent.

About 16 percent (15.9%) of deaths could neither be determined nor identified. The main intention of verbal autopsy approach is to describe health at population level and not at the individual level (MEASURE Evaluation & US Census Bureau, 2007). As a result, the ability of the method to identify 84.1 percent of all deaths that occurred in Kassena–Nankana during the five–year surveillance makes the system appropriate in our present circumstances of low vital events registration.

The World Health Organization (2003) has identified a set of risk factors for mortality in poor countries. Included in the risks are unsafe sex (certainly important for HIV/AIDS), unsafe drinking water (one cause of diarrheal disease), and a variety of other factors such as mal–nutrition, and indoor smoke from burning solid fuels (important in respiratory conditions). The dominant causes of deaths in the district parallels or reflects the risk factors the WHO identified. In the sections that follow, efforts are made to establish whether there are differentials in patterns of these diseases with time, space and personal characteristics.

Table 9: Epidemiologic profile of Kassena–Nankana, 1995 to 1999 based on Group II, III and undetermined causes of death

Cause of death	N	Percent
Group II: Non–communicable diseases		
Cardiovascular diseases		
Cardiovascular disease	229	2
Digestive system disorders		
Acute Abdominal dis.	238	2.1
Liver cirrhosis	17	0.2
Genito–urinary disorders		
Renal disorders	101	0.9
Central Nervous system disorders		
Neuro–psychiatric disorders	41	0.4
Respiratory diseases		
Chronic Obstructive Pulmonary	13	0.1
Malignant neoplasm		
Cancers (breast, cervix and lung)	38	0.3
All other non–communicable (AONCD)	872	7.8
Group III: Intentional and unintentional injuries		
Unintentional Injuries	463	4.1
Intentional Injuries	45	0.4
Unknown/Undetermined	1781	15.9
Total	3838	32.8

Source: NDSS Data Unit, 2009

Annual dynamics of causes of death

Out of the 2189 deaths in 1995, 37.7 percent were due to infectious and parasitic diseases. Respiratory infections accounted for 7.8 percent, injuries 5.1 percent and cardiovascular mortality was 1.9 percent. All other communicable diseases resulted in 9.3 percent deaths while miscellaneous non–communicable deaths were 7.5 percent. Other causes of deaths of public health importance in 1995 comprised diseases belonging to childhood clusters

(3.1 percent). Similarly, nutritional deficiencies (anaemia and malnutrition) explicated 3.7 percent of all mortality cases identified in 1995 as shown in Table 10.

Whereas some improved, others retrogressed. For example, it was thought that malaria, acute febrile, septicaemia and diarrhoea and others in the infectious and parasitic family of diseases would decline. That was not the case. Infectious and parasitic diseases increased by 6.9 percent: from 37.5 percent in 1995 to 40.3 percent in 1999. Respiratory infectious disease (pneumonia and ALRI) increased by 24 percent in 1996 (10.3%). Nutritional disorders also increased in 1996. All other communicable and all other non–communicable deaths nonetheless reduced in 1996 respectively. One disease, measles, which falls under childhood clusters contribution to mortality in 1996 declined from that of 1995 as indicated in Table 10. Verbal autopsy's inability to diagnose all deaths remained stable between 1995 and 1996 (16.2%; Table 10).

Infectious and parasitic infections topped in 1997 (45.1%) which was also an increase over the 1996 trend. Risk of mortality arising from respiratory infectious disease was high among other diseases though it declined by 60.8 percent from 1996. Cardiovascular mortality constituted 1.6 percent of deaths in 1997, an improvement of 18.6 percent over 1996. Nutritional problems (4.3%) and injuries (3.7%) were also noteworthy, apart from mix of all other communicable (9.4%) and non–communicable (6.3%) mortality. Cardiovascular mortality increased by about 33 percent in 1998 while injuries increases by 5 percent as against 3.7 percent in 1997 (Table 10).

Table 10: Patterns of annual causes of death

	Year of Observation (%)						
COD	1995	1996	1997	1998	1999	Total	N
Infectious & para	37.5	40.3	45.1	44.5	40.0	41.5	4646
Maternal	0.8	0.4	0.6	0.8	0.7	0.7	76
Cancers	0.2	0.4	0.1	0.3	0.7	0.3	38
CNS	0.3	0.1	0.1	0.5	0.8	0.4	41
Cardiovascular	1.9	1.3	1.6	2.4	3.1	2.0	226
Respiratory	0.1	0.2	0.1	0.1	0.0	0.1	13
Digestive disorders	3.0	1.5	2.3	2.5	2.1	2.3	255
Injuries	5.1	4.9	3.7	4.9	5.2	4.7	530
Genitor–urinary	0.9	0.6	0.6	1.2	1.3	0.9	101
AOCD	9.3	8.7	9.4	9.9	7.9	9.0	1013
AONCD	7.5	5.7	6.3	6.7	11.0	7.3	821
Childhood clusters	3.1	2.8	0.3	0.0	0.0	1.3	148
Peri–natal	2.5	1.8	1.3	1.5	1.9	1.8	204
Nutritional	3.7	4.9	4.3	3.9	3.6	4.1	460
Respiratory infection	7.8	10.3	7.5	8.0	10.1	8.7	978
Undetermined	16.2	16.2	16.5	12.6	11.4	14.7	1644
Total	100.0	100.0	100.0	100.0	100.0	100.0	
N	2198	2532	2317	2137	2010		11194

Source: NDSS Data Unit, 2009

The health community considers a single reduction in a cause of death that is largely avoidable significant. There were no childhood cluster deaths in 1998 and 1999, suggesting a virtual elimination of measles, the only recorded childhood cluster of disease. As of 1999, some diseases within Group I causes of mortality had experienced sharp decline. For instance, infectious and parasitic deaths had declined by 10.1 percent, which in health was a highly important development. Unlike infectious and parasitic diseases, respiratory infections did not decrease. Instead, it increased by about 20 percent above its input in 1999. It is again relevant to observe that the inability of verbal autopsy to identify deaths began to decline after 1997. In 1999, 88.6 percent of cases could be identified, compared to 83.8 percent in 1995 (Table 10).

Actual cause–specific analysis was conducted to ascertain which of the public health concerned diseases declined or increased over the period. Specifically, malaria is considered. Incidence of malaria was unstable from 1995 to 1997 (17.2%, 20.1% and 23.2% respectively), declined considerably in 1998 to 20.1 percent and to 14 percent in 1999 (See Appendix 1 for more details). As of 1999, deaths due to malaria were overtaken by diarrhoea (14.9%) as the single most important contributor to total deaths. The decline in malaria in 1998 coincided with the global launch of Roll Back Malaria Partnership (RBM) initiative (www.rollbackmalaria.org). The RBM had, amongst its objectives, to strengthen health services and effective prevention and treatment of malaria. These objectives were to be achieved through multi– and inter–sectoral collaboration. The targets were to reduce malaria morbidity and mortality rates of 1998 by 50 percent by 2010 and a further 75 percent of achievements of 2010 in 2015. Activities included provision of affordable

insecticide treated bed net (ITN) and use of sulphadoxine–pyrimethamine (SP) in pregnancy (IPTp) (Owusu–Agyei et al., 2007). This could be a good reason for this decline as KND has often served as trial centre for several malaria intervention programmes since its inception.

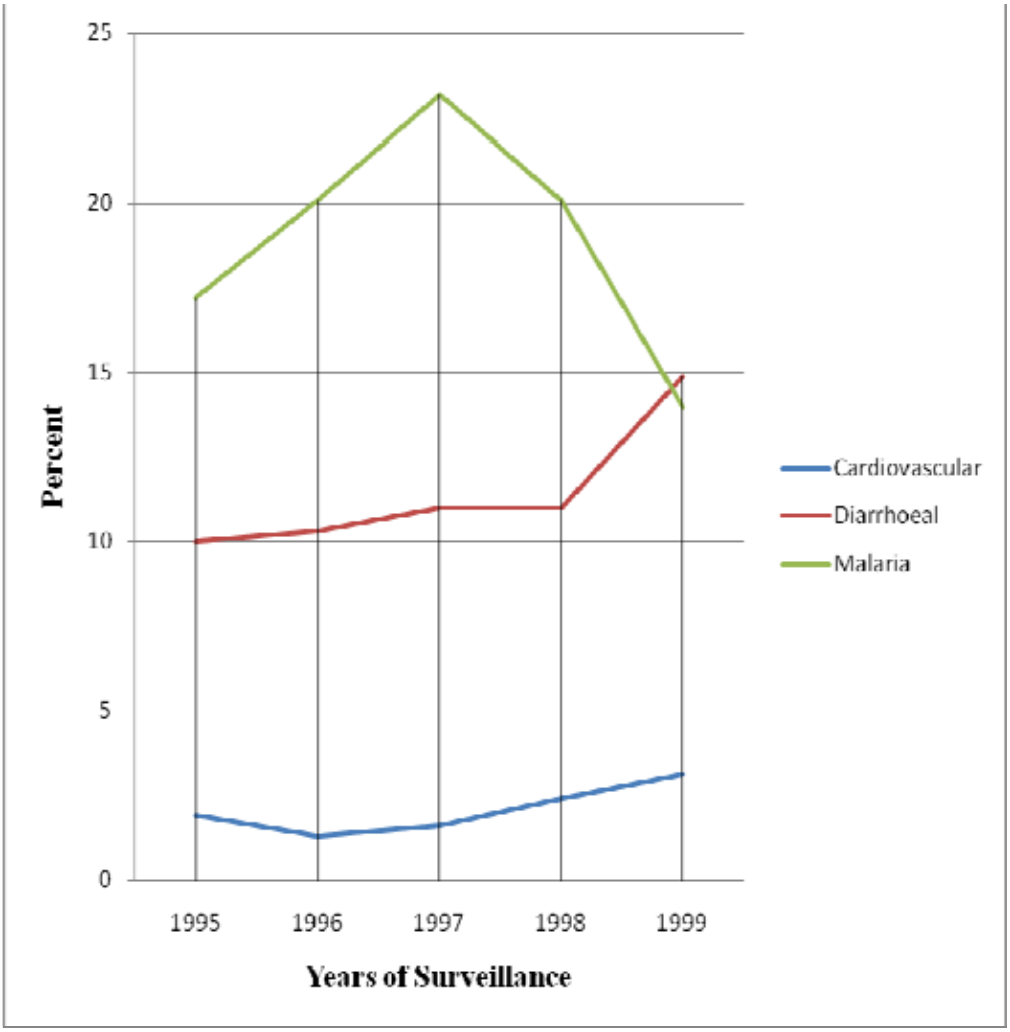

Figure 8: Trends of malaria, diarrhoea and cardiovascular mortality from 1995 to 1999

Source: NDSS Data Unit, 2009

With malaria as the dominant individual cause of death, it is compared with cardiovascular diseases, a condition whose incidence affects timing of the epidemiologic transition profoundly. The peak of malaria was established in

1997. It then assumed a decline in 1998 and 1999. Cardiovascular related mortality reduced in 1996 (1.3%) from its high of 1995 (1.9%) relative to 1996. Cardiovascular diseases influence on total mortality begun to rise in 1997 as evident in Figure 8.

Spatial patterns of causes of death

A common practice in public health is investigating incidences and prevalence of diseases within spatial or geographic perspective. Such ecological analyses are invaluable in shaping targets and the indicators thereof. In the preceding section, it was found out that some causes of mortality were generally declining while others rose. For this section, attention is given to whether these diseases varied by space.

In all the zones, infectious and parasitic diseases emerged as the leading cause of death: 41.9 percent in central, 43.6 percent in east, 43.5 percent in the north, 40.0 in the south percent and then 39.6 percent in the west. It appears that during the five–year period, it was more likely for a person to be killed by any of the infectious and parasitic diseases in all the zones. The proportion of maternal deaths in each zone was lowest in the central zone (0.2%) and highest in the eastern zone (0.9%). For many of the intervention studies in the study area, the NHRC has demarcated the eastern portion of the district as a control cell. Since some of these interventions are aligned towards maternal health, it is probable that the eastern zone could be suffering from the effects of not benefiting from some of the interventions.

Table 11: Spatial disease burdens

COD	Zone (%)						
	Central	East	North	South	West	Total	N
Infect. & P	41.9	43.6	43.5	40.0	39.6	41.5	4646
Maternal	0.2	0.9	0.6	0.8	0.6	0.7	76
Cancers	0.9	0.3	0.4	0.3	0.3	0.3	38
CNS	0.6	0.3	0.3	0.3	0.5	0.4	41
Cardiovascu	3.6	1.9	1.8	1.8	2.3	2.0	226
Respiratory	0.4	0.1	0.1	0.2	0.0	0.1	13
Digestive	3.0	2.6	1.7	2.0	2.7	2.3	255
Injuries	6.4	3.8	4.6	5.1	4.8	4.7	530
Genito–urin.	2.1	0.8	0.9	0.9	0.8	0.9	101
AOCD	8.0	9.2	9.4	8.5	9.6	9.0	1013
AONCD	7.9	7.9	6.3	8.4	6.5	7.3	821
Childhood	1.1	1.2	1.0	1.7	1.4	1.3	148
Peri–natal	1.3	1.7	1.9	1.8	1.9	1.8	204
Respiratory	5.4	6.6	10.0	7.8	11.2	8.7	978
Nutritional	4.3	3.7	3.6	5.1	3.7	4.1	460
Unknown	13.1	15.4	14.1	15.4	14.1	14.7	1644
Total	100.0	100.0	100.0	100.0	100.0	100.0	
N	535	2107	2629	3363	2560		11194

Source: NDSS Data Unit, 2009

Cancer, though constituted a smaller amount of total mortality, was higher in the central zone (0.9%) than the rest (as indicated in Table 11). Cardiovascular mortality was 3.6 percent of mortality in central zone with lower proportions in the east, north, south and west zones. Injury was also

higher in the central zone than the remaining zones: 6.4 percent in central, 3.8 percent in south, 4.6 percent in north, 5.1 percent in south and 4.8 percent in the western zone. Concerning respiratory infections, it was higher in the western zone (11.2%) and lowest in the central zone (Table 11). Chi–Square test results showed a very significant relationship between place (zone) of residence and cause of death from 1995 to 1999 (χ^2=37.128: ρ=0.000).

Demographic dimensions of causes of death

Social relations, distribution of power and the competencies to rally resources shape health status. To a certain extent, it is accepted that roles and exposure, perception of disease, response to illness, compliance to treatment and quality of care all have different influences on diseases burdens between males and females (Allotey & Gyapong, 2005). Recently, Goldstein (2007) has added sex differences in brains of males and females as accounting for differences in diseases, which favour females and subsequently, their mortality. Recognising that there are sex inequalities in many communities in Ghana, causes of death are analysed separately for men and women in the Kassena–Nankana District.

The proportion of infectious and parasitic diseases to female mortality was 42.5 percent compared to 40.6 percent among males. Sex differences in parasitic and infectious diseases intensities have been found to favour females and males at different times (Allotey & Gyapong, 2005). Some parasitic diseases such as malaria may show differences between men and women depending on seasons. During crop cultivation seasons where men are most likely to stay in farms to late evenings, the burden of such diseases are

probable to shift to males. During harvesting where a greater part of the responsibilities turn to women, they are prone to malaria attacks than males (Allotey & Gyapong, 2005). That notwithstanding, the disparities are usually induced by two main arguments: ecological or sociological and physiological (hormonal). Ecological or sociological factors are based on sex–specific behaviours. The second, physiological is linked to testosterone and immune system (Zuk & McKean, 1996). In KND where male dominance is relatively high, the sociological or ecological proposition on sex–infectious diseases, nexus seem more probable than the physiological (testosterone and immune system resistance).

Cardiovascular mortality incidence and prevalence has widely been reported to be lower among females than males prior to menopause in women. The differences become insignificant after menopause (Reckelhoff, 2001). Cardiovascular was responsible for 2.1 percent in females while it amounted to 1.9 percent of total male deaths as indicated in Table 12. Injuries as causes of death are expected to be higher among men than women. The data show that 5.6 percent of mortality among males was due to injuries compared to 3.6 percent for females. These differences in injuries in sex terms are linked to male evolutional instinct of high risk taking behaviour (Kruger & Nesse, 2006).

According to Nelson, Williams and Graham (2001), sex differences in respiratory infections have been established since the 1920s, which has increasingly been positive towards women. This is partly due to gaps in socio–economic status as well as behavioural predispositions such as smoking. Where these differences are found in childhood mortality about sex, X–

chromosome weakness is blamed. Between 1995 and 1999, mortality attributable to infectious diseases among females was 8.7 percent and for males, it was 8.8 percent, indicating no difference. Mortality due to malnutrition and anaemia constituted 4.5 percent of all female deaths and 3.8 percent of male deaths. In Ghana and especially, the northern sectors, several cultural tendencies expose women to nutritional disadvantages, due to male domination, including males determining what women eat (Debpuur et al., 2002) contributes to variations in diseases profile of men and women.

Analysis of certain specific diseases by sex as shown in Appendix 2 indicates few disparities in sex. For example, septicaemia was found to be biased towards males. This happens to be consistent with evidence in the literature Nazer (1981) found septicaemia male–female ratio of 2.1:1. Bang, Sharma, Sanyald, Bang and Ebrahima (2004) also found 60 percent (males) and 40 percent (females) infection rate in Kuwait. In spite of these gaps, biological evidence is still imprecise. Acute febrile mortality did not follow sex disparities as identified by Afifi et al. (2005), which according to them negatively tilts towards males.

Table 12: Causes of death by sex

	Sex (%)			
COD	Female	Male	Total	N
Infectious & Parasitic	42.5	40.6	41.5	4646
Maternal Condi	1.4	0.0	0.7	76
Cancers	0.4	0.2	0.3	38
CNS	0.3	0.4	0.4	41
Cardiovascular	2.1	1.9	2.0	226
Respiratory	0.1	0.1	0.1	13
Digestive Disorders	1.6	2.9	2.3	255
Injuries	3.6	5.9	4.7	530
Genito–Urinary	0.4	1.4	0.9	101
AOCD	9.6	8.5	9.0	1013
AONCD	7.2	7.5	7.3	821
Childhood Clusters	1.4	1.3	1.3	148
Peri–natal	1.9	1.8	1.8	204
Respiratory Infect	8.7	8.8	8.7	978
Nutritional	4.5	3.8	4.1	460
Undetermined	14.3	15.0	14.7	1644
Total	100.0	100.0	100.0	
N	5459	5735		11194

Source: NDSS, Data Unit, 2009

Earlier, it was noted that mortality was higher in early (less than 1 year) and later years (60 and beyond) of life as these two groups accounted for 48.9 percent of mortality from 1995 to 99. This section deals with causes of death by broad age groups. For each of the age intervals, infectious diseases were the commonest cause of death. The specifics, however, showed some differences (Table 13).

Table 13: Age variations in causes of death

		Age Intervals (%)						
COD	0	1–4	5–14	15–44	45–59	60+	Total	N
Infectious	46.8	60.1	53.1	32.8	34.7	34.3	41.5	4646
Maternal	0.0	0.0	0.0	4.5	0.3	0.0	0.7	76
Cancers	0.0	0.0	0.0	0.6	1.0	0.3	0.3	38
CNS	0.0	0.0	0.1	1.4	0.3	0.4	0.4	41
Cardiovascula	0.0	0.0	0.0	1.5	3.0	4.6	2.0	226
Respiratory	0.0	0.0	0.1	0.1	0.3	0.2	0.1	13
Digestive Dis.	0.0	0.0	0.3	4.1	3.5	3.9	2.3	255
Injuries	4.1	4.1	14.8	9.9	3.5	1.4	4.7	530
Genito–Urinar	0.0	0.0	0.4	1.9	1.1	1.5	0.9	101
AOCD	1.6	2.0	1.8	11.7	14.4	15.0	9.0	1013
AONCD	0.0	1.1	5.3	9.2	12.1	12.4	7.3	821
Childhood	1.4	5.5	4.1	0.0	0.0	0.0	1.3	148
Peri–natal	8.9	0.1	0.0	0.0	0.0	0.0	1.8	204
Nutritional	5.4	10.7	5.8	2.0	2.8	1.4	4.1	460
Respiratory In	14.5	9.9	5.8	4.0	7.0	8.0	8.7	978
Undetermined	17.3	6.5	8.2	16.3	16.1	16.7	14.7	1644
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	
N	2283	1570	704	1573	1873	3191		11194

Source: NDSS Data Unit, 2009

Infectious and parasitic mortality accounted for 46.8 percent in neonates and infants, approximately 60 percent among the 1–4 year olds and 53.1 percent for the 5–14 year group. The share of mortality from infectious and parasitic diseases was far lower: 32.8 percent for those aged 15–44 years, 34.7 percent 45–59 years and 34.3 percent for the 60+. The relatively low proportion of infectious and parasitic induced deaths after age fourteen could be due to development of resistance to certain infectious and parasitic

diseases. For example, people who are able to survive several malaria bouts in the childhood period later develop stronger immune resistance to future lethal malaria infections.

All reported cancers occurred within the 15–60 years with 45–59 year cohort recording one percent. Cardiovascular deaths also showed similar trend to cancer and accounted for 1.5 percent among those aged 15–44 years, three percent for 45–59 year olds, 4.6 percent among those adults 60 years and above. This gives a pattern of increasing risk of those diseases with age.

A cause of death that was registered for all ages was injuries. In children less than one year and those aged 1–4 years, their share of injury to total deaths was 4.1 percent each respectively. Injuries were the second highest cause of death among those aged 5–14 (14.8%) after infectious and parasitic diseases. Among ages 15–44 years, injuries accounted for 9.9 percent, also second to infectious and parasitic diseases. The contribution of injury to deaths in age groups 45–59 years was 3.5 percent.

Besides infectious and parasitic diseases, nutritional deficiencies were the dominant cause of mortality in under–five mortality (10.7%). For neonatal and infant deaths, respiratory infections accounted for 14.5 percent and parasitic diseases that could not be determined were 17.3 percent. Age at death showed a positive relationship with cause of death, given Υ of 0.34 which was very significant (ρ=0.000). This translates into 11.6 percent of deaths explained by age at death.

Table 12 has shown that some particular diseases are age–specific. The contribution of acute lower respiratory infection to deaths was observed to be decreasing with age. It contributed 14.5 percent to neonatal and infant

deaths, 9.5 percent in under–5 (1–4 years) deaths and 5.5 percent in late childhood deaths (5–14 years). Deaths from malaria were related to age: malaria was the main cause of neonatal and infant deaths (32.3%), under–five (42.2%) and ages 5–14 years (34.2%) deaths. Among those 15 years and above, deaths from malaria were low: (4.5%), 45–59 years (6.0%) and 60+ years (9.8%). Septicaemia showed a mix of relatively high of 5.6 percent in neonates (1–11 months) to a low of 1.4 percent among 5–14 year olds and 1 percent in 1–4 year groups relative to age. No incidence of septicaemia was registered beyond 15 years (See Appendix 2 for details). Figure 9 shows a graphical pattern of malaria, diarrhoea and acute lower respiratory infections with age. ALRI deaths declined with age. By age 15 and above, ALRI had little effect on total mortality in the district from 1995 to 1999. The magnitude of deaths due to acute lower respiratory infections in relation to age in this study correlates with other studies such as the one by child health and epidemiology research group (CHERG), which estimated that more than 2 million children who are less than five years die of ALRI annually. More than 75.0 percent are recorded in Africa and some Asian countries (Lim, Steinhoff, Girosi et al., 2006).

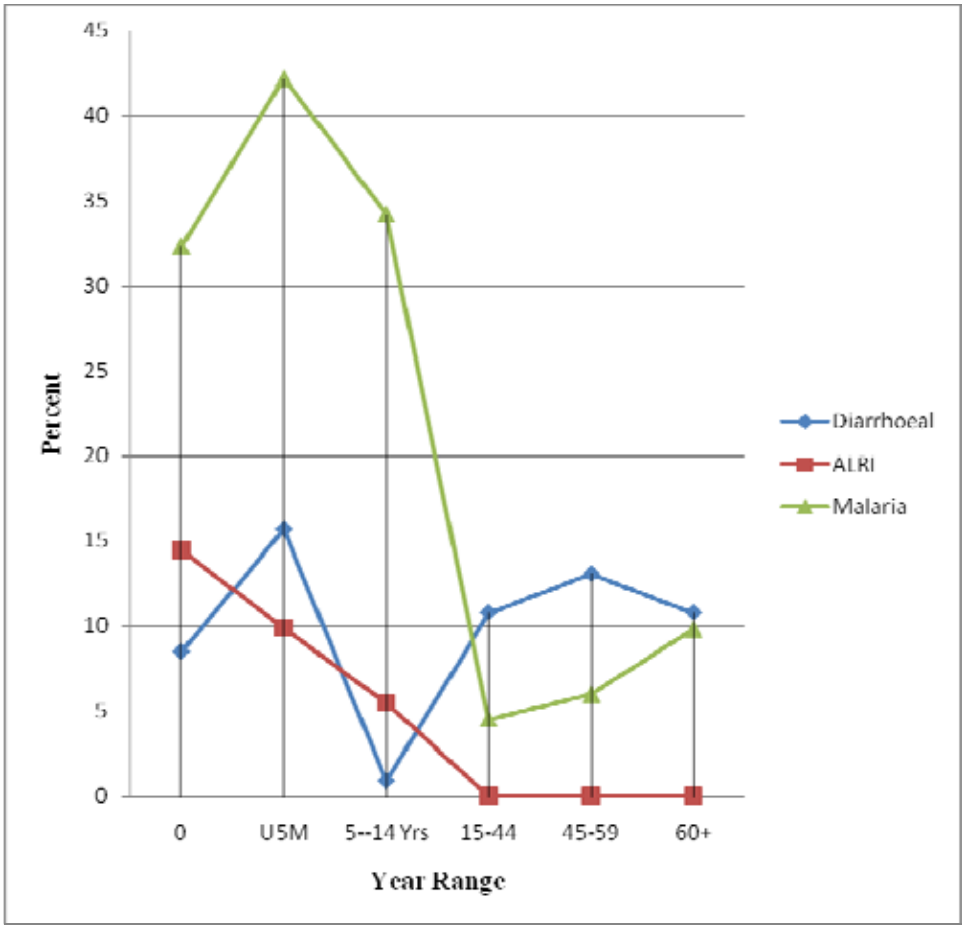

Figure 9: Age variations in diarrhoea, acute lower respiratory infections and malaria diseases

Source: NDSS Data Unit, 2009

Seasonal changes in causes of death

Seasonality of deaths reflects in peaks or declines during either hot or cold weather conditions. Continuous search for reasons why certain diseases are common in cold and warm seasons have yielded positive results in many studies. For infectious and parasitic diseases, the evidence could sometimes be straightforward. For instance, malaria parasites density increases during the rainy seasons. Respiratory diseases and infections are common during cold and hot seasons. Similarly, cardiovascular deaths increase during cold periods as blood clotting is easily facilitated by low temperatures (Gemmel et al.,

2000). However, even these are not conclusive. Canada for instance has one of the coldest climates, yet mortality in winter has been consistently low (Gemmel et al., 2000). Specific causes of death are compared with seasons to check for existence of seasonal fluctuations.

The results are presented in Table 14. Infectious and parasitic diseases (40.6%), which are in Group I, dominated deaths throughout the year as all other non–communicable diseases represented 9.4 percent in the first quarter. Pneumonia and ALRI were also common during the first quarters. In the second quarters, infectious and parasitic diseases dominated (37.4%). The commonest, Pneumonia and ALRI during the first quarters may be explained by the conditions experienced between January and March. Weather situations during this period are usually a mix of warm and somewhat cold conditions, which influences the onset and transmission of respiratory infectious diseases.

Injury–induced deaths (6.2%) were quite high between April and June, compared to the first quarter (4.8%). The second quarter coincides with the beginning of crop cultivation in the district which also signifies mass return of seasonal migrants from the middle and southern belts of the country. These seasonal migrants return with improved means of transportation which are mainly motor bicycle and bicycles (A. Tanle, personal communication, April 29, 2010). The high rate of use of these means of commuting could manipulate the frequency of injuries in the study area.

From July to September, mortality was mainly due to malaria, acute febrile, gastro–enteritis and the other infectious and parasitic diseases. Respiratory infections (pneumonia and ALRI) on their own prompted 7.8 percent of total deaths in the third quarter. During the fourth quarter (October

to December) 42.1 percent of all deaths originated from infectious and parasitic diseases. Respiratory infections (11.2%) were the next leading cause of death for the period. Cardiovascular deaths did not show any massive departure in pattern as it was 2.1 percent of deaths in first and second quarters and 2.0 percent of mortalities in third and fourth quarters.

Table 14: Seasonal differentials of causes of death

COD	Season (%)					N
	Jan–Mar	Apr–Jun	Jul–Sept	Oct–Dec	Total	
Infectious and Par	40.6	37.4	44.8	42.1	41.5	4646
Maternal	0.7	1.1	0.6	0.5	0.7	76
Cancers	0.3	0.4	0.4	0.3	0.3	38
CNS	0.6	0.3	0.5	0.1	0.4	41
Cardiovascular	2.1	2.1	2.0	2.0	2.0	226
Respiratory	0.2	0.0	0.1	0.1	0.1	13
Digestive Dis	2.2	3.3	2.1	1.7	2.3	255
Injuries	4.8	6.2	4.2	4.1	4.7	530
Genito–Urinary	0.5	1.1	1.0	1.0	0.9	101
AOCD	9.4	9.6	8.9	8.5	9.0	1013
AONCD	7.4	8.5	6.6	7.1	7.3	821
Childhood Clus.	2.1	1.5	0.3	1.5	1.3	148
Peri–natal	2.3	1.8	1.7	1.5	1.8	204
Nutritional Def	2.8	3.6	4.9	4.9	4.1	460
Respiratory Infect	9.1	6.1	7.8	11.2	8.7	978
Undetermined	15.0	17.1	13.9	13.5	14.7	1644
Total	100.0	100.0	100.0	100.0	100.0	
N	2665	2278	3021	3230		11194

Source: NDSS Data Unit, 2009

Literature on mortality in relation to seasons suggests that extreme cold weather patterns increases the tendencies of cardiovascular deaths. This high risk has been linked to conditions such as blood clotting and other biological factors. This known and accepted trend was not clearly observed from the data. It is very plausible that minimal numbers of cardiovascular deaths in the district may have blurred this pattern. Large data sets with high incidence of cardiovascular deaths would be needed to verify this assumption. For a disease like respiratory infections, however, the most readily available reason for their high occurrence between October and December may be matched to the onset of the dry weather, which is relatively severely cold in late November through December to early January in the district. This is evident (Table 14) as the contribution of respiratory infections was 9.1 percent during the first quarters. In biological reasoning for instance, respiratory diseases risks are induced by two routes. One, low temperatures facilitate the survival of bacteria in droplets and two, cold has adverse effects on the immune system's resistance against respiratory infections. As a result from breathing cold air, the risk for a pulmonary infection rises due to bronchoconstriction (Maud, Huynen, Dieneke, Matty & Kunst, 2001).

Summary

The pioneers of public health discipline suggested interventions to governments and city managers on public health issues, bearing in mind the influences that the environment has on health and well–being. Following this tradition, the study identifies causes of death which can be used as basis for public health interventions. In terms of space, causes of death varied by zone:

the southern zone had higher deaths than the other four (north, east, west and central). The contribution of various disease groups to mortality in each of the zones differed. For instance, whereas cardiovascular was an issue of concern in the central zone (3.6%), it was neither an issue in the northern nor southern zones (1.8%). On the other hand, infectious and parasitic diseases were of concern in all the zones: central (41.9%), east (43.6%), north (43.5%), south (40%) and (39.6%).

Another relevant issue that emerges from this chapter is the seasonality of causes of death. Although infectious and parasitic diseases occurred throughout the year, their impacts were felt strongly in the second half of the year: 45 percent of deaths occurred between July and September, mainly from infectious and parasitic diseases. Deaths from infectious and parasitic diseases were higher, accounting for 42 percent of deaths. For the period, 42 percent of deaths occurred between October and December. The end of raining season and the beginning of the dry season serve as buffer for both rainfall and cold weather accompanied deaths.

Furthermore, it appears overall mortality is shifting from infectious diseases towards non–communicable diseases. This is evidenced by the proportion of diseases in the cardiovascular domain increased gradually from 1995 to 1999. If the pattern continues, the proportion of infectious diseases such malaria, diarrhoea, TB and other infectious diseases could be expected to decline in the coming years. In general, infectious diseases are associated with under development and this is observed in the data. However, non–communicable chronic diseases are slowly but surely becoming relevant in public health discourse (Boerma, 2008). It is therefore argued that, attention

towards optimum health for residents of rural districts need to focus on both communicable diseases and non–communicable diseases.

CHAPTER SIX

SUMMARY OF FINDINGS, CONCLUSIONS AND RECOMMENDATIONS

Introduction

Finding practical means of ensuring long and quality life of people has and will continue to engage the attention of policy makers. Researching into past and present trends of public health issues is one of the important means of making inputs to policies targeted at achieving prolonged and quality life (Caldwell, 2001; Berridge & Gorsky, 2004). This notwithstanding, developing countries are faced with daunting tasks of achieving this universal priority of man. Partly, this has come about due to institutional weaknesses in generating quality data that can be used to monitor progress and challenges in programmes aimed at providing quality health. To improve quality of life with fewer diseases, several approaches have been used. The sources of declines achieved as observed in the literature review were through multiplicity of approaches.

This study used data from NDSS based on VA from a surveillance system in the KND since 1993. The data from the site were used to examine the structure of mortality and causes of these deaths from 1995 to 1999 with the main objective of evaluating mortality transition processes in the district, using Omran's (1971) epidemiological transition theory as a reference point.

Summary of findings

Based on the specific objectives of the study, the key findings are summarised as follows:

- **Annual deaths**

On annual basis, it was found out that death toll for 1996 was high, (22.6%) compared to other years. At the end of 1999, mortality had declined by about 20.3 percent from 1996. This was also collaborated by the crude mortality rate per 1000 population. Two thirds of causes of death were due to communicable, maternal, prenatal and nutritional related diseases. Non–communicable diseases were the second prevalent causes of death, comprising 13.4 percent followed by injuries, 4.7 percent (intentional and unintentional) while unknown deaths constituted the remainder. The incidence of Group I diseases showed some dynamics after 1996, even though Group I diseases remained dominant. The contribution of Group I diseases to overall mortality was relatively high up to 1996 and stabilised minimally at a decreasing rate between 1997 and 1998 and declined in 1999 (Appendix 5). Injuries followed the patterns observed in non–communicable diseases, reducing steadily from 1995 to 1997 but increased in 1998 and 1999.

- **Spatial patterns mortality and the causes**

Mortality overall was higher in the southern part, accounting for thirty percent of total deaths which occurred. The disease–specific deaths did not differ much because of prevailing diseases that caused the majority of deaths in the zone. In all the zones, communicable, maternal, prenatal and nutritional

deficiencies were widespread. Considered individually, malaria was the main cause of death. The contribution of non–communicable diseases to mortality in central was 18.3 percent, 14 percent in east, 11.4 percent in north, 13.8 percent in the south and 13.2 percent in the west. Within the non–communicable specific diseases identified, cardiovascular was highest in the central zone, the only urban zone in the district. Therefore, risk factors associated with cardiovascular mortality, which includes income, and sedentary lifestyles seem to be emerging in the central zone of the district.

- **Seasonal patterns**

Peaks in morbidity were observed within the second half of the year, except for 1997 where the highest ever registered was in the first half of the year. Mortality among the 5 to 14 year age group was high in January to March. Among the 15 to 59 year olds, no clear seasonal trend was noticed since deaths occurred all year round. There were no sex differentials in month of death. Infectious and parasitic diseases were recorded throughout the year, giving the impression of an area endemic with infectious and parasitic diseases. All other diseases almost alternated with seasons. Malaria was highest in the first, third and fourth quarters. Diarrhoea was severe during the second quarter, coinciding with the commencement of the raining season.

- **Demographic patterns**

Two variables extensively used in natural population dynamics are age and sex. They were considered in relation to annual and cause of death trends. The proportion of deaths over the period was higher for males than females

generally. For certain diseases, septicaemia was mostly common among males under–five years while injury deaths were mostly among males than females, especially, between 15 and 44 years which were consistent with WHO (2007) assertion of increasing injuries among young male adults. Diarrhoea, malaria and malnutrition deaths were higher among females than males. Reported AIDS deaths were higher among females. The range of ages employed for analysis demonstrated some differences: for all age groups, diseases in Group I were the highest. The specifics however differed: malaria, ALRI and diarrhoea were higher in neonates and infants wile infectious and parasitic infections were higher among adults 60 years and above. In all, infectious and parasitic deaths were higher among sixty years old, defying existing knowledge of high prevalence of infectious diseases among young people. The study revealed that attention should be given to child and infant health as well as among adults given the pattern of deaths by age.

Conclusions

The general trend observed in both the structure and causes of mortality in Kassena–Nankana over the five–year period showed declining trend. By 1999, deaths in the district had reduced by about 20 percent over total deaths recorded in 1996. Nonetheless, there were some variations with respect to age, sex, zone and seasons. Mortality appeared to be tilting towards more deaths among adult, especially those 60 years above. Overall, females had higher advantages in terms of survival, and the differences noted were significant with age of a respondent at the time of death.

Spatially, chances of death as well as the causes differed by zone. The southern part of the district showed consistent lag in mortality compared to the other zones, and could reflect unfavourable social, material, demographic and ecological inequalities.

With respect to seasons, mortality, mainly infectious and parasitic diseases, showed responsiveness to changes in climatic conditions. Diseases such as malaria and acute lower respiratory infectious diseases peaked during the raining and dry seasons respectively. Mortality from injuries peaked towards the end of the first and the beginning of the second quarters over the study period.

Finally, the study used Omran's epidemiological transition model as a framework for analysis. Results showed that infectious diseases though receding were still the main causes of death within KND. However, non–communicable degenerative diseases, injuries and violent deaths (hybristic) were emerging as causes of death. From the analysis, KND has displayed the probability of rising injuries accompanying increasing chronic diseases as in the literature. This notwithstanding, the gap between infectious and chronic diseases was too wide to expect any massive outpacing by chronic diseases any time soon. In situating KND within the confines of the theory, it can be said that, using crude deaths, the district could be placed in the third stage of the theory. However, for burden of disease, the area can be placed within the second stage where infectious diseases determine the course of mortality. These observations bring out a dimension of the theory not identified in the literature: Omran used crude death rate as an indication of improving or deteriorating health. Using crude death rate, the nature of change appears to be

exaggerated compared to the use of burden of disease measure as an index for identifying stages in the model. Both expectancy of life at birth and crude death rates are functions of the total population structure of a country. In this case, the burden of disease approach provides a better index for the state of health in the study area.

Recommendations

The findings from the study have policy implications. First, the gradually declining infectious and parasitic diseases but rising non–communicable chronic diseases indicate that, if similar interventions like insecticide treated bed net, vitamin A supplementation and Community Health and Family Planning (CHFP) at KND are provided in similar poverty endemic areas, then the pace of mortality transition in Ghana may be enhanced much more than the current state which is directed by certain (malaria and diarrhoea particularly) infectious diseases. Pence, Nyarko, Phillips and Debpuur (2007) evaluated KND CHFP project and reported 14 percent reduction in under–five mortality after the first five years. This will help Ghana achieve the health–related MDGs within the specified period of 2015. This will require collaborative efforts between Ministry of Health/Ghana Health Service and the various District Assemblies in the country. The Ministry of Health and Ghana Health Service therefore have to re–invigorate the Primary Health Care concept that is more participatory and provides a more sense of ownership to beneficiary communities.

Similarly, the provision of health promoting facilities by the district assembly (e.g. potable water) should be distributed proportionally among

communities, taking into consideration population size and distance of communities from one another. This draws on health needs assessment as suggested in the population health literature. In this study, context (place of residence) has been found to be relevant, as suggested by Krieger (2005).

Public health education campaigns by Ghana Health Service and other health–related Non–governmental organisations like Pathfinder International which operates in the district would have to include information on seasonality of diseases. This will help communities to prepare adequately to respond to seasons that are characterised by specific diseases. Ayaga and Binka (2005) estimated that about six years of life would have been added to life expectancy in the district 1995 if malaria were eliminated in this hyper–endemic environment. This brings out clearly the need to tackle public health preparedness.

Ultimately, the Ministry of Local Government and Rural Development and its decentralised organisations have to be more interested in rural sanitation as well as being tough in implementing laws on sanitation. For instance, a strict implementation of the National Environmental Action Plan (1991) and National Environmental Sanitation Policy of 1999 would be in order (Republic of Ghana, 1991; 1999).

Avenues for further research

This study was descriptive using surveillance mortality data from KND between 1995 and 1999. As a result, the following areas are suggested: Risk factor(s) analysis could also be done to validate the suppositions made here such as distance to nearest health facility, access to roads, housing and

income, and verbal autopsy and “gold standard” (hospital) could be merged to track some of the undetermined/unknown cases.

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Appendix 1: Annual ungrouped causes of mortality

Disease	Year of Observation (%)						
	1995	1996	1997	1998	1999	Total	N
AIDS	0.7	0.8	0.2	1.0	1.1	0.8	85
ALRI	4.3	6.1	4.4	3.8	4.6	4.7	526
AOCD	9.9	9.0	9.2	9.8	7.2	9.0	1011
AONCD	7.9	6.0	6.8	7.3	11.4	7.8	868
Abortion	0.2	0.0	0.0	0.0	0.1	0.1	9
Acute Abdominal	3.0	1.3	2.3	2.3	1.9	2.1	238
Acute febrile	2.8	3.4	2.9	5.9	4.5	3.8	429
Anaemia	2.5	4.0	3.7	3.3	2.8	3.3	367
Birth Injury	0.4	0.2	0.1	0.1	0.6	0.3	32
COPD	0.1	0.2	0.1	0.1	0.0	0.1	13
Cardiovascular	1.9	1.3	1.6	2.4	3.1	2.0	226
Diarrhoea	10.0	10.3	11.0	11.0	14.9	11.4	1271
Hepatitis	0.1	0.4	0.2	0.4	0.3	0.3	30
Injuries/accidents	4.8	4.7	3.0	3.7	4.5	4.1	463
Intentional Injury	0.3	0.2	0.3	0.7	0.6	0.4	45
Liver cirrhosis	0.0	0.2	0.0	.2	0.2	0.2	17
Malaria	17.2	20.1	23.2	20.1	14.0	19.1	2137
Cancers	0.2	0.4	0.1	0.3	0.7	0.3	38
Malnutrition	1.2	0.9	0.6	0.6	0.8	0.8	93
Maternal	0.6	0.4	0.6	0.8	0.6	0.6	67
Measles	3.0	2.8	0.3	0.0	0.0	1.3	146
Meningitis	0.9	1.0	2.8	1.1	0.9	1.4	152
Neonatal jaundice	0.5	0.1	0.0	0.1	0.0	0.2	17
Neuro–psychiatric	0.3	0.1	0.1	0.5	0.8	0.4	41
Pneumonia	3.5	4.1	3.1	4.1	5.5	4.0	452
Prematurity	1.7	1.5	1.2	1.2	1.2	1.4	155
Renal disorders	0.9	0.6	0.6	1.2	1.3	0.9	101
Septicaemia	1.8	1.6	1.2	1.4	0.8	1.4	154
TB	1.8	1.5	2.2	2.7	2.2	2.1	230
Unknown	17.5	16.7	18.1	13.7	13.1	15.9	1781
Total	2198	2532	2317	2137	2010		11194
N	100.0	100.0	100.0	100.0	100.0	100.0	

Source: NDSS Data Unit, 2009

Appendix 2: Actual causes of death by age

Disease	Age Intervals (%)							
	0	1–4	5–14	15–44	45–59	60+	Total	N
AIDS	0.0	0.0	0.0	4.3	0.5	0.2	0.8	85
ALRI	14.5	9.9	5.5	0.0	0.0	0.0	4.7	526
AOCD	0.0	0.0	2.4	12.3	15.6	15.9	9.0	1011
AONCD	0.0	0.0	1.6	11.3	13.3	13.5	7.8	868
Abortion	0.0	0.0	0.0	0.6	0.0	0.0	0.1	9
Acute Abdo	0.0	0.0	0.3	3.6	3.2	3.8	2.1	238
Acute febrile	0.0	0.0	1.0	4.6	6.1	7.3	3.8	429
Anaemia	3.5	8.3	4.1	2.0	2.8	1.4	3.3	367
Birth Injury	1.4	0.0	0.0	0.0	0.0	0.0	0.3	32
COPD	0.0	0.0	0.1	0.1	0.3	0.2	0.1	13
Cardiovascula	0.0	0.0	0.0	1.5	3.0	4.6	2.0	226
Diarrhoea	8.5	15.7	0.9	10.8	13.1	10.8	11.4	1271
Hepatitis	0.0	0.0	0.3	0.8	0.5	0.2	0.3	30
Untentional In	4.2	4.1	11.4	7.8	3.2	1.3	4.1	463
Intentional Inj	0.0	0.0	0.1	2.2	0.4	0.1	0.4	45
Liver cirrhosis	0.0	0.0	0.0	0.4	0.4	0.1	0.2	17
Malaria	32.3	42.2	34.2	4.5	6.0	9.8	19.1	2137
Cancers	0.0	0.0	0.0	0.6	1.0	0.3	0.3	38
Malnutrition	1.9	2.4	1.7	0.0	0.0	0.0	0.8	93
Maternal	0.0	0.0	0.0	3.9	0.3	0.0	0.6	67
Measles	1.4	5.5	4.0	0.0	0.0	0.0	1.3	146
Meningitis	0.4	1.3	5.4	2.7	1.3	0.6	1.4	152
Neonatal jua	0.7	0.0	0.0	0.0	0.0	0.0	0.2	17
Neuro–psychi	0.0	0.0	0.1	1.4	0.3	0.4	0.4	41
Pneumonia	0.0	0.0	0.3	4.0	7.0	8.0	4.0	452
Prematurity	6.7	0.1	0.0	0.0	0.0	0.0	1.4	155
Renal dis.	0.0	0.0	0.4	1.9	1.1	1.5	0.9	101
Septicaemia	5.6	1.0	1.4	0.0	0.0	0.0	1.4	154
TB	0.0	0.0	0.0	2.3	4.6	3.4	2.1	230
Unknown	18.8	9.6	15.6	16.3	16.1	16.7	15.9	1781
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	
N	2283	1570	704	1573	1873	3191		11194

Source: NDSS Data Unit, 2009

Appendix 3: Actual causes of death by sex

Disease	Sex (%)			
	Female	Male	Total	N
AIDS	0.9	0.6	0.8	85
ALRI	4.7	4.7	4.7	526
AOCD	9.6	8.5	9.0	1011
AONCD	7.6	7.9	7.8	868
Abortion	0.2	0.0	0.1	9
Acute Abdominal	1.6	2.7	2.1	238
Acute febrile	4.3	3.4	3.8	429
Anaemia	3.6	3.0	3.3	367
Birth Injury	0.3	0.3	0.3	32
COPD	0.1	0.1	0.1	13
Cardiovascular	2.1	1.9	2.0	226
Diarrhoea	12.1	10.6	11.4	1271
Hepatitis	0.2	0.4	0.3	30
Injuries/accidents	3.2	5.1	4.1	463
Intentional Injury	0.2	0.6	0.4	45
Liver cirrhosis	0.1	0.2	0.2	17
Malaria	19.6	18.6	19.1	2137
Cancers	0.4	0.2	0.3	38
Malnutrition	0.9	0.8	0.8	93
Maternal	1.2	0.0	0.6	67
Measles	1.4	1.3	1.3	146
Meningitis	1.2	1.5	1.4	152
Neonatal jaundice	0.2	0.1	0.2	17
Neuro–psychiatric	0.3	0.4	0.4	41
Pneumonia	4.0	4.1	4.0	452
Prematurity	1.4	1.4	1.4	155
Renal disorders	0.4	1.4	0.9	101
Septicaemia	1.2	1.6	1.4	154
TB	1.6	2.5	2.1	230
Unknown	15.6	16.2	15.9	1781
Total	100.0	100.0	100.0	
N	5459	5735		11194

Source: NDSS Data Unit, 2009

Appendix 4: Quarterly causes of death movement

Causes of deaths	Quarters (%)					N
	Jan–Mar	Apl–Jun	Jul–Sept	Oct–Dec	Total	
AIDS	0.8	0.7	0.6	1.0	.8	85
ALRI	4.2	2.8	4.3	6.8	4.7	526
AOCD	9.4	9.8	9.0	8.1	9.0	1011
AONCD	7.5	9.3	7.0	7.6	7.8	868
Abortion	0.1	0.1	0.0	0.1	0.1	9
Acute Abdominal Dis	2.1	3.1	2.0	1.6	2.1	238
Acute febrile illness	5.2	4.0	3.3	3.0	3.8	429
Anaemia	2.1	2.7	3.9	4.0	3.3	367
Birth Injury	0.4	0.4	0.1	0.3	0.3	32
COPD	0.2	0.0	0.1	0.1	0.1	13
Cardiovascular	2.1	2.1	2.0	2.0	2.0	226
Diarrhoea	8.8	10.4	14.7	11.1	11.4	1271
Hepatitis	0.3	0.5	0.3	0.1	0.3	30
Injuries/accidents	4.2	5.4	3.9	3.4	4.1	463
Intentional Injuries	0.5	0.5	0.2	0.4	0.4	45
Liver cirrhosis	0.2	0.3	0.1	0.1	0.2	17
Malaria	18.2	14.8	21.0	21.1	19.1	2137
Cancers	0.3	0.4	0.4	0.3	0.3	38
Malnutrition	0.7	0.8	1.0	0.8	0.8	93
Maternal	0.6	0.9	0.6	0.4	0.6	67
Measles	2.1	1.5	0.3	1.5	1.3	146
Meningitis	2.6	1.7	0.6	0.8	1.4	152
Neonatal jaundice	0.1	0.2	0.2	0.1	0.2	17
CNS	0.6	0.3	0.5	0.1	0.4	41
Pneumonia	4.9	3.3	3.5	4.4	4.0	452
Prematurity	1.8	1.2	1.4	1.1	1.4	155
Renal disorders	0.5	1.1	1.0	1.0	0.9	101
Septicaemia	1.3	1.4	1.4	1.4	1.4	154
TB	2.3	2.1	1.7	2.2	2.1	230
Unknown	16.2	18.3	14.9	14.9	15.9	1781
N	2665	2278	3021	3230	11194	
Total	100.0	100.0	100.0	100.0	100.0	

Appendix 5: Yearly movement by 2000 GBD grouping

	Year of Observation (%)						
	1995	1996	1997	1998	1999	Total	N
Communicable, Maternal, Prenatal & Nutritional Causes	64.8	69.2	68.6	68.7	64.3	67.2	7525
Non–communicable Diseases	14.0	9.8	11.2	13.8	19.1	13.4	1495
Injuries	5.1	4.9	3.7	4.9	5.2	4.7	530
Unknown	16.2	16.2	16.5	12.6	11.4	14.7	1644
Total	100.0	100.0	100.0	100.0	100.0	100.0	
N	2198	2532	2317	2137	2010	11194	

Source: NDSS Data Unit, 2009

Appendix 6: Chi–Square Tests of sex and age at death

	Value	Df	Asymp. Sig. (2–sided)
Pearson Chi–Square	21.935	5	0.001
Likelihood Ratio	21.966	5	0.001
Linear–by–Linear Association	2.123	1	0.145
N of Valid Cases	11194		

Appendix 7: Chi–Square Tests of age and month of death

	Value	Df	Asymp. Sig. (2–sided)
Pearson Chi–Square	3.820E2	5	0.000
Likelihood Ratio	364.038	5	0.000
Linear–by–Linear Association	37.221	1	0.000
N of Valid Cases	11194		

Appendix 8: Chi–Square Tests of zone and cause of death

	Value	Df	Asymp. Sig. (2–sided)
Pearson Chi–Square	37.128	12	0.000
Likelihood Ratio	36.479	12	0.000
Linear–by–Linear Association	0.066	1	0.787
N of Valid	11194	1	

Appendix 9: Chi–Square Tests of sex and seasonality

	Value	Df	Asymp. Sig. (2–sided)
Pearson Chi–Square	20.115	11	0.044
Likelihood Ratio	20.127	11	0.044
Linear–by–Linear Association	0.708	1	0.400
N of Valid Cases	11194		

Appendix 11: Data release agreement form

Data Release Agreement
between
The Navrongo Health Research Centre
and
Joshua Amo-Adjei, University of Cape Coast

April 2009

This agreement is between the Navrongo Health Research Centre (NHRC), and Joshua Amo-Adjei, University of Cape Coast in the receipt and handling of data for the thesis project of the MPhil in Population and Health.

In accordance with the Collaboration Agreement between THE UNIVERSITY OF CAPE COAST, CAPE COAST (UCC) and THE NAVRONGO HEALTH RESEARCH CENTRE (NHRC), the NHRC agrees to provide data for Joshua Amo-Adjei to write his thesis project titled ***"Longitudinal analysis of morbidity and mortality in the Kassena-Nankana District: 1995-2000"*** as part of the requirements of the MPhil in Population and Health. A brief description of the data files in this agreement is attached.

The terms of the agreement are, as follows:

i) The NHRC, as the host agency for the research, remains the owner of data collected by the Centre, irrespective of who is assisting or analyzing data.
ii) Individuals analyzing NHRC data are not allowed to share data with third parties.
iii) Use of the data is limited only to the MPhil thesis project that forms part of the Field Attachment program.
iv) Data released for the MPhil thesis project remain the property of the NHRC and must be handed back to the NHRC at the end of the thesis work.
v) A copy of the thesis must be submitted to the NHRC following acceptance by University of Cape Coast
vi) Any publication from the thesis must be approved by the NHRC

By signing this document, I agree to use the data only for the purposes of the MPhil thesis project, and according to the terms stated above. I further agree that I shall work on the data myself, and that no other person(s) shall have access to the data.

NHRC data remains the property of the Centre. The NHRC reserves the right to withhold publication of any material based on the data in this agreement.-

Particulars of Person Requesting Data	**Particulars of Person Releasing Data**
Name: JOSHUA AMO-ADJEI	**Name:** MARTIN ADJUIK
Address: DEPARTMENT OF POPULATION AND HEALTH, UNIVERSITY OF CAPE COAST	**Address:** NHRC, P.O. Box 114 NAVRONGO
Signature:	**Signature:**
Date: 16-04-2009	**Date:** 16/04/09