Chapter One: Background - Environment and Health

1.1 Environment and Health in Ecosystem Theory

Ecosystems have many desirable or beneficial attributes to humanity and their general importance is often measured in relation to the overall services they offer. The benefits humans derive from them may be direct or indirect depending on the nature of the ecosystem services. The services include food, fibre, wood fuel, medicines, and the intangibles such as water purification and other provisioning as well as regulatory functions. The dependence of humanity on ecosystem services for survival imposes external stresses or disturbances. For this reason, most of the pressures on ecosystems largely come as anthropogenic activities which influence biogeochemical, hydrological, and ecological processes from local to global scales [1]. These disturbances are not necessarily intended to be detrimental until they become so in magnitudes that can affect the structure and function of ecosystems adversely in an irreversible manner. Under such conditions, the pressures impair the innate ability of ecosystems to perform their natural self-recovery functions (ecosystem resilience), which if prolonged, may change the states and form of ecosystems irreversibly. Like all complex adaptive systems, the self-recovery ability of ecosystems may ultimately be compromised depending upon the magnitude, importance and duration of the external force of change (driver). Here the concept of ecosystem resilience emerges because different ecosystems are more adapted differently to different stressors [1-3]. If the change is such that ecosystems can no longer sustain their roles with respect to provisioning, regulation, support and cultural services, then the ecosystem function is said to be compromised. At that point, the ecosystem health would be adversely affected and thus jeopardize human well-being in all four dimensions (i.e. basic material for good life, health, security and good social relations). For example,
changes in ecosystem can significantly alter the amounts of food and fibres, fuel
wood, genetic resources, biochemical and natural medicine, ornamental resources and
fresh water, with significant impacts on food security and human survival.

1.2 Ecosystems and Human Wellbeing

Ecosystem health has both direct and indirect influences on human health through the
delivery of a variety of services for human basic needs and livelihood capabilities [4-
6]. The Millennium Ecosystem Assessment (MA) framework recognizes that through
their provisioning services, ecosystems affect human health in just a few predictable
casual-chains and in yet several unpredictable pathways by supplying food, fresh-
water (for many metabolic, physiological, and biochemical processes) fuel wood
for energy, fiber and many bio-chemicals for medicines [3, 6]. In addition, through
their regulatory services, ecosystems affect human health indirectly through their
moderation and regulatory effects on climate, disease, water, air and waste cleansing
[3].

These services support and promote human health functions by guaranteeing the
ability for adequate nourishment, ability to be free from avoidable diseases, ability to
have adequate and clean drinking water, ability to have clean air, the ability to have
energy to keep warm and cool as well as the ability to maintain clean physical
surroundings in homes [3, 7-10]. Deterioration in ecosystem function or health would
lead to a reduction in per capita amounts of their services, which may bring about a
decline in the overall human health status [3, 8, 11]. It is important to note that,
ecosystem damage almost always hits those living in poverty the hardest. The
overwhelming majority of those who die each year from air pollution are believed to
be poor people in developing countries [3, 11, 12].
1.3 Ecosystem Change

Ecosystems broadly defined as adaptive systems of both biotic and abiotic components of the natural environment represent an outcome of several years of a series of evolutionary processes of natural successions. While it is recognized that ecosystems are continuously undergoing natural change, much of the ecological change observed today is a product of the interaction between natural systems and humans [13].

Humans belong to a subset of the biotic component of the ecosystem and like all other biotic components; they depend on a variety of ecosystem services for survival needs, which aggregately translate into human well-being.

The interaction between humans and ecosystems continue to shape the ultimate integrity of ecosystem function in a variety of both predictable and unpredictable ways [1, 13]. The world’s present million or so species are the modern-day survivors of an estimated several billions species that have ever existed [13]. While ecosystem change and specie extinctions have occurred periodically in the geological time scale, the present rate of loss of the world’s biological diversity is at its greatest due to human influences since life began [8, 13]. This is the cause of the increasing global concerns about whether the ecosystem may still be able to support life on earth at the current scale of their exploitation.

Ample evidence exists to show that up to 25 percent of the earth’s total diversity is at serious risk of extinction during the next 20-30 years [8, 9, 13, 14].

While the past extinctions had been blamed on natural causes, today humans are largely responsible for the current ecological change and extinctions [8, 11, 13, 15]. Factors that cause changes in ecosystems and their services are called drivers. A driver therefore is any natural or human-induced factor that directly or indirectly
causes a change in ecosystem integrity [11, 15]. The MA conceptual framework recognizes two types of drivers: direct drivers, (drivers that unequivocally influence ecosystem processes and can be identified and measured to differing degrees of certainty) and indirect drivers (drivers that operate more diffusely, often by altering one or more direct drivers and their influences are established by understanding their effects on the direct drivers) [3, 16].

Different drivers have different effects on ecosystems both in temporal and spatial scales [1, 3, 9, 17]. These effects vary in importance, magnitude and strength [1, 3]. While some of the effects may be minor, others may be considerably profound or while some may have short-term stress, others tend to have long-term stress on ecosystem function. In many other cases while some of the effects of drivers on ecosystem function may be insignificant so as to be reparable, and/or reversible, the effects on ecosystem function of most known drivers are irreparable and irreversible [3, 18, 19].

Moreover, changes in ecosystem integrity can directly alter the abundance of human pathogens, example cholera and insect vectors such as mosquitoes, black fly and tse-tse-fly. The change in ecosystem state may also significantly alter the quality of air, which may directly alter the epidemiology of respiratory and coronary diseases in populations. Thus, the nature of the “wedlock” between ecosystems change and human health is dependent upon a complex interplay of a host of drivers that operate at different scales in space and time.
1.4 Scales of ecosystem change

‘Scale’ refers to the spatial extent, temporal duration or institutional level of the unit of analysis. It is expressed in units of length (or area), time, or social organization (e.g. individuals, households, communities etc.) [3].

Ecological change phenomena and processes have varying characteristics largely determined by the location of their occurrence, the driving forces, and their influences on other natural processes. They occur and operate at a wide variety of scales, geographically, temporally and institutionally [16, 20, 21]. These can range from very small change phenomena as a tree fall in a large rainforest, variation of waste disposition in urban spaces, etc., to very large ones like the replacement of a whole forest by human settlements [22]. The change processes may also vary in time-scales from very short period such as the temporary migration of birds to very long time-intervals as the process of desertification [16, 20, 23]. The characteristic spatial scales of ecological systems are largely set by factors such as the home range of individual mobile organisms, or the range of influence of sessile organisms in the case of living agents, the inducing agents and the area over which a disturbance occurs [24]. The scale of an ecosystem change may also be influenced by the distance over which a material bounded by the change is transported within its resident life as in the case of carbon dioxide which can be transported in its multi-year effective lifetime. On the contrary, the same wind fields can only transport tropospheric ozone a few hundred kilometres after which it is consumed by atmospheric reactions; thus its characteristic scale is regional.

In other instances, the temporal characteristic of the scale of an ecological change may be set by the lifespan of organisms, the turnover rate of material pools, and the average period between successive disturbances at a location [18, 19, 23]. An
important distinction, particularly for determining system resilience, lies in how ‘fast’ or ‘slow’ a change process progresses to a threshold of irreversibility typically relating to the quantum of the external driving force.

1.4.1 Global Scale
The spatial scale characteristics of any ecological change event are governed by the geographic spread of the drivers operating to cause the change and the nature of the ecosystem components involved in the process [1, 3, 18]. Often, ecological change phenomena are local which means they are location specific and operate about and within a given location [1, 18, 23]. The ecological importance of these changes lies in the magnitude of their impact on the ecosystem structure and function as well as on human health [24], although many of the change processes may have synergistic or cumulative effects, which may be experienced in a wider global scale. For example, the aggregate effect of local level carbon dioxide emissions in several locations around the globe is the large-scale ozone effect which causes global warming and climate change with concomitant heat related mortalities [16, 24-26]. Depending upon the magnitude of the change, some of the processes may be reversible and more often; many may not be reversible [1, 27]. While the effects of local change processes (i.e. carbon dioxide emissions) may be insignificant at the local scale and in the short-term, their long-term effects on humans at the global scale may be phenomenal [26].

1.4.2 Sub-global Scale
The cause, action and effects of an ecological change process may be confined to a given geographic boundary such as the clearing of land for farming, the construction of a football park, the sinking of a well, a volcanic eruption etc. Such change
processes are regarded as sub-global ecosystem change phenomena and are well recognised to affect local and regional populations in many different ways [1, 3, 27]. For example, the characteristic scale of an individual household in a freehold tenure system may be the area of land which it owns; for a community it may be a village or municipal boundary, and for a country it is the area included in the national borders and the exclusive economic zone over the ocean. Interactions between humans and ecosystems are most directly observable at local, micro or lower scales [3]. Examples of such direct human-ecosystem interactions include agriculture, forestry or land-use change, sanitation and access to clean drinking water and wastewater discharges [1]. The characteristics of some change processes may exist in more multiple scales. For instance, whereas climate change processes occur at both global and sub-global levels, such processes could be local in action where the actual changes implied are mainly determined by local changes in climate and the ecosystem’s response to it [1, 23]. Examples of such local scale or microclimate change phenomena include “heat islands”, waste accumulation and particulate plumes at traffic intersections in urban centers.

1.5 Ecosystem Change and health implications

Ecosystem change affects human health through a complex web of proximate causal chains, but the most direct and obvious human health and ecosystem change nexuses are water, air and the soil. Water is an important medium for human survival and through its lack and/or contamination or its pollution; severe ill-health consequences on humans can ensue [28-33]. Similarly, the contamination and pollution of the air through various dangerous gaseous emissions may also provoke profound ill-health concerns [34-36]. Equally imperative is the deposition of residuals and wastes (both
solid and liquid wastes) in the soil, which changes the microbial profile of the soil - a condition that may impact adversely on human health [37-39].

Regrettably, ecosystem damage almost always hits hardest, those living in poverty and who might not have been responsible for the damage. Evidence exists to show that the overwhelming majority of those who die each year from air and water pollution are poor people in developing countries [6, 40-44].

The major contaminants in water include toxic chemicals and minerals such as pesticides, heavy metals, and bacteria from human excrement, organic pollutants and suspended solids among others [45, 46]. The result of water contamination is water related and water based diseases such as diarrhoea, dysentery, intestinal worms, guinea worm and various debilitating skin conditions. Diarrhoea and dysentery account for an estimated 20 percent of the total burden of disease in developing countries and every year, polluted water causes nearly 2 billion cases of diarrhoea, resulting in the death of some 5 million people (including 3 million children) in these countries [12, 21, 44, 47-49]. Air pollution from industrial emissions, car exhaust and burning of wood fuels (e.g. coal, gasoline and wood fuel) at home kills more than 2.7 million people every year – mainly from respiratory damage, some cancers, and heart and lung disease [12, 21, 47, 48, 50-58]. Some ill-health conditions directly relate to sanitation infrastructure in both developed and developing countries [30, 59-63].

Domestic solid waste continues to increase worldwide in both absolute and per capita terms as incomes of people and consumption of goods and services increase [38, 46, 64, 65]. In cities of developing countries, an estimated 20-50 percent of domestic solid waste generated remains uncollected and much higher proportion occurs in urban slums where garbage lifting is limited because of lack of access routes for tipping-trucks [12, 39]. Poorly managed domestic solid waste is a major cause of
many mortalities and high morbidity in many large cities in developing countries [39, 66]. In many urban communities in developing countries where sanitation services are inadequate, waste heaps become mixed with excreta, contributing to outbreaks of and spread of sanitation related infectious diseases [60, 67-69]. Children in such communities are reported to be five times more likely to fall ill than their counterparts in high-income residences [39, 70, 71].

More generally, uncollected domestic waste is the most common cause of blocked urban drainage channels in Asian and African cities, thus increasing the risk of flooding and water-borne diseases, which affect the poorer populations living near domestic waste dumps [39, 71-76].

1.6 Vector ecology and infectious disease transmission

Changes in the ecosystem directly affect vector ecology and indirectly affect vector-borne disease transmission [77-80]. While some of the changes may favour sustained vector breeding, other change phenomena may tend to be less favourable for vector breeding and growth. In instances where the change processes favour vector breeding on sustainable basis; holoendemic disease transmission develops over time [81, 82]. On the contrary, if the change process does not favour vector survival, then vector resistance or adaptation strategies may begin to develop in those areas [83-85].

Understanding the ecological change processes and vector adaptation strategies is very crucial for the development of disease control measures through environmental management [12, 78, 86, 87]. For instance, malaria transmission is strongly associated with location [88]. Malaria is known to be highly prevalent around specific mosquito breeding sites and can normally be transmitted only within certain distances from the breeding sites. The range of dispersal is typically between a few hundred meters and a kilometre, but rarely exceeds 2-3 kilometers [88, 89]. Clustering of malaria is widely
reported and persons with clinical symptoms tend to cluster within small geographic ranges, usually at household and community levels [54]. In areas or clusters of low endemicity, the level of malaria risk or case incidence could vary widely between households because the specific characteristics of houses and their locations affect the contact dynamics between humans and vectors [88-90]. The exact nature of the influence of ecological change events on the patterns and dynamics of infections is highly dependent on human/vector interactions and area characteristics [77, 91-95]. For example, where endemicity is high, the effects of differences in human/vector contact rates on malaria case incidences in different households may be less pronounced [88-90, 96, 97]. This may be a consequence of the blurring of the proportional relationship between inoculation rates and case incidences by super-infection and exposure-acquired immunity [88, 98].

1.7 Urban Ecosystems

Urban systems form the matrix of urban ecology, and in terms of ecosystem services, they are primarily sites of consumption [27, 99-102]. They contrasts with the other systems such as cultivated systems (engineered or artificial ecosystems), dry-lands and coastal systems (natural ecosystems), which are primarily sites of production and harvesting of ecosystem services [3, 100, 103]. Understanding the ecosystems in urban areas will not only help with ecosystem management, environmental risk reduction, disease control and direction of planning resources, but also may help to deepen our understanding of how urban systems function more broadly to influence urban health [104]. Urban systems are not only characterized by a varied landscape, comprising a range of ecosystems and habitats, but are also generally viewed as a whole that comprises several mosaics of distinct community areas. Thus urban change
processes may be seen as the spatial heterogeneity of the different community (cluster) types or neighbourhood types, which may exert different degree of influences on urban health. Urban environments may also be conceived along demographic perspective which means a change process could be perceived in terms of population numbers as often observed with the transition from rural to urban areas. Typical rural-urban gradients include not only increasing human population density, and increasing shares of impermeable land cover, but also decreasing population density for many non-human species [31, 101, 105-109] as well as changing levels of species diversity. The dynamics of ecosystem change in and around urban centres are also influenced by a number of features characteristic of how urban landscapes change, such as a high rate of introduction of alien species (exotic insect vectors and rodents), high habitat diversity and fragmentation, and a high rate of (human-induced) habitat disturbance [110, 111].

Changes at very different scales often combine to create new challenges for humans. Urban development and trade, for example, enabled the epidemics that devastated Europe in the middle ages, and introduced large parts of the world to infectious diseases that were never encountered [112]. Indeed, urban researchers have long viewed urban systems not as individual settlements but as networks of urban centres connected by flows of capital, people, information and commodities regionally [113] making a careful study of the association between the spatially varied urban structure and urban health outcomes appropriately relevant.

1.7.1 Physico-chemical Component

The physico-chemical component of urban ecosystem includes the atmosphere and the chemicals contained therein. This implicitly is the surrounding air and its
constituent chemicals as well as particulate matter. Since the industrial revolution many countries have experienced sustained high levels of air pollution in their major cities and industrial prefectures resulting in the contamination of urban air by noxious gases and minute particles of both solid and liquid matter in concentrations that endanger health [58, 114-118]. The major sources of air pollution are transportation engines, power and heat generation, industrial processes, and the burning of fossil fuel and solid waste [42, 48, 57, 58, 119]. While in industrialized nations, a rapid rise of particulate and chemical concentrations in urban air are largely contributed by industrial activities, in developing countries, major contributors to increased deterioration in urban air quality are less of industrial activities, but more of increased use of fossil fuel and old and/or improperly maintained vehicular fleet [120, 121]. A study of transportation and its impact on environmental quality in Senegal, found that the health costs associated with vehicle emissions were among the factors costing that country the equivalent of five per cent of its Gross Domestic Product (GDP) [122]. In Northern Africa, cities in which refineries and coal power stations are sited tend to experience sulphur dioxide concentrations double that recommended by the World Health Organization [123]. In general, the number of motor vehicles over 30 years has nearly doubled in the past 10 to 15 years in East and West Africa. In Uganda for example, the number of registered vehicles has more than quadrupled since 1971 [122-125]. Older cars emit up to 20 times more gaseous pollutants than newer ones. In less than three decades, urban centers in Sub-Sahara Africa have doubled their gaseous emission loads which are reportedly responsible for the elevation in the prevalence of cardiovascular diseases in the region [122, 123]. A study conducted by the Clean Air Initiative in Cotonou in Benin to establish urban air concentration levels reported overwhelmingly high emission loads with carbon monoxide (CO)
concentration of 18 mg/Nm$^3$ at traffic intersections. The concentration levels for hydrocarbons (HCs) were far above 18 mg/Nm$^3$ [122]. A similar study conducted by the Ghana environmental protection agency (Ghana-EPA) and Ghana Health Service (GHS) to establish the baseline ambient air concentration for a policy switch from the use of leaded fuel to unleaded fuel showed unacceptably high levels of lead (Pb) and cadmium (Cd) in the blood of high exposure population group in Accra [122, 126]. Lead levels in soils, and air were abnormally above the WHO recommended limits with its concentration in the blood of 396 exposed subjects from 8 institutions [126].

1.7.2 Biological Component

Urbanism is implicitly modernism in many respects. Urbanisation, which therefore means a large-scale process of modernisation, results in considerable habitat destruction leading to a wanton replacement of original species by human populations and their activities. Urbanisation is a disruption of a natural ecosystem (reduction in specie diversity), which establishes a new couple-system between humans and their activities on the one hand and the indigenous species on the other hand. Although it is widely agreed that urbanisation leads to reduction in specie diversity, many studies that have been conducted on urban microbiology have reported high microbial diversity in urban settings. Studies conducted in many large cities with poor sanitation infrastructure have documented evidence of prolific diversification of microbial phylogeny and communities [127-129]. Uncollected garbage and solid waste dumps constitute breeding grounds for insect vectors and the development of many microbial communities [10].
1.7.3 Infrastructure and Built Environment

The built environment (BE) affects ecosystem functions, ecosystem services and the overall urban landscape. The artificially built structures affect airflows and thus exert some influence on urban air quality and human health. Urban areas have many attractive and beneficial influences to human well-being that may be attributable to the BE. Nevertheless, many conditions which are the products of the BE but injurious to humans also exist in the urban areas [130]. One important feature of BE is its ability to create micro-climate conditions in urban areas that differ markedly from the overall urban climate (e.g. urban heat islands) and the climate of peripheral rural areas. The network of urban roads has direct influence on urban vehicular volume, daily fuel consumption and emission levels [131]. Transportation, especially when based on weak transport policy could be a major cause of air pollution [122]. Air pollution has many adverse health effects, including obstructive pulmonary and cardiovascular diseases [132]. Transport systems based largely on the use of private cars rather than mass transits are a major contributor to local warming and to urban air quality degradation [33, 133]. Other adverse effects from car-based transport systems include the degradation of local ecosystems and urban landscape due to dust in the case of un-tarred urban roads, traffic accidents, and the stress and loss of productive time spent in traffic [28, 86, 125, 134]. Poorly designed urban areas aggravate many forms of human and environmental stresses in urban spaces. Urban road surfaces and crowded buildings are able to retain heat, which together with the loss of vegetation contribute to “heat islands” and exacerbate the adverse effect of particulate matter in urban air [36, 101, 134].
1.7.4 Housing

Housing type is a major determinant of the ultimate health status of residents in human settlements [36, 96]. This is because the nature of housing structure may create localised microclimate far different from the general neighbourhood climate [36, 135]. Buildings act as windshields and have great influence on airflow within settlements [36, 96, 136, 137]. They also trap solar energy in cities and may thus cause the elevation of local area temperatures [138]. Depending on the type of the local climate of an area, the architecture-induced microclimate may influence the course and distribution of health outcomes which completely differ from that of a scenario with a different architecture [139-141]. Once built, housing quality tends to determine the social class and composition of the residents who reside in the given neighbourhood [142, 143]. Large single-family homes will tend to perpetuate occupancy by the upper socioeconomic groups [139, 144-147]. Large high-rise apartment buildings with small dwelling units and modest amenities tend to perpetuate occupancy by lower income families [55, 136, 141, 146, 148, 149]. In many metropolitan areas, where building codes are strongly enforced, there is a tendency to construct housing stock that influences the composition of the occupants [144]. Therefore spatial segregation of urban populations to a large extent is determined by the interaction of a range of physical, structural and social factors which include socioeconomic status, race/ethnicity, configuration and quality of housing [150]. In a study that was conducted to determine the settlement and social adjustment patterns among female migrants in Dhaka, it was reported that migrants initially experienced extreme difficulties in locating affordable housing and shelter [151, 152]. The women did not have much choice and settled in highly congested
areas and that, relatives and friends provided a few months of start-up assistance in terms of shelter and food upon arrival [151].

1.7.5 Socioeconomic

Economic conditions are not quite obvious features of an urban center as its population size, and are less easy to define and measure [55, 141, 153-155]. However, urban populations tend to cluster in a manner that reveals distinctive social and economic structure in the urban landscape [136, 156, 157]. Urban residents of approximately the same income brackets tend to cluster together naturally [158-160]. For instance, while rich urban residents are able to afford well planned housing in relatively unpolluted urban areas, those who are poor are spatially segregated to highly polluted, congested and low-cost housing [159, 161]. Many studies have classified urban areas and the distribution of urban population using the World Bank’s classification of low, lower middle, upper middle, and high income quintiles [149, 162-164] and methods used in measuring socioeconomic status of residents have been based on household assets [159, 165]. Socioeconomic variables of urban population tend to affect individuals’ health seeking behaviour, which in turn affects their overall health status [160, 166, 167]. In such circumstances, poor individuals are unable to visit health centers due to financial difficulties and inability to pay treatment bills [168].

1.7.6 Social-cultural

Over the last five decades, urban areas have experienced an increased ethnic, cultural and social diversity as more ethnic groups have moved from the rural areas to urban centers [153, 169-172]. Urbanisation has played a major role in intensifying both ethnic and cultural heterogeneity in urban areas thus making both features of urban
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populations very important determinants of urban health [173-176]. Urban populations are ordered by the way residents from different social and ethnic groups settle and neighbourhoods appear to be homogenous in terms of the level of income, education and occupation of residents [149, 162, 163]. In urban areas in America, whether from enforced segregation or association by choice, black populations, Hispanics and other distinctive ethnic minorities tend to concentrate in different neighbourhoods from of those of native-born Americans [150]. Such ethnic and cultural diversification is a feature of all urban areas and a key determinant of urban health inequality [170, 177-180].

1.8 Health Consequence of Changing Urban Structure

The physical, economic, social and cultural characteristics of urban life all have great, but varying levels of influence on urban population health [134, 152, 181-183]. Urban ecology affects urban health through such processes as population clustering, changes in architectural structure and the physical environment as well as changes in social organization [184]. Additionally, health is affected by a mix of both biotic and abiotic factors such as the climate, air quality, population density, housing stock, the nature of economic and industrial activities, income distribution, transport systems and opportunity for leisure and recreation [141, 185-187]. Therefore, a structural change in anyone or more of these aspects of urban life has considerable implications for urban health [55, 70, 144, 145, 147, 188-190]. This is because the structure of the built environment (i.e. how closely dense or how sparse) tends to produce a particular urban microclimate, which may have significant influence on the diversity of urban microbial populations [77, 191-193]. This in turn may influence the trends of infectious and communicable disease transmission in urban settings [146].
Urban structural change may significantly influence the distribution of particulate matter as well as the emission composition in urban air [34, 42, 194, 195]. This can have profound implications in respect of the risk of cardiovascular diseases in urban settings [137, 145, 146, 188]. A longitudinal study of a migrant Kenyan low-blood pressure (BP) population living in an urban environment had significantly higher BPs than a cohort of matched, non-migrant controls [196]. While preliminary conclusions attributed these observations to selective migration, the BPs of 90 males studied prior to migration were almost identical to those found in the age-specific rural controls in the low-BP community from which they came (120.9/59.0 mm Hg vs 120.5/60.1 mm Hg) [196]. Indeed, on the basis of subsequent analysis of pre-migration data supported by other evidence from the Kenyan Luo Migrant Study, it was concluded that the higher BP levels of the Luo migrants were not due to selective migration, but rather, a consequence of environmental changes, including changes in electrolyte intake, which occurred rapidly after migration [55].

1.8.1 Income and employment
In all urban areas, residents may be grouped under one of two distinctive sub-categories – the formal and the informal sector urban economies, based on their general employment and income levels [197]. The overall urban political economy is an expression of the arithmetic sum of these two sub-categories built properly on classical economic theories that unite microeconomic and macroeconomic functions [198]. However, the urban informal sector remains a dominant sub-sector of the urban economy as urban population fast outpaces the formal sector job openings [197, 199, 200]. Many studies suggest that employment and income levels have large influence on resident’s health and the overall urban health outcomes [186, 201, 202]. The
nutritional status of residents is a function of household food holding capacity, which in turn is determined by household income status [203-207]. Recently conducted research works on urban nutrition have reported that malnutrition is widespread among low-income groups in urban centers in Africa [198, 203-209]. There is a growing number of low-income groups who are excluded from attending health care facilities due to financial barriers and the inability to afford treatment cost out-of-pocket in Ghana [178, 210-212].

1.9 Conclusion

Urbanisation is an urban ecological change and has been dramatic in low income countries during the last three decades. Much of this is due in part to high reproductive rates among urban adolescents, and partly because of rural population influx to the urban areas [197, 213-217]. Approximately, 3 billion of the world’s people now live in urban centers and population projections suggest that well over 70% of the global population will be found in urban areas by the year 2025 [33, 218]. These populations will be surrounded by poor urban environmental conditions which may cause health problems, especially in the low-income communities [39, 219]. In poor countries, these environmental problems would relate to inadequate provision of basic services and the lack of sanitation facilities including, inadequate potable water as well as poor housing conditions and living arrangements.

Often, large-scale migration from rural to urban areas drives deterioration of biophysical and socio-environmental conditions with complex public health consequences in the urban centers. Sheer population numbers far outstrip the existing public services and the physical infrastructure in the urban centres. However, because of inadequate municipal budgets, the city authorities are usually unable to provide the
basic services equally across urban areas leading to inequalities in environmental health. Large inequalities in urban environmental sanitation tend to be a feature of urban areas experiencing rapid urbanization.

Additionally, competing demands for urban lands for transportation, housing, industry, agriculture and sports increase the value of potential lands for housing and for residential purposes. As a result, low-income groups are compelled to build in areas with deplorable environmental conditions [70, 144, 147, 187, 190, 197, 210-212, 220-229]. In Ghana, despite massive evidence that rapid urbanization is associated with deterioration in urban environmental conditions, urban centers continue to expand rapidly without regard to planning and environmental regulations [39, 66]. The result is the proliferation of substandard structures which lack access routes for garbage removal leading to waste accumulation in residential areas. This has significant influence on vector breeding and disease transmission. There is evidence that the poor among urban residents suffer the worst from the effects of poor environmental conditions [39, 187, 203, 209, 216, 230]. However, no studies have been conducted to determine the level of association between malaria/diarrhoea mortality and the urban environmental quality conditions as well as how much worse off the poor are affected compared to their richer counterparts [22, 39, 140, 189, 231-233].

1.10 Problem Statement and Research Questions

There is a growing consensus that environmental change affects human health in many ways and contributes to a wide variety of diseases and premature deaths [6, 16, 54, 114, 139, 140, 189, 214, 233-239]. The cause of death data and information are
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hard to find in Africa and where they exist, they are hardly used in epidemiological analysis to inform health policy decisions. Although almost all the countries in Africa have national Vital Registration Systems (VRS) (i.e. Births and Deaths Registries) which record and report annual births and deaths, the reporting systems are still weak. Nevertheless, despite the obvious structural weakness, the data generated from the VRS when combined with urban environmental variables could generate very useful policy relevant information on environmental determinants of urban health in African megacities.

The environmental health of urban settlements in Africa remains one of the least understood both biophysically and socio-environmentally [39, 51, 180, 231, 233, 240-242]. By their transient nature, urban settlements in countries south of the Sahara have a reputation of high instability with unpredictable environmental changes which may fuel complicated human health outcomes. In Ghana, urban sanitation infrastructure is inadequate, largely open-drain type and mainly narrow drainage channels [39, 50, 121, 201, 231, 243-249]. In the urban centers, the limited sanitation services are not evenly distributed across urban space providing for wide environmental health inequalities, e.g. certain areas have more intense waste accumulation than others. Areas close to lagoons and other large surface water bodies are more prone to flooding and offer cheaper lands for residential purpose than those areas far away from these water bodies. The flood prone areas tend to attract low income groups and therefore tend to be home to residents with low socioeconomic status. Additionally, rapid waste accumulation in these areas offer good opportunities for insect vector breeding and high infectious disease transmission compared to areas where waste lifting is far more frequent. In general, the result of the combination of differing levels of sanitation services, waste collection, different housing arrangements and the effects
of water bodies provide for high heterogeneity in infectious disease (especially malaria and diarrhoea) transmission in the urban complexes. While high heterogeneity in transmission had been reported for malaria and diarrhoea in urban complexes, a key question in this study was, “could the observed urban malaria and diarrhoea mortalities show the same level of heterogeneity in the urban complexes in Ghana”? In addition, whereas neighbourhood urban environmental conditions have been reported to influence urban health, no studies have been conducted to describe how much each of the different environmental conditions and neighbourhood characteristics was contributing to the observed urban malaria and diarrhoea mortalities in Ghana. While there is a clear understanding of the relationship between the state of wastes disposal, pathogen load in storm water and outbreak of enteric diseases such as diarrhoea and cholera in urban areas surrounded by garbage fields, there are no reported studies which show the relationship between neighbourhood urban environmental conditions and the observed infectious disease (e.g. malaria and diarrhoea) mortalities in the urban complexes [250-252]. To this end, an important unresolved general question was to what extent was each urban environmental condition/neighbourhood characteristic currently contributing to the pattern of the observed urban malaria/diarrhoea mortality in Accra and how could this be reasonably determined given the limited availability of mortality data? Finally, how were the urban neighbourhood environmental quality conditions associated with the existing urban socioeconomic status (SES) (i.e. what is the precise nature of the association between the different area-based SES variables and the urban environmental conditions)? Solutions to the general questions would offer great opportunities for national disease control authorities to strengthen malaria and
diarrhoea control programs and intervention strategies in urban centers in rapidly urbanization areas in low income settings.

More specific research questions in this study included the following:

a) what were the age- and sex-specific malaria and diarrhoea mortality patterns in urban Accra and how are these associated (if any at all) with each of the environmental variables?

b) was there a difference in the way age-group specific malaria/diarrhoea mortality was associated with the environmental variables, e.g. which age-class was more susceptible and by how much?

c) what was the relationship between urban environmental conditions and the observed urban malaria and diarrhoea mortalities and what fraction of the observed mortalities were contributed by each environmental variable?

d) how were malaria and diarrhoea mortalities distributed in space across the city complex, given the wide inter-cluster variation in the environmental conditions and neighbourhood characteristics?

e) what was the precise nature of the association between the different area-based measures of SES and the urban environmental conditions?

f) what was the precise nature of the association between the different area-based measures of SES and the observed urban malaria and diarrhoea mortalities?

g) was there a difference in the levels of environmental health inequalities across urban socioeconomic landscape?

h) what amount of variability in urban neighbourhood environmental conditions could be explained by area-based socioeconomic factors?

and
i) did lagoons/large water bodies within urban spaces contribute to excess malaria/diarrhoea?

1.11 Aim of the study
The general goal of this study was to investigate, and describe the spatial characteristics of malaria/diarrhoea mortality in Accra in the hope of deepening our understanding of how urban environmental conditions/neighbourhood characteristics (census variables) contributed to the observed malaria/diarrhoea mortality in a large city in Africa.

1.12 Multiple Objectives of the Study
The ultimate objective of this study was to deepen our understanding of and inform policy on how urban structure was influencing the observed urban malaria and diarrhoea mortality in cities with poor water supply & sanitation, poor hygiene conditions and complex living arrangements. In order to answer the research questions raised, the study undertook the following tasks:

i. reviewed existing body of literature on urban health and mortalities determine what was already known and what gaps there were,

ii. pulled out and collected into database, routinely reported death events in Accra over 1998-2002 period and allocated the death events to the census clusters,

iii. obtained urban environmental data for Accra from the 2000 population census database,

iv. produced a digital map of Accra showing the 70 ‘Census Clusters’ or ‘Localities’ used in the 2000 Population and Housing Census,
v. mapped the pattern of spatial distribution of malaria and diarrhoea mortality,

vi. compared the risk of malaria/diarrhoea mortality in different census clusters,

vii. determined the kind of association between area-based SES conditions and the quality of neighbourhood urban environmental conditions,

viii. determined the amount of variability in urban neighbourhood environmental conditions that could be explained by area-based socioeconomic factors,

ix. assessed the levels of environmental health inequalities across urban socioeconomic landscape,

x. studied the spatial patterns of the malaria and diarrhoea mortalities in an urbanizing area with declining environmental quality and social services in a low income setting,

xi. compared the spatial patterns of the observed urban malaria and diarrhoea mortalities, and

xii. found out if there were differences in the quality of the neighbourhood urban environmental conditions across the different wealth quintiles.

1.13 Theoretical Framework

The focus of research in environmental health has always been on the debilitating effects of various environmental exposures such as toxic chemicals and dangerous radiation, emissions and biological as well as physical contaminants in the natural environment [16, 21, 57, 58, 114, 115, 117, 140, 148, 253-256]. It is now almost a tradition that every conceived research design on environmental determinants of health from onset almost always ascribes or attributes poor health to a complex of environmental agents [255, 256]. However, an important fact often remains that, some environmental exposures may have positive health outcomes [256]. Frumkin (2001)
in his article “beyond toxicity – human health and the natural environment”, found evidence from four aspects of the natural world: animals, plants, landscape and wilderness – to support this hypothesis. Through research, it is now well known that air pollution can cause pulmonary and respiratory diseases, that heavy metal contamination may cause neurotoxicity and that global climate change would likely fuel increased transmission of some infectious diseases [34, 131, 169, 223, 257-264]. For instance, small drains, swamps, puddles, etc may be associated with increase larval density and therefore high malaria transmission [265-267], while large surface water environments in general tend to be associated with low larval density and therefore low malaria transmission. It has been demonstrated that a high heterogeneity of transmission intensity was an important characteristic of urban malaria in a study conducted in Dar-es-Salaam, Tanzania [265]. Other studies have reported that high frequencies of flooding were also surrounded by environmental media which offered good breeding opportunities for microbial agents and pathogens [29, 268-270]. Incidentally, those areas were reportedly associated with high frequency of diarrhoeal diseases [62, 63, 271-273]. In the past, malaria was widely reported to be exclusively a rural illness, but it is now generally accepted that urbanization is associated with increasing breeding sites for both malaria vectors and diarrhoea pathogens [265]. Many recent studies have demonstrated that rapid urbanization in developing countries was contributing to development inequalities and differential provision of sanitation services which in turn provided for high heterogeneity of mosquito vector and diarrhoea pathogen transmission intensity in urban complexes [266, 267]. Conceptually, this study assumed that, a typical urban environment was broadly a spatially heterogeneous whole, consisting of many different neighbourhoods or clusters at differing levels of environmental quality conditions (e.g. sanitation,
hygiene, water contamination, etc), exhibiting high spatial heterogeneity for malaria/diarrhoea transmission. The proposed model, while it did not assume that every urban ecosystem configuration would necessarily convey negative health effects, it recognized a health function gradient from a state of positive health outcomes (e.g. good health or absence of diseases) coinciding with a healthy environmental state (improved hygiene, sanitation, greenery, etc conditions) composed of positive health determining factors. This as opposed to a negative health state (e.g. worse environmental performance – poor hygiene, sanitation and brownfields conditions) that coincided with an environmental state composed largely of negative health determinants, see box5 in Figure 1.1.

The simplified model in Figure 1.1 assumed that urbanization was a driver of many human pressures (box 1 in Figure 1.1) which interacted in a variety of complex ways with environmental media (box 2 in Figure 1.1) and human systems to either produce positive health outcomes or negative ones or both shown as box 3 in Figure 3.1. Some of the environmental media and human pressures included, land use cover change such as agriculture, horticulture, landscaping, mining, industry, commerce, transportation, housing, solid and liquid wastes generation, environmental protection and biodiversity conservation, disease control programmes, healthcare and health interventions, distribution of surface water bodies and surface water quality, potable water supply or its contamination among others. The model assumed that the environmental interactions produced outcomes that conferred beneficial attributes on human health and therefore drove health functions toward the positive state i.e. good health along the green arrows in Figure 1.1. On the contrary, the complex interactions could also produce outcomes that were inimical to human survival and therefore hypothesized to drive the human health functions towards the negative health state
along the red arrows, thus manifesting complex episodes of disease conditions whose terminal outcome was mortality or death. However, in reality, these interactions did not generate outcomes in this clear dichotomy, but rather, most of them tended to generate a mix of both beneficial and detrimental effects and the ultimate human health outcome depended on how the intervening variables such socio-economic factors interacted with the physical urban environmental conditions. For example, urban environmental protection initiatives such as an upgrade of slums would result in improvement of urban health status and move the health function along the green arrow from box3 to box4 (i.e. the absence of illness/disease).
Chapter One: Background - Environment and Health

3. Effects of Human-Environment Interactions
- Surface water conditions – lagoons/swamps
- Environmental quality condition
  - Water pollution/sanitation conditions
  - Cluster hygiene
  - Waste accumulation
  - Cluster housing/living arrangements, etc.

2. Environmental Media
- Ambient air medium
- Soil/land – medium, incl. piped water
- Drains/lagoons/swamps, etc.
- Environmental interventions

4. Good Health
- Disease absence
- +ve Health state

1. Human Pressures
- Pop. growth & urbanisation
- Urban agriculture
- Housing & construction
- Transportation
- Commerce/markets
- Health interventions

5. Vector & microbial transmission
- Mosquito breeding
  - Culex sp, Aedes sp (non-malaria sp).
  - Anopheles, sp
    - gambiae/funestus
    - arabeniensis/merus
    - culicifacies/stephensi, etc
- Microbial/pathogen contamination
  - Salmonella sp, Yersinia enterocolitica
  - E. coli, Shigella,
  Campylobacter
  - Staphylococcus, Bacilli, etc

6. Morbidity
- Disease presence
  - Malaria
  - Diarrhoea

7. Health Intervention
- Disease mgt
- Treatment
- Bednet use
- Microbicide use

8. Mortality
- Malaria deaths
- Diarrhoea deaths
- -ve Health state


Figure 1.1: Health and Ill-Health Continuum: A theoretical model
On the contrary, environmental deterioration, *e.g.* poor hygiene, poor sanitation, overcrowding and poorly constructed structures; poor neighbourhoods environmental conditions would drive the health function along the red arrow to ill-health (box6) and death (box8) from box3 through box5. Many studies have already demonstrated high level of association between box3 and box6 in both rural and urban settings. However, only limited information which shows the association between box3 and box6 in rural setting exists while there is no such information showing the nature of the link between box3 (environmental conditions) and box8 (mortality) in urban setting in Ghana.

Finally, the model assumed that between box6 and box8 laid an intervening condition, *e.g.* health interventions (disease management, therapy, etc – box7) with a potential to push the health function back to good health along the green arrow. This condition could act as a confounding factor or effect modifier in this study (see box9) depending on the interpretation of the socio-economic factors often tied to health intervention variables and existing health policy. In areas where the health policy required free treatment for malaria and diarrhoea as in the case for Ghana, this condition was considered an effect modifier as income levels tended to highly correlate with environmental quality conditions.

### 1.14 Major Hypothesis

The main hypothesis of this work was that, the burden of malarial and diarrhoeal mortalities disproportionately affected urban residents living in neighbourhoods with low provision of sanitation services and poor neighbourhood urban environmental conditions in greater intensity than residents in urban settings with better neighbourhood environmental quality conditions.
1.14.1 Subsidiary Hypothesis

The subsidiary hypotheses were:

I. that neighbourhood urban environmental conditions contributed to excess malaria and diarrhoea mortalities.

II. that malaria/diarrhoea mortality varied with cluster distances from lagoons within urban areas.

III. that urban living arrangements, housing structure and type of construction materials were likely to be associated with high malaria and diarrhoea mortalities.
Chapter Two: Literature Review

2.1 Introduction

The effect of ecosystem change on human wellbeing has been studied for several decades [2, 3, 6, 16, 44, 53, 57, 114, 140, 234, 274]. This has strengthened scientific consensus that ecosystem change affects human well-being in several ways and at multiple scales. For instance, the process of urbanization (urban ecological change) alters the mix of natural and artificial elements in the urban landscape and thus changes many attributes that affect urban environmental quality (e.g., air quality, water quality, air temperatures) and ecosystem function (e.g., nutrient cycling, soil properties, water purification), both of which affect urban health. More specifically, urbanization or urban change both in time and in space tends to alter many components of urban environments as follows:

- solid/liquid wastes accumulation due to poor waste collection services and lack of sanitation facilities
- sealing of urban land surfaces leading to reduction in infiltration and increased overland flows which increase the frequency of urban flood disasters
- poor urban water supply infrastructure, leading to intermittent interruptions in supply and widespread contamination of potable water
- urban air pollution (both indoor and outdoor air quality deterioration) through increased motorization and wood fuel consumption
- structural changes in urban economies produce socioeconomic inequalities and inequitable access to health services across different income groups which in turn produce urban health inequalities and finally
uneven distribution of urban environmental sanitation services across urban space produces different urban neighbourhood conditions of different environmental quality.

While strong scientific evidence exists to show that the causal chain between ecological change and human health is both complex and multi-dimensional, most past efforts to understand the linkage between urban change and human health have focused on single-variable to single-effect analysis [111, 187, 275].

At global level, the millennium ecosystem assessment, which aimed to provide deeper understanding of this subject, attempted to evaluate and quantify the various dimensions of environmental change consequence on human wellbeing in an integrated fashion [2, 10, 26, 27, 49, 259, 276-278].

At the national level, a few such assessments included the study on intra-urban morbidity differentials [213, 214, 231, 243] and the Accra study on household environmental problems in the Greater Accra Metropolitan Area (GAMA) [213]. The Accra study was part of a larger tri-city project in the south, covering Accra in Ghana, Jakarta in Indonesia and Sao Paulo in Brazil. Although this study touched in great detail, on nearly all aspects of urban environmental problems, the analysis of data and interpretation of results were done on a piecemeal basis [213, 231, 243, 279]. This unit-by-unit type of interpretation, failed to bring out the effects of synergistic interaction of the environmental variables and the contribution by each variable to the expressed urban morbidity. In the current study, it was envisaged that a more useful and holistic approach to more informative interpretation required the use of some physical or concrete tool, which combined quantitative measures of environmental variables and health outcomes.
In order to identify gaps in the subject area within the regional context, a literature review was conducted relying upon the following databases and resources: CabDirect, PubMed, African healthline, ELDIS, government reports from Ghana, Library catalogue and similar documents. A search in all the databases was conducted using search terms such as “malaria/diarrhoea and environment”, “urbanization”, “mortality”, “death”, “urban*”, “town*”, “city”, “cities”, “Africa”, “West Africa”, “Ghana”, “ecosystem change”, etc, in different combinations.

The search effort yielded 2814 and 2885 published materials for malaria and diarrhoea respectively from different parts of the world. When the search terms were further restricted successively from Africa through to Ghana, 32 and 13 published materials were found for malaria and diarrhoea respectively, see Table 2.1 below.

<table>
<thead>
<tr>
<th>Region</th>
<th>Number of Published Materials</th>
<th>Number of Published Materials</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Malaria</td>
<td>Diarrhoea</td>
</tr>
<tr>
<td>Global</td>
<td>2814</td>
<td>2885</td>
</tr>
<tr>
<td>Africa</td>
<td>1110</td>
<td>375</td>
</tr>
<tr>
<td>West Africa</td>
<td>112</td>
<td>30</td>
</tr>
<tr>
<td>Ghana</td>
<td>32</td>
<td>13</td>
</tr>
</tbody>
</table>

Therefore given the paucity of published materials on the subject in Ghana, other resources, e.g. catalogued library texts, government reports, Ghana Ministry of Health annual reports were included. After a scrutiny of the 32 published materials on malaria and 13 on diarrhoea, a selection based on 27 most relevant ones to this study (18 on malaria and 9 on diarrhoea) was made and summarized as follows.
## Table 2.2: Summary of Published Studies on Malaria and Diarrhoea in Ghana

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Title of study</th>
<th>Study objectives and methods</th>
<th>Main findings/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Kreuels, et al</td>
<td>2008</td>
<td>Spatial variation of malaria incidence in young children from a geographically homogeneous area with high endemicity</td>
<td>The spatial variation of malaria incidences and socioeconomic factors were assessed over 21 months, from January 2003 to September 2005, in 535 children from 9 villages of a small rural area with high <em>Plasmodium falciparum</em> transmission in Ghana. Household positions were mapped by use of a global positioning system, and the spatial effects on malaria rates were assessed by means of ecological analyses and bivariate Poisson regression controlling for possible confounding factors.</td>
<td>Malaria incidence was surprisingly heterogeneous between villages, and ecological analyses showed strong correlations with village area ($R^2 = 0.74$; $P = .003$) and population size ($R^2 = 0.68$; $P = .006$). Malaria risk was affected by a number of socioeconomic factors. Poisson regression showed an independent linear rate reduction with increasing distance between children's households and the fringe of the forest. The exact location of households in villages is an independent and important factor for the variation of malaria incidence in children from high-transmission areas.</td>
</tr>
<tr>
<td>2. Ahorlu, et al</td>
<td>2006</td>
<td>Socio-cultural determinants of treatment delay for childhood malaria in southern Ghana.</td>
<td>Socio-cultural determinants of timely appropriate treatment seeking for children under 5 years suspected of having a perceived malaria-related illness were studied to assess the determinants of delays in malaria treatment. Caretakers of children with suspected malaria were interviewed about illness-related experiences, meanings and behaviour in two endemic villages in southern Ghana.</td>
<td>Only 11% of children suspected of having a perceived malaria-related illness received timely appropriate treatment consistent with the Abuja target of treating malaria within 24 h of illness onset; 33% of children received appropriate treatment within 48 h. Reported perceived causes of phlegm predicted timely, appropriate treatment within 24 h of illness onset ($P = 0.04$) in a multivariate logistic regression model; playing on the ground ($P &lt; 0.01$) predicted such treatment within 48 h. Two categories of distress, paleness or shortage of blood ($P = 0.05$) and sweating profusely ($P = 0.03$), also predicted timely, appropriate treatment within 24 h in a multivariate logistic regression model. Knowing that mosquitoes transmit malaria was not associated with timely, appropriate help seeking for the children, even though such knowledge may promote personal protective measures, especially use of bednets. Patterns of distress and PC were related to timely, appropriate help seeking, but not as expected. Effects on health seeking of illness-related experience and meaning are complex, and explaining their role may strengthen interventions for childhood malaria.</td>
</tr>
<tr>
<td>3. Ronald, et al</td>
<td>2006</td>
<td>Malaria and anaemia among children in a high transmission area.</td>
<td>A cross-sectional house-to-house survey of <em>P. falciparum</em> parasitaemia, clinical malaria, anaemia, and anaemia due to malaria was conducted among 296 children from 184 households in a high transmission area in Ghana.</td>
<td>In total, 296 children were tested from 184 households. Prevalences of <em>P. falciparum</em>, clinical malaria, anaemia, and anaemia due to malaria were assessed. The study aimed to assess the impact of malaria on child health and well-being.</td>
</tr>
</tbody>
</table>
children in two communities of Kumasi, Ghana: a cross-sectional survey

Anthropometric indices, and intestinal helminths was conducted in April-May 2005. Data collection included child and household demographics, mosquito avoidance practices, distance to nearest health facility, child's travel history, symptoms, and anti-malarial use. Risk factors for P. falciparum and anaemia (Hb < 11 g/dl) were identified using generalized linear mixed models.

**Risk factors for P. falciparum**
- older age
- rural travel
- lower socioeconomic status

**Risk factors for anaemia**
- P. falciparum infection
- Moshie Zongo residence
- male sex
- younger age

Heterogeneities in malariometric indices between neighbouring Kumasi communities are consistent over time.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Objective</th>
<th>Method</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sama, et al.</td>
<td>2006</td>
<td>Age and seasonal variation in the transition rates and detectability of Plasmodium falciparum malaria</td>
<td>Objective: To assess the effect of acquired immunity on the duration of Plasmodium falciparum infections in order to understand the models of malaria transmission. Method: Use of a dynamic model for infection incidence, clearance and detection of multiple genotype P. falciparum infections and fitted model to a panel dataset from a longitudinal study in Northern Ghana</td>
<td>Models indicate that there is seasonal variation in the infection rate, and age dependence in detectability. Best fitting models had no age dependence in infection or clearance rates, suggesting that acquired immunity mainly affects detectability.</td>
</tr>
<tr>
<td>Rindsjo, et al.</td>
<td>2006</td>
<td>Presence of IgE cells in human placenta is independent of malaria infection or chorioamnionitis, in Sweden and Ghana</td>
<td>Objective: To investigate the role of IgE cells in human placenta in malaria infection foetal deaths. Method: Use of immunohistochemical staining pattern for IgE to assess placental IgE distribution and malaria infection/deaths in foetuses</td>
<td>No difference in the amount or distribution of IgE(+) cells between malaria-infected and non-infected placentas, nor between different degrees of chorioamnionitis. The IgE score in the placenta did not correlate with the levels of IgE in maternal serum or plasma. However, the IgE score was significantly higher in second- compared to third-trimester placentas (P = 0.03). Opinion held that a maturation time-point in the fetus and in the intrauterine environment during the second trimester, or it might be associated with the increased number of intrauterine fetal deaths in the second trimester.</td>
</tr>
<tr>
<td>Kobbe, et al.</td>
<td>2006</td>
<td>Seasonal variation and high multiplicity of first Plasmodium falciparum infections in children from a holoendemic area in Ghana, West</td>
<td>Objective: To assess the prevalence and multiplicity of Plasmodium falciparum infections in Ghanaian infants. Method: Typing of the genes encoding the merozoite surface proteins 1 and 2 (msp-1, msp-2) in 1069 three month-old infants over a recruitment period of one year in an area holoendemic for malaria in Ghana</td>
<td>The occurrence of early infections was dependent on the season (month-stratified prevalence 6.4-29.0%). Diversity of msp-alleles was extensive and significantly higher in the dry than in the rainy season. The level of infection prevalence and the high multiplicity of infections (median 4, maximum 14 strains per isolate) in the first months of life suggested early contacts with parasites exhibiting a wide repertoire of antigens and, most likely, multiple infections per single mosquito bite.</td>
</tr>
</tbody>
</table>
### Africa

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Title</th>
<th>Objective</th>
<th>Method</th>
<th>Findings</th>
</tr>
</thead>
</table>
| Klinkenberg, et al.| 2006 | Urban malaria and anaemia in children: a cross-sectional survey in two cities of Ghana | **Objective:** To describe the epidemiology of urban malaria, an emerging problem in sub-Saharan Africa.  
**Method:** Cross-sectional surveys of communities in Accra and Kumasi, Ghana, determining risk factors for malaria infection and anaemia in children aged 6-60 months. | Malaria prevalence rates ranged from 2% to 33% between urban communities. 47.1% of children were anaemic (Hb<11.0 g/dl). Factors associated with malaria prevalence were low socio-economic status, age and anaemia. The attributable risks of anaemia and severe anaemia (Hb<8.0 g/dl) caused by malaria were 5% and 23% respectively. Malaria in urban areas displayed a heterogeneity and complexity that differed from the rural environment. |                                                         |
| Ehrhardt, et al.   | 2006 | Malaria, anaemia, and malnutrition in African children--defining intervention priorities | **Objectives:** To investigate the interactions among malaria, anaemia, and malnutrition in childhood morbidity in sub-Saharan Africa.  
**Method:** Evaluation of plasmodial infection, anaemia, and nutritional indices in 2 representative surveys comprising >4000 children in northern Ghana in 2002. | Infection with Plasmodium species was observed in 82% and 75% of children in the rainy and dry season, respectively. The fraction of fever attributable to malaria was 77% in the rainy season and 48% in the dry season and peaked in children of rural residence. Anaemia (hemoglobin level, <11 g/dL) was seen in 64% of children and was, in multivariate analysis, associated with young age, season, residence, parasitemia, P. malariae coinfection, and malnutrition (odds ratio [OR], 1.68 [95% confidence interval [CI], 1.38-2.04]). In addition, malnutrition was independently associated with fever (axillary temperature, > or = 37.5 degrees C; OR, 1.59 [95% CI, 1.13-2.23]) and clinical malaria (OR, 1.67 [95% CI, 1.10-2.50]). Malnutrition was found to be a fundamental factor contributing to malaria-associated morbidity and anaemia, even if the latter exhibits multifactorial patterns. |                                                         |
| Tetteh, et al.     | 2004 | An analysis of the environmental health impact of the Barekese Dam in Kumasi, Ghana | **Objective:** To examine the impact of the Barekese Dam in Ghana on the health status of three riparian communities downstream against a control.  
**Method:** Baseline analysis of environmental health status of communities with reference to traditional endemic communicable water-related diseases in the catchment area (e.g. malaria, urinary schistosomiasis, infectious hepatitis, diarrhoeal diseases and scabies). Case-control study was then conducted in the three phases of the dam (pre-construction, at the end of the construction and in the late operational phases) to analyse the health status of the communities as a function of the phases of the dam. | The control community consistently had a much better health status than two of the riparian communities, which were closer to the dam in all the three phases. However, it had a better health status than the third riparian community, which was farthest downstream, only in the first two phases. The study concluded that there was a strong association between the presence of the dam and poorer health status of the downstream communities in close proximity to it. |                                                         |
10. Appawu, et al. 2004 Malaria transmission dynamics at a site in northern Ghana proposed for testing malaria vaccines

**Objective:** To study malaria transmission dynamics in Kassena Nankana district (KND), a site in northern Ghana proposed for testing malaria vaccines.

**Method:** Mosquito sampling for 1 year using human landing catches in three micro-ecological sites (irrigated, lowland and rocky highland) yielded 18,228 mosquitoes. Transmission was highly seasonal, and the heaviest transmission occurred from June to October. The intensity of transmission was higher for people in the irrigated communities than the non-irrigated ones. An overall annual entomological inoculation rate (EIR) of 418 infective bites was estimated in KND. There were micro-ecological variations in the EIRs, with values of 228 infective bites in the rocky highlands, 360 in the lowlands and 630 in the irrigated area. Approximately 60% of malaria transmission in KND occurred indoors during the second half of the night, peaking at daybreak between 04.00 and 06.00 hours.

11. Afrane, et al. 2004 Does irrigated urban agriculture influence the transmission of malaria in the city of Kumasi, Ghana?

**Objective:** To verify the possible impact of irrigated urban agriculture on malaria transmission in cities, we studied entomological parameters, self-reported malaria episodes, and household-level data in the city of Kumasi, Ghana.

**Method:** A comparison was made of larvae abundance/distribution of Anopheles spp between city locations without irrigated agriculture, city locations with irrigated urban vegetable production, and peri-urban (PU) locations with rain-fed agriculture. Polymerase chain reaction (PCR) analysis of Anopheles gambiae sensu lato revealed that all specimens processed were A. gambiae sensu stricto. The pattern observed in the night catches was consistent with household interviews because significantly more episodes of malaria and subsequent days lost due to illness were reported in peri-urban and urban agricultural locations than in non-agricultural urban locations.


**Objective:** To profile the severity and seasonality of malaria and anaemia in Kassena-Nankana District of northern Ghana.

**Method:** Random cross-sectional surveys, timed to coincide with the end of low (May 2001) and high (November 2001) malaria transmission seasons and to yield information as to the potential value of haemoglobin (Hb) levels and parasitaemia as markers of malaria morbidity and/or malaria vaccine effect.

Parasitaemia was found in 22% (515 of 2286) screened in May (dry-low transmission), and in 61% of the general population (1026 of 1676) screened in November (wet-high transmission). Malaria prevalence in May ranged from 4% (infants <6 months and adults 50-60 years) to 54% (children 5-10 years). A significantly lower risk of parasitaemia among infants [odds ratio (OR) 6-8] and young children (OR 3-4) living in the central, more urbanized sector of the study area.


**Objective:** To study the incidence density of infection and disease caused by Plasmodium falciparum in children aged six to 24 months living in the holoendemic Sahel of northern Ghana was measured during the wet and dry seasons of 1996 and 1997.

**Method:** A cohort study of 259 and 277 randomly selected children received supervised curative therapy with quinine and Fansidar and primaquine for those The incidence density of parasitemia after radical cure was 4.7 infections/person-year during the dry season and 7.1 during the wet season (relative risk = 1.51, 95% confidence interval [CI] = 1.25-1.81; P = 0.00001). Although the mean parasitemia count at time of reinfection in the dry season (3,310/microl) roughly equaled that in the wet season (3,056/microl; P = 0.737), the risk ratio for parasitemia > 20,000/microl during the wet season was 1.71 (95% CI = 1.2-2.4; P = 0.0025). No seasonal differences in
Chapter Two: Literature Review

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Study Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>14. Ofosu-Okyere, et al.</td>
<td>2001</td>
<td>Novel Plasmodium falciparum clones and rising clone multiplicities are associated with the increase in malaria morbidity in Ghanaian children during the transition into the high transmission season. <strong>Objective:</strong> To study the effect of the onset of the malaria transmission season on disease incidence. <strong>Method:</strong> Blood samples were collected from 40 children living in the rural town of Dodowa, between February and August 1998. P. falciparum parasite densities were calculated and PCR genotyping was carried out using the polymorphic MSP-1 and MSP-2 genes as target loci for the estimation of the number of parasite clones in each sample. The study showed that the probability of a Ghanaian child having a symptomatic malaria episode is positively associated with both increasing numbers and novel types of <em>P. falciparum</em> clones.</td>
</tr>
<tr>
<td>15. McGuinness, et al.</td>
<td>1998</td>
<td>Clinical case definitions for malaria: clinical malaria associated with very low parasite densities in African infants. <strong>Objective:</strong> To determine case definitions for the diagnosis of clinical malaria, age- and season-specific estimates of the fraction of fevers attributable to malaria. <strong>Method:</strong> A cohort study of 154 children recruited at birth and monitored for fever and malaria infection until 2 years of age. Estimates of AF varied with age and season. For infants, AF was 51% during the wet season and 22% during the dry season; for children over one year of age, AF was 89% during the wet season and 36% during the dry season.</td>
</tr>
<tr>
<td>16. Binka, et al.</td>
<td>1994</td>
<td>Patterns of malaria morbidity and mortality in children in northern Ghana. <strong>Objective:</strong> To study patterns of malaria morbidity and mortality in children in northern Ghana. <strong>Method:</strong> A malaria prevalence survey was carried out in young children in northern Ghana between October 1990 and September 1991, in an area with continuous mortality and morbidity surveillance. There was marked seasonal variation in malaria deaths, reported fevers, parasite rates and mean parasite densities, with parasite rates reaching 85-94% in the wet season. The monthly numbers of malaria deaths were highly correlated with rainfall in the previous 2 months (r = 0.90, P &lt; 0.001). Parasite rates were highest in the oldest children (5-7 years), but parasite densities and rates of febrile illness were highest in those 6-11 months old.</td>
</tr>
<tr>
<td>17. Afari, et al.</td>
<td>1993</td>
<td>Seasonal characteristics of malaria infection in under-five children of a rural community. <strong>Objective:</strong> To assess the seasonal characteristics of malaria infection in under-five children of a rural community. <strong>Method:</strong> Blood samples analysis in the under-five children conducted at Gomoa Onyade/Otsew Jukwa, from December, 1986 to September, 1987. Crude parasite rates ranged from 19.6 to 33.5 per cent in the dry season (December and March) and 33.0 to 44.0 per cent in the wet season (June and September). <em>P. falciparum</em> was the predominant parasite species by parasite formula analysis with higher rates in the rainy season (94.2 to 95.8 per cent) compared to that of the dry season (51.4 to 78.8 per cent). <em>P. malariae</em> with normal glucose-6-phosphate dehydrogenase activity and 20 weeks of post-therapy follow-up consisted of three home visits weekly and examination of Giemsa-stained blood films once every two weeks. Anaemia was observed to exist in this region, probably because the longitudinal cohort design using first parasitemia as an end point prevented the subjects from developing the repeated or chronic infections required for anaemia induction.</td>
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<td><strong>18. Chinery, W. A. 1984</strong></td>
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<td>Effects of ecological changes</td>
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<td>on the malaria vectors Anopheles funestus and the Anopheles gambiae complex of mosquitoes in Accra, Ghana</td>
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<td><strong>Objective:</strong> To study the effects of ecological changes on the malaria vectors Anopheles funestus.</td>
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<td><strong>Method:</strong> A review of studies has been conducted on mosquito breeding, indoor resting density and some parasitological and vectorial indices in Accra since 1911.</td>
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<td>An. gambiae s.l. has adapted to breeding appreciably in water-filled domestic containers in recent times (viz. 21.14 +/- 4.4% of all breeding), compared with a low frequency of breeding in such domestic containers in the earlier years 1911-1930 (viz. 1.97 +/- 1.67% of all breeding). Its breeding has also increased (viz. 5.3% to 25.4% of all breeding) in the numerous polluted water habitats created as a result of urbanization. Diminished malaria parasite rates between 1912 and 1964 were attributed to the almost complete elimination of An. funestus and decrease in breeding intensity of An. gambiae s.l.</td>
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<td><strong>Diarrhoea studies</strong></td>
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<td><strong>19. Osei and Duker 2008</strong></td>
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<td>Spatial dependency of V. cholera prevalence on open space refuse dumps in Kumasi, Ghana: a spatial statistical modeling.</td>
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<td>In this study, exploitation of the importance of two main spatial measures of sanitation in cholera transmission to determine the relationship between refuse dumps and cholera transmission in an urban city, Kumasi. These are proximity and density of refuse dumps within a community.</td>
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<td>A spatial statistical modelling carried out to determine the spatial dependency of cholera prevalence on refuse dumps show that, there is a direct spatial relationship between cholera prevalence and density of refuse dumps, and an inverse spatial relationship between cholera prevalence and distance to refuse dumps. A spatial scan statistics also identified four significant spatial clusters of cholera; a primary cluster with greater than expected cholera prevalence, and three secondary clusters with lower than expected cholera prevalence. A GIS based buffer analysis also showed that the minimum distance within which refuse dumps should not be sited within community centres is 500 m. Authors concluded that proximity and density of open space refuse dumps play a contributory role in cholera infection in Kumasi.</td>
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<td><strong>20. Reither el al. 2007</strong></td>
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<td>Acute childhood diarrhoea in northern Ghana: epidemiological, clinical and microbiological characteristics</td>
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<td><strong>Objective:</strong> To examine the microbiological causes and clinico-epidemiological aspects were examined during the dry season 2005/6 in Tamale, urban northern Ghana [280].</td>
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<td><strong>Methods:</strong> Stool specimens of 243 children with acute diarrhoea and of 124 control children were collected. Patients were clinically examined, and malaria and anaemia were assessed. Rota-, astro-, noro- and adenoviruses were identified by (RT-) PCR assays. Intestinal parasites were diagnosed by microscopy, Watery stools, fever, weakness, and sunken eyes were the most common symptoms in patients (mean age, 10 months). Malaria occurred in 15% and anaemia in 91%; underweight (22%) and wasting (19%) were frequent. Intestinal micro-organisms were isolated from 77% of patients and 53% of controls (P &lt; 0.0001). The most common pathogens in patients were rotavirus (55%), adenovirus (28%) and norovirus (10%); intestinal parasites (5%) and bacteria (5%) were rare. Rotavirus was the only pathogen found significantly more frequently in patients than in controls (odds ratio 7.7; 95%CI, 4.2-14.2), and was associated with</td>
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stool antigen assays and PCR, and bacteria by culturing methods. young age, fever and watery stools. Patients without an identified cause of diarrhoea more frequently had symptomatic malaria (25%) than those with diagnosed intestinal pathogens (12%, $P = 0.02$). Rotavirus-infection was the predominant cause of acute childhood diarrhoea in urban northern Ghana. The abundance of putative enteropathogens among controls indicated prolonged excretion or limited pathogenicity. In this population with a high burden of diarrhoeal and other diseases, sanitation, health education, and rotavirus-vaccination was shown to have expected substantial impact on childhood morbidity.

<p>| 20. Obiri-Danso, et al. 2005 | Aspects of health-related microbiology of the Subin, an urban river in Kumasi, Ghana | <strong>Objective:</strong> To assess the influence of urban waste, sewage and other human centred activities on the microbiological quality of the river Subin, which flows through the metropolis of Kumasi, Ghana, and serves as drinking water for communities downstream. <strong>Method:</strong> Three sites, Racecourse, Asafo and Asago, on the Subin were monitored over a year for total coliforms, faecal coliforms, enterococci and biochemical oxygen demand. Bacterial indicator numbers (geometric mean $100\text{ ml}^{-1}$) varied from $1.61 \times 10^9$ to $4.06 \times 10^{13}$ for total coliforms, $9.75 \times 10^8$ to $8.98 \times 10^{12}$ for faecal coliforms and $1.01 \times 10^2$ to $6.57 \times 10^6$ for enterococci. There was a consistent increase in bacterial loading as the river flows from the source (Racecourse) through Kumasi. Bacterial numbers were significantly ($p &lt; 0.05$) higher during the rainy season compared with the dry (harmattan) season. The biochemical oxygen demand ranged from $8 \text{ mg l}^{-1}$ at the source of the river to $419 \text{ mg l}^{-1}$ at Asago; none of the sites achieved internationally accepted standards for water quality. |
| 21. Boadi &amp; Kuitunen 2005 | Environmental and health impacts of household solid waste handling and disposal practices in third world cities: the case of the Accra Metropolitan Area, Ghana | <strong>Objective:</strong> To examine the impacts on environment and health of household-level waste management and disposal practices in the Accra Metropolitan Area, Ghana. <strong>Method:</strong> A simple questionnaire survey. Only 13.5 percent of respondents are served with door-to-door collection of solid waste, while the rest dispose of their waste at communal collection points, in open spaces, and in waterways. The majority of households store their waste in open containers and plastic bags in the home. Waste storage in the home is associated with the presence of houseflies in the kitchen ($r = .17$, $p &lt; .0001$). The presence of houseflies in the kitchen during cooking is correlated with the incidence of childhood diarrhoea ($r = .36$, $p &lt; .0001$). Inadequate solid waste facilities result in indiscriminate burning and burying of solid waste. There is an association between waste burning and the incidence of respiratory health symptoms among adults ($r = .25$, $p &lt; .0001$) and children ($r = .22$, $p &lt; .05$). |
| 22. Curtis, V. 2003 | Talking dirty: how to save a million | <strong>Objective:</strong> To promote public-private hand-washing programmes in Ghana and India. The most effective way of stopping infection is to stop faecal material getting into the child's environment by safe disposal of |</p>
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<th>Study</th>
<th>Year</th>
<th>Title</th>
<th>Objective</th>
<th>Method</th>
<th>Findings</th>
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<td>23. Mensah, et al. 2002</td>
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<td>Street foods in Accra, Ghana: how safe are they?</td>
<td>To investigate the microbial quality of foods sold on streets of Accra and factors predisposing to their contamination.</td>
<td>Use of structured questionnaires to collect data from 117 street vendors on their vital statistics, personal hygiene, food hygiene and knowledge of food borne illness. Standard methods were used for the enumeration, isolation, and identification of bacteria.</td>
<td>Most vendors were educated and exhibited good hygiene behaviour. Diarrhoea was defined as the passage of 3 or =3 stools per day by 110 vendors (94.0%), but none associated diarrhoea with bloody stools; only 21 (17.9%) associated diarrhoea with germs. The surroundings of the vending sites were clean, but four sites (3.4%) were classified as very dirty. The cooking of food well in advance of consumption, exposure of food to flies, and working with food at ground level and by hand were likely risk factors for contamination. Examinations were made of 511 menu items, classified as breakfast/snack foods, main dishes, soups and sauces, and cold dishes. Mesophilic bacteria were detected in 356 foods (69.7%): 28 contained Bacillus cereus (5.5%), 163 contained Staphylococcus aureus (31.9%) and 172 contained Enterobacteriaceae (33.7%). Concluded that street foods can be sources of enteropathogens.</td>
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<td>24. Shier, et al. 1996</td>
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<td>Drinking water sources, mortality and diarrhoea morbidity among young children in northern Ghana</td>
<td>To describe seasonal and geographical variations in drinking water sources; to look for other predictors of water source use; and to establish whether the drinking water source was associated with the risk of child death or the period prevalence of diarrhoea among young children.</td>
<td>Part of the Ghana Vitamin A Supplementation Trials’ Survival Study which was carried out in one of the districts of the Upper East Region between January 1989 and December 1991. Boreholes were used as the main source of drinking water by about 60-70% of respondents. They were used slightly more frequently in the dry season. In the rainy season, the use increased of more traditional sources such as rainwater or holes dug in stream beds. The use of boreholes was greatest in the northern zone of the study area and was more common in those who had had some formal education and were of higher socioeconomic status. Some association was found between reported drinking water source and diarrhoeal morbidity, although this association appeared to be seasonal. No significant association was found between drinking water source and child mortality.</td>
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<td>25. Armah, et al. 1994</td>
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<td>Seasonality of rotavirus infection in Ghana</td>
<td>To study human rotavirus (HRV) infection and its seasonal distribution was studied over a 12-month period in Ghana.</td>
<td>Collection and analysis of a total of 561 stool samples, 447 diarrhoea stools and 114 non-diarrhoea stools. Rotavirus was detected during 10 of the 12 months and showed a seasonal trend. It was high during the relatively cool dry months and low during the wet season. Peaks of infection were in February (26.2%) and September (24.5%). HRV was detected in 67 of 447 of the diarrhoea stools (15.0%) and in eight of 114 stools.</td>
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Chapter Two: Literature Review

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<th>stools (controls), obtained from children attending three polyclinics in Accra.</th>
<th>controls (7.0%). The HRV isolation rate was highest (20.2%) in the under-18-months age group. The RNA electropherotype of the HRV isolates was predominantly (83.6%) of the long type. Non-group A HRV was detected in 14.9% of the HRV-positive samples.</th>
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<td>26.</td>
<td>De Sherbinin, A.</td>
<td>1993</td>
<td>Spotlight: Ghana Objective: To initiate discussion on water supply economies in Africa Method: Review</td>
<td>The underdeveloped water supply systems make water-borne diseases, such as diarrhoea and bilharzia, serious health threats. Insect-borne onchocerciasis is also a problem.</td>
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<td>27.</td>
<td>Agbodaze &amp; Owusu</td>
<td>1989</td>
<td>Cockroaches (Periplaneta americana) as carriers of agents of bacterial diarrhoea in Accra, Ghana Objective: To demonstrate that cockroaches could play an important role in the transmission of pathogenic organisms (diarrhoeal pathogens), especially in our environment. Method: Characterisation of cultures of enteric bacterial pathogens from bodies and intestinal contents of 208 cockroaches (Periplaneta americana), collected from kitchens in Accra and some surrounding villages.</td>
<td>Six of them harboured three different serogroups of Salmonella, one had Shigella dysenteriae, 64 had Coliforms, 13 had Proteus species, two had Pseudomonas species and the rest (122) carried none of the bacterial species mentioned above. The presence of Salmonella species Shigella dysenteriae and Coliforms in these insects, which were collected from kitchens where foods are kept, points to the facts that these insects could play an important role in the transmission of these pathogenic organisms, especially in our environment. Permanent solution to these bacterial diarrhoea disease problems could only be solved when food, animals and the environments are free of these microbes.</td>
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2.2 Burden of Morbidity and Mortality

Assessment of data from health facilities and annual reports showed evidence that the Ghanaian population was at the cross-roads of an epidemiological transition [281]. Many studies have reported that while the burden of infectious diseases was still huge, that of non-communicable diseases had increased considerably over last two decades [282, 283]. While the major causes of child mortality reportedly include malaria, diarrhoea, respiratory infection, and neonatal conditions, those for adult deaths were predominantly; HIV infection, hypertension, diabetes mellitus and road traffic [281]. Although a broad range of risk factors had been reported to be responsible for the observed burden; low level of literacy, poor sanitation, under-nutrition, alcohol abuse, sedentary life styles and unhealthy diets were the major determinants of ill-health contributing to the observed high morbidity and mortality rates in the Ghanaian population [39, 60, 68, 154, 160, 281, 284].

2.3 The Burden of Diarrhoea Specific Morbidity and Mortality

The cause and persistence of diarrhoeal morbidity and mortality in the populations in Ghana has remained a major concern to health authorities. For instance, the biology of the agents of diarrhoeal causation has been abundantly documented in literature [2, 10, 28, 29, 91, 97, 111, 150, 235, 285-287]. Yet, diarrhoea has remained a huge challenge to public health, especially in poor urban neighbourhoods where infectious diseases still constituted a huge threat to the quality of life. Diarrhoea is one of the leading cause of morbidity and mortality among children today and it is estimated that well over 12 million people die from diarrhoeal related causes [288]. Despite report of moderate
reduction in annual diarrhoeal deaths at the beginning of this millennium; probably due to the implementation of more or less potentially powerful interventions in the preceding decades, the disease has remained a leading cause of child mortality globally [280, 288-292]. Evidence from literature suggests that such global trends appear to be declining due to remarkable progress in diagnosis and the development of effective control strategies [63, 272, 293-296]. Additionally, despite the global reduction in diarrhoeal morbidity, country-specific situations continue to vary widely [296-298]. In Ghana, diarrhoea still exerts a significant pressure on health facilities and is reported as the fourth most common illness in Outpatient Department (OPD) [45, 250, 252, 272, 299, 300]. Although the analyses of clinical and OPD records have shown that diarrhoea had been declining during the last decade, its contribution to outpatient visitation is still substantial, making a little over 40 percent of the reason for outpatient visitations in Ghana in 2007 (Fig 1.1). While the proportion of hospitalization due to diarrhoea has been declining since 2000; its contribution to the overall outpatient attendance gives cause for worry and paints a gloomy picture about Ghana’s capacity to attain the Millennium Development Goals (MDGs). It is not exactly clear from routine clinical records what combinations of factors account for its sustained transmission which places diarrhea among the top-five causes of infant deaths in Ghana [45, 250-252, 289, 297, 299, 301].
2.4 The burden of malaria morbidity and mortality

Malaria is a debilitating outcome of the parasitic association between a host (usually the human host) and an infective agent called *Plasmodium sp* [37, 92, 302-309]. Malaria is extremely devastating, remaining widespread throughout the tropics, but also occurring in some temperate regions [97, 302, 304, 308, 310]. It exerts a heavy burden of ill health and death; especially amongst children and pregnant women, in high transmission areas [97, 308, 311, 312]. It poses a dangerous risk to travellers and immigrants with imported cases increasing in non-endemic areas [97, 309].

Around 90% of the malarial deaths occur in Africa and mostly in young children [302, 308, 313, 314]. Malaria is Africa’s leading cause of under-five mortality (20%) and constitutes 10% of the continent’s overall disease burden [89, 97]. It accounts for 40% of public health expenditure, 30-50% of inpatient admissions, and up to 50% of outpatient visits in areas with high malaria transmission rate [97, 314].
Each year, an estimated 0.7-2.7 million people who die from malaria, an overwhelming 75% of them reportedly occurs in African children [97] due to poor case management and other factors working together. In particular, Ghana national malaria control programs continue to rely on effective case management while attempting the large-scale deployment of insecticide-treated bed nets and awaiting the discovery of an effective and affordable vaccine [82, 315-318]. While awaiting vaccine discovery, an important control strategy has focused on manipulation of both environmental and social factors to be able to strengthen control programs [90, 317, 319-321]. Previous studies demonstrate a lack of complete understanding of how urban environmental conditions and neighbourhood characteristics are currently influencing the pattern of urban malaria mortality in Ghana [90]. For malaria control strategies to be effective, they should concentrate on targeting measures at ecological factors which influence malaria mortality, the groups at highest risk of disease and those who are prone to death from malarial attacks [37, 83, 92, 98, 288, 303, 322]. However, this cannot be done effectively unless there is a clear or unambiguous understanding of how these urban environmental factors are associated with urban malaria mortality.

It is evident from many studies that the persistence of the disease is a consequence of a combination of social, economic and behavioural factors operating across a broad spectrum of ecological/environmental factors [318, 320, 323]. Efforts to eradicate malaria have increased and some success has been achieved in reducing its case fatality rate in many areas [90, 183, 283]. However, analysis of routine outpatient data in Ghana shows that the disease has consistently remained the topmost cause of ill health and death in both rural and urban areas (Fig 2.2).
Despite all the efforts by the national malaria control program to bring the disease under control and the intensive mass campaigns for its prevention, the burden of the disease continues to grow contributing nearly 50 percent of the reason for outpatient visitation among related infectious diseases in Ghana in 2007 (see Fig 2.2). Although there seems to be a consensus that urban environmental/ecological conditions and neighbourhood characteristics appear to have a very strong influence on malaria morbidity in Ghana, it is unclear how these factors contribute to the current patterns of the observed urban malaria mortality [89, 306]

2.5 Conclusion
This review found many studies on many aspects of urban health and in a wide variety of settings [90, 181, 183, 324-326]. The range of variables observed by the review was wide
and included the influence on human health outcomes of different aspects of urban ecosystem (e.g. physico-chemical, biophysical and socio-economic characteristics) [90, 168, 181, 183, 265, 324, 327]. The studies reported in the review have varied in the degree of sophistication and while including different measures of health such as morbidity, disability, immunity, and parasite resistance to drugs, none included mortality as the health outcome [134, 185, 248, 256, 328]. The causal chains linking disease and biophysical states of urban ecology as well as many intermediate pathways have been studied, and while some of the variables have been more thoroughly examined than others, none has investigated the association between mortality and environmental conditions in low income settings [90, 101, 241, 255, 289, 303, 306, 329-331]. For example, the influence of urban environmental factors on morbidity has been more widely and thoroughly investigated than the effects of these factors on mortality. Additionally, the differences between the status of rural and urban population health (applies only morbidity and not mortality) has been thoroughly investigated than disaggregated analyses of urban sub-population groups.

In Ghana, researches describing the effects of urban ecological change on human health were scanty and covered only few environmental variables [92, 97, 113, 255, 304, 330, 332]. Moreover, while only a few studies have investigated the link between malaria/diarrhoea morbidity and environmental/climatic factors (with greater focus on climatic than biophysical environmental factors), none has explored such relationships between urban/environmental conditions/neighbourhood characteristics and malaria/diarrhoea deaths [89, 90, 92, 252, 303-305, 308, 309, 314, 322, 329, 333, 334]. The few studies that investigated the relationships between ecological factors and health
(e.g. morbidity) were more widely conducted in rural than in urban settings in Ghana [84, 92, 252, 303, 304, 307, 312, 319, 322, 329, 333, 335]. A few other studies have observed a strong association between environmental/climatic factors and malaria/diarrhoea diseases [45, 250-252, 289, 297, 299, 301, 304, 310, 313, 314, 329, 332, 336, 337]. However, none of the studies has assessed and described the nature and level of the association between the environmental factors (e.g. water supply & sanitation, hygiene, housing/living arrangements, open drains, influence of large water bodies such as lagoons within settlements, etc.) and malaria/diarrhoea mortality [45, 81, 83, 92, 98, 250-252, 289, 297, 299, 301, 302, 304, 305, 307, 310, 312-314, 319, 329, 332, 333, 336, 337]. Of all the studies reviewed, only Shier and others, 1996 attempted to assess the level of association between rural water supply and diarrhoeal deaths. This study reported that while there was a strong positive association between rural water supply sources and diarrhoeal illnesses, no significant association was found between drinking water sources and diarrhoeal deaths in rural Ghana [301]. This finding was theoretically consistent because, while the ingestion of polluted/contaminated water could potentially increase the incidence of diarrhoeal disease, the interaction with intervening factors such as early detection/diagnosis and treatment, efficient case management, etc., could affect the course of transgression from illness to death [45, 98, 251, 297, 301, 302, 305, 311, 313, 314, 322, 329, 330, 336, 338]. The effects of the intervening factors on the course of transgression from disease to death could be different in urban areas where deteriorating urban environmental conditions and urban poverty could fuel widespread transmission of infectious diseases [28, 198, 329, 331, 339-341]. There was massive evidence that the causal chain linking environmental factors and mortality were influenced by
socioeconomic factors such as income, employment and education [89, 90, 301, 302, 304, 305, 310, 330, 337]. However, it is not completely understood how the observed urban mortality is contributed by each of the environmental factors if socio-economic factors were held constant. For instance, in rural areas the relationship between environmental conditions and health could be less pronounced because the effects of the environmental factors on health outcome and poverty were less strongly associated compared to the case in urban centers [28, 39, 45, 59, 73, 83, 92, 101, 103, 108, 111, 141, 151, 200, 241, 255, 279, 285, 329, 334, 341-344]. Urbanization has been widely implicated as the major cause of deterioration in urban environmental quality (e.g. urban water supply, sanitation, hygiene, etc) and indirectly influences the transmission of infectious diseases and urban mortalities due to malaria and diarrhoea in particular [41, 57, 90, 97, 104, 113, 130, 136, 138, 184, 206, 211, 232, 234, 299, 311, 330, 331, 341, 345-348]. It could be possible that the interaction among deteriorated environmental quality, intensified urban poverty and low education could influence the intermediate processes and thus alter the sequence of events in the pathway from disease to death in a much more complex way in urban areas than in rural areas. While some entomological studies have reported that Anopheles mosquito breeds well only in clear standing water such as in puddles, small containers and other discarded receptacles, there are conflicting opinions suggesting that waste water in open drains could support mosquito breeding and therefore promote malaria transmission [81, 89, 97, 104, 111, 285, 303, 307, 312-314, 322, 329, 344, 349]. Sattler and others have demonstrated that the larvae of *A. gambiae* were found in habitats organically polluted by rotten vegetation, human faeces or oil in Dar es Saalam and thus contributed to excess malaria morbidity. However, it is not clear if such habitats could
contribute to excess malaria mortality as well. Finally, no research work has reported to have assessed whether or not large water bodies such as lagoons in urban spaces supported Anopheles mosquito breeding, and whether such large water bodies may thus contribute to more localised excess malaria mortality in nearby residential areas [28, 45, 73, 83, 89, 92, 98, 101, 103, 108, 111, 250, 255, 285, 289, 334, 336, 344]. Most researchers agree that research findings from studies conducted on vector ecology in rural settings might not be applicable to urban settings and for this reason, it is reasonable to explore urban specific understanding of malaria and diarrhoea mortalities.
Chapter Three: Research Conceptualization and Study Design

3.1 Methodology

This analysis explored a large number of environmental and socioeconomic variables (explanatory variables) with the response variables (malaria/diarrhoea mortality) using a mix of models to test the stated hypotheses. An ecological study design which used the city-level urban environmental and socioeconomic data from the 2000 Census and mortality data, aimed to deepen our understanding of the inter-relationship among urban environmental conditions, urban socioeconomic conditions and health outcomes (malaria/diarrhoea mortality) in a rapidly urbanizing setting in a low income economy.

The analysis was complex and therefore called for a complex research design which employed a combination of several models. Some of the models used included Principal Component Analysis (PCA), bivariate and multi-variate linear regression analysis and geostatistical approaches. The study had a strong Geographic Information System (GIS) component utilizing a digital map which allowed for spatial regression of mortality data on environmental and socioeconomic variables and the assessment of excess mortalities.

The study adopted the boundary classification into census clusters of the study setting (the city of Accra) which had already been used during previous national population censuses and the Ghana Demographic and Health Surveys. The unit of analysis was the census clusters, also referred to as “localities or supervision areas” by the Ghana Statistical Service (GSS). The assessment of the influence of rivers, lagoons and other water bodies was one of the interests in this study and therefore the polygons of these features were combined with those of the census clusters. The geostatistical approaches
used were local and global autocorrelation analyses as well as Geographic Weighted Regression (GWR).

3.2 Study Area and Sample Population

This research was conducted in Accra, the capital city of Ghana which occupies a total land area of 238,537 square kilometres and had a total population of 18.9 million as of 2000 census [7, 225, 228, 350-353]. For administrative convenience, the city has been divided into six (6) submetro districts which are made up of 70 census clusters and 1700 Enumeration Areas (EAs) for census enumeration purposes.

Greater Accra Region, where Accra is located is the smallest of the ten political regions in Ghana. However, it is the largest of the ten leading urban centers. The population of Accra was 1.7 million in 1990 and 2.7 million in 2000. The city harbours over 30% and nearly 15% of the urban and of the total population of Ghana respectively. Regarding sanitation and urban environmental conditions, the generation and annual rate of increase of solid waste is high in Ghana, especially in the urban centers. For instance, the per capita production of refuse in Accra was 0.40 kg/day [354, 355] in 2000 and nearly 70% by weight was degradable organic materials; representing 0.3 million metric tons of total wastes generated annually [38, 355, 356]. In terms of city-wide sanitation, over 50 percent of the solid waste generated remained uncollected in 2000 [355]. The general topography of the study area is a flat low-lying terrain, underlain with clayish and impervious soils and characterised by inadequate and undersized drains (Fig. 3.1).
The city is drained by two rivers namely; the Odaw River and the Korle River and their tributaries. Dotted at several points by lagoons, swamps, large drains and other water bodies which are largely polluted by both solid and liquid wastes (Fig. 3.1), the city forms several neighbourhoods and community areas with differing levels of urban environmental quality conditions.

This provides for high habitat diversity and high heterogeneity in vector breeding and pathogen transmission across the city. The high habitat diversity together with hot and humid climate; provide excellent breeding opportunities for Anopheles mosquitoes. The different levels of pollution of the water bodies present high uncertainty for their suitability to support Anopheles sp larval growth and development, although there is no doubt about their suitability to support diarrhoea pathogen growth and transmission [289]. For example, the amount of total Kjeldahl nitrogen (TKN), the chemical oxygen demand (COD) and the total organic carbon (TOC) will dramatically affect the stability
of wastes strewn lagoon as a medium of support for mosquito larval growth [63, 69, 270, 294, 296]. The TKN values for lagoon water less than 50 mg/litre or greater than 500 mg/litre (4.2 lbs/1000gal) do not support early stages of mosquito larvae [265, 301]. TOC values that fall outside the range of 100 to 1000 mg/litre are also effective, as are COD values of less than 400 mg/litre and greater than 2000 mg/litre. Total nitrogen appears to be the most important characteristic of lagoon water regarding the control of mosquitoes. High TKN values alone will dramatically reduce larval survival even if TOC and COD values were ideal for mosquito breeding [252, 265, 266, 301]. All these factors combine to offer a unique opportunity for assessing the influence of a fast degrading urban complex on malaria and diarrhoea mortality.

3.3 Study Population
The study population was the urban sub-population of Ghana. The urban population of Accra was used as a generalizable sample of the urban population of Ghana.

3.4 Study Design and Data Sources (Study variables)
The study used multiple datasets, which called for a mix of data collection strategies. In the study, geographically contiguous EAs of fairly similar population and area characteristics were grouped into census clusters or localities within urban Accra. This was based upon the 2000 Population and Housing Census sampling frame for enumeration. The census 2000 database held several cluster level measures of socioeconomic status including educational attainment, literacy rate, school enrolment, religion, ethnicity, marital status, employment status, type of employment, economic
activity status (e.g. whether employable or not) and environmental variables such as: per capita waste generation, total waste generation, amount of solid wastes uncollected (waste deposition), volume of liquid wastes disposed through the sewer system, volume of liquid wastes by non-sewer disposal, number of households with pit-latrines, number of households with toilet/bath facility in different house, number of households with pan-latrines, etc which were obtained by a written permission of the Government Statistician. We extracted the relevant socioeconomic and environmental variables from the database for each of the 70 clusters in Accra.

In all, we extracted a total of 118 socioeconomic and environmental variables. For all the 70 clusters in Accra, we computed appropriate summary measures of the environmental and socioeconomic variables at aggregate cluster level. The 118 variables were sub-grouped into eleven sub-categories; namely, waste generation, sanitation and water supply, housing type and construction materials, hygiene conditions, education level, occupation type, place of work, household energy use, economic activity, marital status and ethnicity. In order to deal with multicollinearity and to include only variables with significant associations in the dataset, we carried out a stepwise removal multiple regression modelling and correlation analyzes (using STATA 9, College Station, TX) thus excluding several redundant variables on the basis of the magnitude of their variation inflation factors (VIFs). We excluded variables on household energy use because no evidence exists in literature which demonstrates a link between them and the outcome measure (i.e. malaria and diarrhoea mortality). Finally, variables on marital status and ethnicity were excluded in the analysis by dint of their cultural sensitivity.
3.5 Mortality Data Collection
Death registers were obtained from the Ghana Births and Deaths Registry (i.e. the Ghana Vital Registration System - VRS) for the years 1998 to 2002 inclusive. The information on death events e.g. place address or normal place of residence of the deceased, age, nationality, cause of death, month of death, etc contained in these registers was entered into a database. To do this, a template of the death registration form was created using Microsoft Access forms wizard (Fig. 3.2). Using this electronic form, relevant information on all deaths as recorded on death certificates were captured and stored in Microsoft Access database. The total number of deaths recorded and coded according to ICD-9 in the registers over this period in urban Accra was 24,716 out of which 1,292 and 1,001 were deaths attributed to malaria and diarrhoea respectively. The codes for the 9th edition of the International Classification of Disease (ICD-9) were defined as follows:

a) **Diarrhoea:** broadly included all diarrhoea, cholera and gastroenteritis of presumed infectious origin which were defined by codes A00 and A09 according to ICD-9, e.g.

i) cholera

ii) catarrh, enteric or intestinal

(1) colitis NOS (not otherwise specified)

(2) enteritis haemorrhagic

(3) gastroenteritis septic

(4) diarrhoea

(a) NOS

(b) dysentery

(c) infectious diarrhoeal diseases
b) **Malaria**: classified broadly in the range of codes B50 – B54 according to ICD-9.

This included all other parasitologically confirmed malaria.

Once the death records were transferred into an electronic database, the database was cleaned, validated and the death records were allocated to the 70 clusters in Accra according to the house address of normal place of residence prior to death. The death records were then linked to the clusters via cluster codes in a Geographic Information System (GIS) (ArcMap 9.3.1).

![Figure 3.2: Electronic Template of Death Registration Form](image)

### 3.6 Physical Urban Environmental Data

The Ghana Statistical Service (GSS) classified urban environmental conditions in the census 2000 as follows:

- Population and demographics


- Cluster hygiene conditions
  - number of households with Water Closet (WC) per cluster
  - number of households with pit-latrines per cluster
  - number of households with Kumasi Ventilated Improved Pit (KVIP) latrine per cluster
  - number of households with bucket/pan latrine per cluster
  - number of households using public toilets per cluster
  - number of households without toilets (bush/field defecation) per cluster
  - number of households with baths for exclusive use by members of households per cluster
  - number of households using public baths per cluster
  - number of households using open spaces around house for bath per cluster

- Cluster water supply & sanitation
  - total amount solid wastes generated per cluster
  - percentage solid wastes collected per cluster
  - percentage solid wastes uncollected per cluster
  - quantity of solid wastes properly managed at public dump sites per cluster
  - number of households with waste dumps inside homes per cluster
  - total amount of liquid wastes generated per cluster
  - quantity of liquid waste managed through the sewer system per cluster
  - quantity discarded in the open drains/gutters per cluster
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- quantity discarded in open spaces in the compound of households per cluster
- percentage households with pipe-borne water (potable water) per cluster
- percentage of households using public standpipes per cluster
- percentage of households using well/hand dugout pond water per cluster
- percentage of households using borehole water per cluster
- cluster distance from water bodies/lagoons

- Cluster housing & living arrangements
  - number of households with mud/mud brick/earth as construction materials per cluster
  - number of households with wood as construction materials per cluster
  - number of households per cluster, with metal sheet/slate/asbestos as construction materials
  - number of households per cluster, with cement blocks/concrete as construction materials
  - average number of persons per housing unit per cluster
  - number of persons per square meter of cluster (cluster population density).

A considerable amount of evidence exists in literature demonstrating the effect of the physical environment on human health. However, no consensus exists on the standard for definition environmental quality classifications that allow for cross-study comparisons. In literature, different studies have different environmental classifications, thus making comparison of study outcomes between and among different studies rather impossible. In this analysis, the environmental variables were obtained from the census database already
classified into 1) “population and waste generation”, 2) “water supply and sanitation”, 3) “hygiene conditions” and 4) “built structure, form, construction material type and living arrangements”. In all, a total of 65 environmental variables were extracted from the census database under the four distinct sub-categories and summary measures (e.g. the proportions of cluster level conditions) appropriately computed. In order to detect and deal with multicollinearity as well as include only variables with significant associations in the analysis, we conducted correlation analysis and evaluated the correlation matrix for strongly associated covariates. We also conducted a stepwise backward removal multiple regression modelling and assessed the variance inflation factor (VIF) prior to determining the final model for the analysis. After this process, a total of 60 variables were maintained for use in the next stage of analysis. As a first step, we explored the large number of environmental variables under each sub-category to determine the direction of their eigen vectors using PCA. In a data reduction strategy, we again used PCA to decompose the variables under each sub-category into a manageable uni-directional variable which was employed to develop distinctly ordinal urban environmental zones of differing levels of urban environmental quality conditions [165]. The resulting environmental zones nominally defined as “extremely deteriorated zone”, “moderately deteriorated zone” and “least deteriorated zone” represented worst, somewhat worse and good environmental conditions respectively.

3.7 Socioeconomic Data

The influence of socioeconomic factors on health outcomes has long been recognized and past research effort has focused on the relationship between socioeconomic status (SES)
and health inequalities among different subpopulation groups [159]. Socioeconomic status (SES) is frequently implicated as a contributor to the disparate health observed among racial/ethnic minorities, women and elderly populations [155, 174, 357, 358].

There is scientific consensus that several factors (both SES and the physical environment, Fig. 3.3) interact to influence health and health status among populations and, as a consequence, health disparities [159, 174, 357, 359-361]. In the U.S., socioeconomic status (SES) is among the factors most frequently implicated as a contributor to the disparities in health observed among populations [155, 159]. Other factors include lifestyle, the cultural and physical environment, living and working conditions and social and community networks [362, 363]. Adler et al. present three pathways through which SES impacts health, which include its association with healthcare, environmental exposure, and health behaviour and lifestyle [364]. In Figure 3.3, a simplified theoretical model of SES, environment and health interaction is presented.

Although analysis of data on socioeconomic status is nearly always included in epidemiologic research, its specific use is often dependent on data availability [155, 159, 362, 365]. While it is often concluded that differences in SES are the cause of differences in health outcomes among population groups, there is often little, if any, discussion of the specific manner in which SES might have exerted its influence within the context of the study outcomes [366-368]. This then leaves gaps in the causal path with respect to the chain of causal events leading from the multiple pressures from neighbourhood socioeconomic conditions driving changes in neighbourhood environmental conditions which in turn directly influence health outcomes, see Fig. 3.3. In epidemiological studies, experimental designs are often targeted at finding out whether observed differences in
health outcomes among study subjects or groups are indeed real differences or may merely be due to chance [181, 368-370]. However in more complex study settings such as ecological designs, the existence of confounders and effect modifiers (intervening physical environmental media) do not lend easy interpretation of findings from studies which aim to look at the influence of SES variables on health outcomes. In other words, evaluation of the influence of SES on health disparities is difficult to achieve realistically without first understanding the influence of these variables on the physical environmental conditions.

Figure 3.3: Interaction among Area-base SES, Environmental Quality and Health
(Source: Fobil et al., 2010, IJERPH, pp125-145)

Secondly, the precise role of SES variables in determining the observed health outcomes in populations is not clearly defined i.e. whether these factors themselves alone directly influence health outcomes (e.g. issues of economic barriers to healthcare) or they do so through an intermediate (e.g. intervening physical environmental media) [46, 155, 360].
Another question to ask is: are SES variables confounders or mere effect modifiers? The answer to this question is “it depends on several considerations” both on the perspective of SES variables or on the perspective of health outcomes. As provided in the conceptual model in Fig. 3.3, if it can be established that any given SES variable influences a physical environmental variable which in turn influences a particular health outcome, then the SES variable is an effect modifier. On the other hand, if it can be demonstrated that a given SES variable has no direct association with any physical environmental variable, but has direct association with a given health outcome, then the SES variable is a confounding variable in environment and health analysis.

Consequently, given the amount of spurious effects SES variables cast upon environment and health analysis, it becomes a worthwhile undertaking to investigate the kind of effects the different SES variables exert on environmental variables in urban settings, i.e. what is the precise nature of the association between the different contextual or area-based SES variables and the different environmental variables?

Although it must be acknowledged that no standard measures of the concept of SES exist and there is only very little agreement in the literature on its definition and its exact measurement, construction of proxies for the SES variables is possible and already widely applied in SES and health inequality research [159, 366, 371]. For instance, in the absence of individual level data on social backgrounds, area-based measures of socioeconomic status constructed based on social and economic aspects of the area in which study subjects reside can be used. In Australia where this technique has already been widely applied, the units of measurement have been based on postcodes, Statistical Local Areas, Local Government Areas and Census Collection Districts. For the purposes
of construction of area based SES measures, Census Collection Districts or census clusters were adopted as the units of analysis.

The census 2000 database held several cluster level measures of socioeconomic status including educational attainment, literacy rate, school enrolment, religion, ethnicity, marital status, employment status, type of employment, economic activity status (e.g. weather employable or not, etc. There were 53 of these socioeconomic variables in total and were grouped under 4 main categories as:

(a) economic activity status
(b) educational attainment
(c) occupation
(d) place of work
(e) marital status, and
(f) ethnicity.

In the study, marital status and ethnicity were excluded because they were regarded as politically and culturally sensitive. We explored the remaining variables using PCA to determine their relationships with each other and to be able to develop a uni-dimensional measure of SES, e.g. socioeconomic zones (quintiles) for the study area. The variables used in constructing area-based socioeconomic measures were computed as a proportion of individuals with a given socioeconomic characteristic among the total number of individuals in a cluster and these were stratified by sex (i.e. the SES measures were constructed separately for males and females). These area-based measures were used as proxies for cluster level socioeconomic conditions in lieu of the traditional or
conventional measures of SES using household incomes, asset-based measures, consumption or expenditure measures, etc., because they can be more reliably measured compared to their traditional counterparts. For instance, while most people feel reluctant to talk about incomes and earnings and may not be reporting correct income levels, it is fairly easy to accurately count the number of unemployed or those in employment or economically active and economically inactive people in a survey. For this reason, the measures of economic status adopted in this study seem superior to the conventional ones.

We demonstrate here how these measures were constructed and give explanation of the rationale for their application in this analysis. The economically active and inactive cluster level population was simply computed as the as a proportion of the cluster level total population. The factors measure how active or inactive a given cluster population is in terms of self-sustaining itself through economic activities. This is a measure of the ability of the population to take advantage of economic opportunities available to all the resident population given the same level of skills. Similarly, employment and unemployment rates were computed as proportions of cluster level total populations. Conceptually the employment and unemployment status of a given population measures the ability of a given area to generate wealth to strengthen the community level social capital. The social standing of any community depends on whether its residents are working or not.

The economically active people are those who are fit and legally permitted to engage in any form of employment without cause to worry about any harm to their wellbeing. In Ghana, the legal age for first employment is 18 years at the minimum. The legally
accepted retiring age in Ghana is 60 years in public service. The legal definition of economically active age range in Ghana is 18 to 60 years [351, 372]. Strictly speaking however, the social definition of economically active population relates primarily to human capabilities and wellbeing i.e. to be able to perform a task without recourse to inimical effects on one’s health and wellbeing. But whichever way economically active or economically inactive population is defined, in terms of socioeconomic status of communities, the proportion of economically active population gives an index of how many members of communities there are potentially employable and to contribute to the pool of income or wealth of the communities. The greater the number of community members in this group, the larger this proportion and the brighter the chance to generate wealth in the communities. This means that the greater the proportion of economically active population in a given community, the higher its socioeconomic status and the smaller the proportion, the lower the socioeconomic status of the community. Conversely, the economically inactive population is potentially not employable, therefore the larger the proportion of economically inactive population of a given community, the lower the socioeconomic standing of the community.

In terms of employment and unemployment, members of a community in employment are the ones that earn income and therefore contribute to the pool of wealth in the community. Community members who are unemployed do not earn regular income and therefore do not contribute to the pool of wealth of the community. For this reason, the higher the proportion of the employed cluster population the higher the socioeconomic status of the given cluster. However, if the proportion of unemployed population in a
given community or cluster is high, then the socioeconomic status of the cluster or community is low.

3.8 Geographic Information System and Digital Map Production

The Accra metropolis consists of 1700 Enumeration Areas (EAs) drawn up for the 2000 National Population and Housing Census survey. For purposes of this study, not only geographically contiguous EAs, but also EAs with similar population characteristics were merged to produce census clusters (the units of the analyses) according to the sampling frame used by Ghana’s Statistical Service in the 2000 national population census. The boundaries of these clusters were digitised to produce polygons of the census clusters, which were merged together to produce a complete digital map of urban Accra. In the digitization process, we made use of hardcopy toposheets, some of which were drawn to scale while others were not. The toposheets that were drawn to scale were in the scale of 1:2500 resolutions and were in forms ready for direct digitisation. This was done by plotting and registering four (4) points or codes (geo-codes) of four (4) physical landmarks (arranged in a rectangle) identified on the hardcopy maps into a computer which was connected to a digitiser in MapInfo environment. Polygons of the census clusters produced were transferred into ArcView for further processing and refinement into the final digital map of Accra. Two approaches were used in creating the polygons of census clusters as follows:

Procedure (1): Procedure (1) involved the identification of key features or landmarks on the maps based on the census description of the enumeration areas. Coordinates (geo-codes) [x-y coordinates] of the physical landmarks were then taken using a geographic
positioning system (GPS). These were plotted and registered into MapInfo which was installed on a computer connected to a digitiser. The rectangles were used as reference points for various boundary lines to be traced by a digitiser.

**Procedure (2):** Procedure (2) was much simpler. The procedure involved the identification of four points (forming a rectangle) on a map already drawn to scale, but which were also found on the maps that were not drawn to scale. The scaled-maps were used as a base-maps or standards from which geo-referenced points were projected onto the non-scaled maps (i.e. maps not drawn to scale). The projected geo-referenced points now formed rectangles on the non-scaled maps. The rectangles were plotted and registered into a computer connected to a digitiser, which enabled all cluster boundary lines to be traced as with procedure (1).

The maps produced from the separate approaches were superimposed and used to validate the digitization process. A perfect overlap was evidence of consistency and proof of validity of the two processes.

3.9 Framework for Data Analysis (Estimation of Mortality Summary Measure)

The main hypothesis of this work was that:

- a disproportionately greater burden of malarial and diarrhoeal mortalities lay on residents living in neighbourhoods and communities characterized by worse urban environmental conditions with low provision and coverage of sanitation services compared to their counterparts living in neighbourhoods and communities with better coverage of these services, and
• a subsidiary hypotheses being that, excess malaria/diarrhoea mortality was associated with worse indices of urban environmental and socioeconomic conditions.

Key Considerations in the Analysis:

Units of Analysis: Census clusters – were defined by the statistical system of Ghana as a group of geographically contiguous census enumeration areas of fairly homogeneous populations based upon defined characteristics such as accessibility of population to enumerators, socioeconomic and cultural factors [351, 373-375]. For this reason and for the purpose of this study, a cluster with fairly homogeneous population was regarded as one in which all deaths had an equal probability of being enumerated regardless of cause.

Exposure: The exposure condition was defined as all physical and non-physical urban environmental and socioeconomic conditions e.g. type of urban water supply, housing characteristics, solid/liquid wastes, lagoons, type of construction materials for housing, cluster population density, etc., all of which were obtained from the 2000 Ghana census database.

Exposed Population: - all individuals, born alive and had spent at least 24 hours of life after birth, and those who lived up to the 100th Birthday. By the definition of the exposed population, the numerator excluded all foetal deaths, e.g. still births (expulsion of a product of pregnancy without any signs of life: neo- and peri-natal deaths, abortion and miscarriages). The reason for excluding all foetal deaths was that, such deaths had occurred before ever directly experiencing the exposure under consideration. Individuals dying after 100 years were taken as dying from natural causes, since immune decline
begins to be experienced at about age 60 and above. After 100 years therefore, immunity may have decreased substantially for senescence to be completely responsible for cause of death.

**Sample Size Calculation**: This was an ecological study which used already collected data from a National Population Census database and routinely available mortality data from the vital registration system. Therefore sample size calculation was neither relevant nor required. However, it was necessary to calculate and/or estimate the statistical power of the study if the probabilities (risks) of dying from malaria and diarrhoea in each of the 70 clusters were known. We assumed that PMR in each cluster constituted the probability (risk) of dying from a specific cause in the given clusters. The statistical power was then computed as a range using the smallest and the largest risk difference for each specific cause (malaria and diarrhoea) and the corresponding cluster population. The calculation of the statistical power for detecting a difference between two samples (with sample sizes s1 and s2) and known proportions or risks (*i.e.* p1 and p2) at 5 percent significance level was achieved using a computer program studysize2.0. This was done iteratively by inputting values for proportions p1 and p2 as well as population sizes s1 and s2 into the computer program. In this particular case, the smallest and the largest risk differences were used to estimate the least and largest power obtainable for malaria and diarrhoea mortality (see Table 3.1).

These were extreme theoretical values and despite the case that malaria PMRs in some clusters might have fallen within low power range (Table 3.1), the overall statistical power was still reasonable since such extreme cases were not considered in the analysis.
### Table 3.1: Power calculation of study

<table>
<thead>
<tr>
<th></th>
<th>PMR</th>
<th>Pop.</th>
<th>PMR</th>
<th>Pop.</th>
<th>Power range</th>
<th>PMR</th>
<th>Pop.</th>
<th>PMR</th>
<th>Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria</td>
<td>0.008</td>
<td>7313</td>
<td>0.017</td>
<td>2128</td>
<td>32.1-99.9%</td>
<td>0.115</td>
<td>22140</td>
<td>0.121</td>
<td>8929</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>0.011</td>
<td>9517</td>
<td>0.018</td>
<td>17369</td>
<td>74.3-99.6%</td>
<td>0.092</td>
<td>17092</td>
<td>0.182</td>
<td>8053</td>
</tr>
</tbody>
</table>

**Explanatory variables (see section 3.6):**
- cluster hygiene conditions
- cluster water supply & sanitation
- cluster socioeconomic conditions
- cluster housing & living arrangements.

**Response (outcome) variable**
- cluster level malaria/diarrhoea mortalities (the fraction of deaths due to a specific cause *i.e.* malaria and diarrhoea).

**Potential biases:**
- Potential confounders:
  - age and sex (was addressed by calculating age- and sex specific proportional mortality ratios)
  - levels of cluster registration completeness, misclassification/ascertainment biases
- chance/random errors (discussed under data quality section).

**Potential effect modifiers:**
- socioeconomics *e.g.* income status of cluster residents, level of education, *etc.*
3.9.1 Data Quality Issues and Methods for a Valid Summary Measure Derivation

It was recognized that the use of routine data had several advantages including the fact that they were cheaply available as compared to survey data, which usually took several months and lots of money to obtain. However, one major disadvantage that was contemplated was that, the use of routinely generated data also presented many quality problems. Although it was conceded that survey data too had their own quality issues, the mortality data from Ghana VRS were suspected to be burdened with the following potential biases and quality issues:

- different levels of death registration completeness by cluster,
- death record losses from recording points (registration centres) to storage point (Births and Deaths Registry),
- data input errors during data entry from death certificates onto computer,
- misclassification or inaccurate diagnoses of diseases which may have led to death ascertainment biases.

3.9.2 Different levels of death registration completeness by cluster

Leakage in death registration e.g. illegal burials, non-reporting on some deaths, etc tended to result in incompleteness of death registration which could differ by age, sex and cluster. This could have affected the overall mortality reporting by the Ghana VRS data. Table 3.2 presents the annual death registration coverage for both rural and urban areas in Ghana for years 2000 to 2006 inclusive. Table 3.3 shows that for urban Accra from 2000 to 2004. The variability in registration completeness by age, sex, cluster, etc could affect the results, interpretation and conclusions of studies which made use of these data. In
other words, mortality rates (crude rates) calculated directly from these routine data without any controlling procedures could produce misleading results. To be able to deal with this meant the use of sophisticated epidemiologic procedures to compute mortality summary measures valid for comparative purposes. In the case of death registration incompleteness, standardization procedures (e.g. either direct or indirect standardization) were deemed sufficient to handle such data quality issues.

### Table 3.2: Annual Deaths Reporting Coverage in Ghana (2000-06)

<table>
<thead>
<tr>
<th>Region</th>
<th>Year</th>
<th>Population</th>
<th>Expected Deaths</th>
<th>Registered Deaths</th>
<th>% Coverage</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Regions</td>
<td>2000</td>
<td>18,912,079</td>
<td>226945</td>
<td>45402</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>2001</td>
<td>19,422,705</td>
<td>233072</td>
<td>51639</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>2002</td>
<td>19,947,118</td>
<td>239635</td>
<td>49630</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>2003</td>
<td>20,485,690</td>
<td>204857</td>
<td>47492</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td>2004</td>
<td>21,038,804</td>
<td>210388</td>
<td>50625</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>2005</td>
<td>21,606,852</td>
<td>216069</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>22,190,237</td>
<td>221902</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Source: GSS/Ghana Births and Registry, 2006

### Table 3.3: Annual Deaths Reporting Coverage in Urban Accra (2000-04)

<table>
<thead>
<tr>
<th>Variable/Year</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expected</td>
<td>13916</td>
<td>10309</td>
<td>13947</td>
<td>13633</td>
<td>15160</td>
</tr>
<tr>
<td>Reported</td>
<td>7078</td>
<td>6468</td>
<td>6514</td>
<td>6318</td>
<td>7304</td>
</tr>
<tr>
<td>Coverage (%)</td>
<td>50.86</td>
<td>62.74</td>
<td>46.71</td>
<td>46.34</td>
<td>48.18</td>
</tr>
</tbody>
</table>

Source: GSS/Ghana Births and Registry, 2006

#### 3.9.3 Death record losses or data leakage

Death record losses were observed to be a consequence of lifting, transfer and transportation from one point to the other, storage, etc of death record files. Such losses could usually be detected directly and much less problematic than data leakage through non-reporting and/or illegal burials. Our interviews with staff at the VRS of Ghana revealed that records were first transmitted as monthly statistical summaries and then subsequently as annual death registers. This provided a feasible avenue for comparing tallies from the two data transmission processes in order to detect, account
and correct for all such losses. Data losses led to registration incompleteness and its data quality implications were handled by the same procedures used for addressing death registration incompleteness.

3.9.4 Data entry errors

Data entry errors occurred in the form of typological errors. These did not affect the data quality in a manner that the analysis could have led to invalid conclusion or deceptive outcomes. The data entry errors were generally random in nature and did not affect one cluster more than other. Errors of this nature were also random errors and their effects were distributed equally by cause and by cluster. These errors were easily detected and corrected simply by running field-code checks in Microsoft Excel 2003 and Stata 9.0.

3.9.5 Misclassification or inaccurate diagnoses (cause of death ascertainment bias)

Misclassification or inaccurate diagnoses were classified under common errors in practice and largely led to cause of death ascertainment biases. Misclassification routinely arose from the following causes:

a) physician’s lack of appreciation of the clinical symptoms for an illness,
b) physician’s prejudice about neighbourhood characteristics of the area an individual lived prior to death,
c) physician’s prejudice about socioeconomic status of the deceased,
d) physician’s prejudice about season (either wet or dry) i.e. physicians may over-diagnose malaria in wet season than in dry season,
e) imprecise predictive ability (variable predictive values) of diagnostic test or instrument.

We recognized two (2) types of misclassification (or ascertainment) biases; viz differential and non-differential misclassifications. Non-differential misclassification occurred when the amount of diagnostic error in certifying a death event as from malaria/diarrhoea or otherwise was the same in all death events presenting to a pathologist or physician. Thus, non-differential misclassification occurred when the degree of measurement or medical cause of death certification error was the same in death events occurring in different clusters. Conditions more likely to give rise to non-differential misclassification were lack of appreciation of clinical symptoms and low prediction of diagnostic test or instrument as in (a) and (e) above. Non-differential misclassification is a random error, equally distributed among all deaths in different age-groups, sexes and clusters. This meant that in different age-groups, sexes, and census clusters there would be an equal chance of assigning fever cases as malaria in different sub-population groups. In this case, if census clusters differed in the structure of their populations, the cause of death ascertainment in the different age-groups, sexes and clusters did not differ because different sub-population groups had equal likelihood of ascertainment within each sub-group (for each cause of death).

Differential misclassification (ascertainment bias) on the other hand occurred when the degree of measurement or ascertainment error was different in different death events within different subpopulation groups (e.g. age-groups and sexes) or in different clusters. Therefore if the different groups were not evenly distributed among all sub-population groups in different clusters that could lead to variation in cause of death.
ascertainment in different age-groups, sexes and clusters. McGuinness and others in a study to determine case definitions for diagnosis of clinical malaria, age- and sex-specific diagnosis of the fraction of fevers attributable to malaria (AF) varied with age and season. For infants, the AF was 51% during the wet season and 22% during the dry season. For children over one year of age, AF was 89% during the wet season and 36% during the dry season. Although this fraction differed between age-groups and between seasons, it was found to be constant within age-groups and within seasons.

Differential misclassification (ascertainment bias), a systematic form of error would gave rise to different levels in ascertainment by age, sex and by cluster. Causes of this form of ascertainment bias had been largely due to human stereotypes which include prejudices about neighbourhood characteristics and socioeconomic conditions in different clusters. As an illustration, consider that two people died of an illness which manifested as discharge of 2-3 watery stools per day (being definition of diarrhoea). Also consider that the two people died in two different geographic locations i.e. a neighbourhood with a low income and poor environmental quality – cluster “A” and the other neighbourhood with a high income population and clean environmental conditions – cluster “B”. In assigning medical cause of death presumptively without laboratory confirmation, the certifying physicians could simply conclude that diarrhoeal causes were more probable explanation of loose stools in neighbourhood with poor environmental conditions than in neighbourhoods with clean environment conditions. Under the influence of such stereotypes, physicians could inaccurately assign the cause of 2-3 loose/watery stools per day (underlying cause of death) to diarrhoea more likely so for persons coming from cluster “A” compared to those coming from cluster “B”. This meant that such biases
could lead to diarrhoea over-diagnoses in cluster “A” compared to cluster “B”. Such prejudices about socioeconomic status would lead to similar over-diagnoses in low income residential areas compared to high income neighbourhoods. Fortunately, certifying physicians look beyond these stereotypes during cause of death ascertainment in Ghana.

3.9.6 Methods for Addressing the Biases – Strengths and Weaknesses

This section has been devoted to a discussion of some of the analytical tools or procedures available for handling the data quality issues described. In the particular case of the death registration, the quality issues which were most likely to affect the results of this study in a significant way were:

- different levels of death registration completeness,
- misclassification or inaccurate diagnoses of diseases which may lead to cause of death ascertainment biases, and
- misclassification due to seasonality – seasonal misdiagnoses.

The section presents a discussion on the strengths and weaknesses of the methods available for use in handling the quality issues. The discussion of the strengths and the weaknesses then informed the choice of the method used in deriving a more valid malaria/diarrhoea mortality summary measure for this analysis. There was a wide range of techniques available for use in estimating mortality from deficient data.

However, the techniques varied in the levels of their sophistication, robustness and the extent of their adequacy to fully address all the potential quality problems. Although direct and indirect standardisation procedures, Preston-Coale and Brass methods could address the issue of variation in the levels of registration completeness, they did not
address misclassification (ascertainment bias) and the mortality data used in this study did not even meet the data requirement of those methods [376-378]. Furthermore, indirect standardisation was shown to be of limited application in this study because standard rates (available from the Ghana Demographic and Health Surveys - GDHS) were only available for under-5s and infant mortalities. In the specific case of child mortalities, these rates were not even available. Detailed description of these traditional procedures is available in literature and a discussion on them was not considered. Instead, the ensuing sections will focus attention on discussion of the novel procedures developed specifically for this study.

3.9.6.1 Wet Season Mortality to Dry Season Mortality Ratio

Misclassification associated with seasonal influences (i.e. vary from season for both malaria and diarrhoea) are very common biases. For example, it is well known that mosquitoes breed better in wet seasons than in dry seasons due to overabundance of poodles and standing pools of water in wet season compared to dry season. For this reason, physicians will most likely over diagnose both malaria and diarrhoea in wet season than in dry season. In the diagram (Fig 3.4), there are four (4) clusters (i.e. C1, C2, C3 and C4) shown in sky-blue colour. There are two sets of double-ended arrows, a set of four in green colour and another set of four in deep blue colour. The deep blue arrows with double arrows represent “within cluster variation” (wcv) in mortality and the green arrows show “between cluster variation” (bcv) in mortality as a consequence of seasonal biases in diagnoses.
In order to address seasonality biases, the task was to get a mortality outcome measure which was independent on the “between cluster variation”. The ratio of wet season mortality to dry season mortality would completely address the issue of both seasonality bias and misclassification because the two absolute parameters for mortality, from which the ratio was computed, came from same the cluster. The ratio did not depend on other clusters because its derivation did not involve mortality figures from the other clusters. This meant that the associated seasonality bias would cancel out in the resultant summary measure since its effect was the same in both the numerator and the denominator. Table 3.4 shows how to compute wet and dry season mortality ratio. The ratio addresses the problem of seasonal variation in diagnosis in that it is entirely unique to a given cluster and independent of any other cluster.

Table 3.4: Ratio of wet season mortality to dry season mortality

<table>
<thead>
<tr>
<th>Cluster</th>
<th>Wet-mot</th>
<th>Dry-mot</th>
<th>Ratio = [(wet-mot)/(Dry-mot)]</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1</td>
<td>4/1000</td>
<td>2/1000</td>
<td>2</td>
</tr>
<tr>
<td>C2</td>
<td>8/1000</td>
<td>2/1000</td>
<td>4</td>
</tr>
<tr>
<td>Cnth</td>
<td>16/1000</td>
<td>2/1000</td>
<td>8</td>
</tr>
</tbody>
</table>

**Characteristics of the ratio [(wet-mot)/(Dry-mot)].**

- it is a relative value,
- it measures the magnitude of mortality in wet season in relation to that in dry season,
such a measure considers two different values within one single cluster,

- the ratio in one cluster does not depend on another ratio in a different cluster,
- the ratio being a relative measure, it is not affected by misdiagnosis and ascertainment biases inherent in the two absolute values from which it is derived.

**Limitation of the ratio \[(wet-mot)/(Dry-mot)\]**

The ratio combines mortality figures from both wet and dry seasons in its derivation and cannot therefore be used to compare seasonal effects on the mortality outcome. A key assumption was that seasonality did not vary within cluster and therefore, the summary measure would be rendered invalid if the assumption was violated, i.e. if seasonal intensity varied within cluster.

3.9.6.2 Instantaneous Death Rate or Force of Mortality (µ)

In deriving instantaneous death rate or force of mortality (µ), a fairly homogeneous population conditions was assumed as in Preston-Coale and Brass [376, 379] but with slight amendment to the assumptions therein. The slightly modified assumption was that “the completeness of death reporting although not 100 percent complete will be the same for same ages”. This meant that probability of recording death events among individuals within same age group would be the same. On the basis of this assumption the total mortality in any given cluster was decomposed into the following components:

(a) \([_{(AC)}]\) = “all-cause of death”

(b) \([_{(SfC)}]\) = “specific-cause of death”

(c) \([_{(AOC)}]\) = “all-other-cause of death”

\[1\] \([_{(AC)}] - [_{(SfC)}] = [_{(AOC)}]\) {e.g. “all-cause of death” - “specific cause of death” = “all-other cause of death”}
A relationship was then established among the three (3) components of mortality, in a view to derive a summary measure on mortality that would be unaffected by all the five (5) data quality issues associated with mortality reporting in Ghana. In order to do this, an assumption was invoked and some logical inferences drawn from the assumption as follows:

**Main Assumption:** There is no strong reason why mortality data incompleteness (i.e. caused by under-registration of deaths, under-reporting, record losses, etc) should vary by cause (e.g. malaria or diarrhoea or hypertension, etc), “all-cause of death” (all deaths, which includes the given “specific cause” of death) and all-other cause (all death excluding a given specific cause of death) (see footnote for the mathematical relationship among the three mortality components) within age groups in a given cluster. In other words, in any one cluster, the level of mortality registration factor “k₁” (pronounced k-prime) and registration incompleteness factor “k” will be the same within age groups or within a cohort of the same birthday. The premise of this assumption is that, it is the “fact-of-death” rather than its cause (“cause-of-death”) that is important in determining whether a given death event is reported or not.

**Key Inference from the assumption above:** If this assumption holds true, then in any given cluster, say C, the “reported specific cause of death” \( [R_{(SRC)}] \) = “true specific cause of death” \( [T_{(SRC)}] \) multiplied by “reporting incompleteness factor” (k) and that the “reported all-cause of death” \( [R_{(AC)}] \) = “true all-cause of death” \( [T_{(AC)}] \) multiplied by incompleteness factor (k), see Table 3.5.
Mathematical formulation of inference above:

Expressing the above inference using cluster (C₁) values in Table 3.5, the following models were constructed:

\[ \text{[R(SfC)]}_1 = \text{T(SfC)}_1 \times k_1 \]  

and

\[ \text{[R(AC)]}_1 = \text{T(AC)}_1 \times k_1 \]

Solving for \( k_1 \), equations (1) and (2) can be re-written as:

i.e. equation (1) becomes

\[ k_1 = \frac{\text{[R(SfC)]}_1}{\text{T(SfC)}_1} \]

and

equation (2) becomes

\[ k_1 = \frac{\text{[R(AC)]}_1}{\text{T(AC)}_1} \]  

But \( k_1 \) (death registration incompleteness and/or misclassification factor in cluster C₁) in equations (3) and (4) is the same, and so (3) = (4)
\[
\frac{[R_{(SfC)}]}{[T_{(SfC)}]} = \frac{[R_{(AC)}]}{[T_{(AC)}]} = \beta \tag{5}
\]

Now, “if the quotients (\(\beta\)) of the terms on the left hand side of the equation and that on the right hand side of the equation are the same, then the quotients (\(\beta^1\)) of the numerator terms and that of the denominator terms are also the same”.

\[
\frac{[R_{(SfC)}]}{[R_{(AC)}]} = \frac{[T_{(SfC)}]}{[T_{(AC)}]} = \beta = \beta^1 \tag{6}
\]

Equation (6) gives a cross-product ratio (\(\beta\)) which can be computed (i.e. \([R_{(SfC)}]\) and \([R_{(AC)}]\) are known) from already collected mortality data. The summary measure \(\beta\), gives an estimate of the fraction of any specific cause of death (e.g. malaria, diarrhoea, hypertension, etc) in any one cluster with a given schedule of all-cause mortality. Additionally, (\(\beta\)) measures the force (stress) of mortality due to any specific cause relative to all-cause of death at any given time and it is the fraction of specific cause mortality in relation to all-cause mortality.

This means that if \(\beta_1\) in cluster \(C_1\) is not affected by \(k_1\), in the same cluster, then \(\beta_2\) in cluster \(C_2\) will not be affected by \(k_2\) in cluster \(C_2\), and that holds for all other clusters. It was then concluded that, (\(\beta\)) was an unbiased estimate of cause specific mortality in relation to all-cause mortality in different clusters and could be used to make valid comparison of specific cause mortality across different clusters.

A final consideration was the possibility to obtain (\(\beta\)) from “reported specific cause and “reported all-other cause” (i.e. all-cause mortality \([T_{(AC)}]\) minus specific cause mortality \([T_{(SfC)}]\); \(T_{(AC)} - T_{(SfC)} = T_{(AOC)}\)). Using the figures in cluster \(C_1\), as usual, this was achieved by imagining that the “reported specific cause of death” \([T_{(SfC)}]\), multiplied by \(k_1\) would give a certain value \((m_i) = \{[R_{(SfC)}]\}\) and that the “reported all-
other cause of death” \( \{T_{(AOC)}\}_1 \) multiplied by \( k_1 \) would also give another value \( d_o = [R_{(AOC)}]_1 \). Therefore, using figures in cluster \( C_1 \), in Table 1 as usual, we obtained the following mathematical expressions:

\[ [R_{(SfC)}]_1 \cdot k_1 = m_i \]  \hspace{1cm} (7)
\[ [R_{(AOC)}]_1 \cdot k_1 = d_o \]  \hspace{1cm} (8)

Now, dividing equation (7) by equation (8) as before, we obtained the following expression:

\[ \frac{[R_{(SfC)}]_1}{[R_{(AOC)}]_1} = \frac{m_i}{d_o} = \beta_o \]  \hspace{1cm} (9)

The quotient \( \beta_o \) in equation (9) is an estimate of the fraction mortality of specific cause relative to all-other cause mortality in a cluster with a given mortality schedule. This estimate is also unbiased with respect to \( k \) at cluster level. A point to note here is that \( \beta_o \), just like \( \beta \), is not affected by both misclassification and incompleteness biases for similar reasons.

Dividing equation (9) by equation (6), we obtained the relationship between \( \beta_o \) and \( \beta \) as follows:

\[ \frac{\beta_o}{\beta} = \frac{m_i}{d_o} + \frac{m_i}{d_i} = \frac{d_i}{d_o} \]  \hspace{1cm} (10)

Also, from equation (9), \( \beta_o = \frac{[R_{(SfC)}]_1}{[R_{(AOC)}]_1} \) and \( \beta \) in equation (6) = \( \frac{[R_{(SfC)}]_1}{[R_{(AOC)}]_1} \)

\[ \therefore \frac{\beta_o}{\beta} = \frac{[R_{(SfC)}]_1}{[R_{(AOC)}]_1} \cdot \frac{[R_{(SfC)}]_1}{[R_{(AOC)}]_1} = \mu \text{ (cross product ratio).} \text{ The effects of } [R_{(SfC)}]_1 \text{ also cancels out, but it does contribute to generating the final outcome estimate } (\mu) \text{ and therefore gives an indirect estimation of the contribution (fraction of deaths due to a specific} \]
cause) of \([R_{(SC)}]\) to all-cause mortality deaths in a given area with a defined schedule of mortality.

\[
\Rightarrow \frac{\beta}{\beta_0} = \frac{[R_{(AC)}]}{[R_{(AOC)}]} = \mu
\]

The quantity \((\mu)\) in equation (11); a cross product ratio, is an estimate of the fraction of deaths to a specific cause. It measures the force or stress of mortality due to a given specific cause (e.g. malaria, diarrhoea, hypertension, etc.) in a given cluster with a defined mortality schedule. Since \((\mu)\) was derived from \(\beta\) and \(\beta_0\) which were unaffected by the incompleteness and misclassification factors, it was also unaffected by these factors. In reality, \(\mu\) measures the inverse of the proportion of specific cause of death or the inverse of the force (stress) of mortality (defined as instantaneous death rate and represented as the ratio of probability function to distribution function) [377].

Attributes of \((\mu)\)

(a) \((\mu)\) varies from the value 1 to infinity (specific cause = all-cause) depending on the magnitude of the cause-specific mortality at any one point in time and its trajectory is described by a curve,

(b) the integral of the curve (i.e. area under the curve) gives the absolute value of a given specific cause,

(c) the derivative of the curve (i.e. instantaneous change) of the curve at any one point in time gives \((\mu)\), the fraction of deaths due to a specific cause,

(d) \((\mu)\) is a ratio and unaffected by the magnitudes of the numerator and denominator, and finally

(e) \((\mu)\) is not affected by the levels of uncertified cause of deaths (unknown cause of death) and death records with unknown place of residence addresses (see Table
3.5). This is because individuals without home addresses were regarded as homeless people and were excluded from the target population. Since there was no strong reason why there will be ascertainment bias by cause, records with unknown cause of death did not affect ($\mu$).

Table 3.6 summarises the main quality issues and gives logical explanation of how they can be surmounted.

**Table 3.6: Highlights of key quality issues and proposed solutions**

<table>
<thead>
<tr>
<th>No</th>
<th>Bias</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Registration incompleteness:</td>
<td>Illegal burials, relocation of aged people and data losses all ultimately result in incompleteness of death registration. But the incompleteness factor; denoted by (k), of death registration is a function of the statistical/registration system and cluster population characteristics, which cancels out in computing the ratio $\mu$. So when $\mu$ is computed for specific age groups in each cluster, the factor (k) which is the same in a given should cross out.</td>
</tr>
<tr>
<td>2</td>
<td>Misclassification :</td>
<td>Malaria/diarrhoea diagnoses or ascertainment may vary from physician to physician. But this should not vary in a given cluster because factors pre-empting misdiagnoses are constant within each cluster. This means misclassification will remain constant throughout each and therefore even out when computing for $\mu$. The diagnostic test or equipment may give variable results, but this will also be constant for a given cluster. Finally, because physicians are not assigned as per cluster basis, this error is random and should not cause ascertainment levels to differ by cluster.</td>
</tr>
<tr>
<td>3</td>
<td>Ascertainment bias based upon area characteristics</td>
<td>Given that misclassification due to physician’s personal attributes does not cause ascertainment to vary by cluster, in other circumstances, the levels in the ascertainment may still vary all the same by cluster as a consequence of variation in area characteristics, socioeconomic statuses, etc., of different clusters. But this will be expected to be the same in a given cluster (i.e. area characteristics and socioeconomic conditions within each cluster are fairly homogeneous). So in deriving $\mu$, the factors present in both the numerator and denominator will reasonably cross out.</td>
</tr>
<tr>
<td>4</td>
<td>Ascertainment bias based upon seasonality</td>
<td>Both malaria and diarrhoea diagnoses may be affected by seasons (wet &amp; dry seasons) e.g. there might be over-diagnoses in wet season compared to dry season. But the seasonality effects on ascertainment will not affect this analysis because the study considers exclusively variation in spatial components, other than temporal components. Moreover, the two seasons are experienced by all clusters equally (e.g. severity and duration) and the same levels of over-diagnosis will occur during wet seasons in all clusters. There should be no strong reason why seasonality should cause ascertainment levels to vary by cluster.</td>
</tr>
<tr>
<td>5</td>
<td>Accuracy of allocation of deaths by place and time</td>
<td>Deaths are registered in Ghana based on normal place of residence prior to death, place of death, date of death and date of registration for 99.7 percent of the events recorded for the period. These were all recorded and therefore there was no confusion about death misallocation by place and time.</td>
</tr>
</tbody>
</table>
3.10 Proportional Mortality Ratio and Fraction of due to Specific Cause

Although there was enough proof that $\mu$ was unaffected by all the data quality issues, its derivation was fraught with a lot of assumptions thus making its use less preferable to such more direct and widely applied estimates of mortality, such as Proportional Mortality Ratios (PMRs).

To surmount these data quality issues fairly robustly and more directly, a computation of proportional mortality ratio (PMR) was adopted using the routinely collected mortality data. The fraction of cluster level deaths due to specific cause was estimated from the cluster level PMRs and Table 3.7 displays the components which make up the formula for calculating the PMR.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Cluster Level Reported Mortality</th>
<th>Cluster Level True Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cluster Level Reported Specific Cause</td>
<td>Cluster Level Reported All Cause</td>
</tr>
<tr>
<td>0-4</td>
<td>$s_1$</td>
<td>$a_1$</td>
</tr>
<tr>
<td>5-9</td>
<td>$s_2$</td>
<td>$a_2$</td>
</tr>
<tr>
<td>10-14</td>
<td>$s_3$</td>
<td>$a_3$</td>
</tr>
<tr>
<td>15-19</td>
<td>$s_4$</td>
<td>$a_4$</td>
</tr>
<tr>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>95-99</td>
<td>$s_{19}$</td>
<td>$a_{19}$</td>
</tr>
<tr>
<td>100+</td>
<td>$s_{20}$</td>
<td>$a_{20}$</td>
</tr>
</tbody>
</table>

In this study, the concept of PMR was used in the context of cluster level and all-cause city level all-cause mortalities. On the basis of this, a PMR was defined as the proportion of deaths in a specific cluster that were due to a specific cause-of-death (malaria or diarrhoea) divided by the proportion of deaths in all clusters (city level) that were due to that specific cause of death (malaria or diarrhoea). The Proportional Mortality Ratio is
ideally used where information on the population at risk is not reliable or unavailable at all. Although information currently exists on population at risk at cluster level, because of high mobility among urban dwellers, data on cluster level population was not used. The PMR was not based on mortality rates; it compared the deaths due to a particular cause of death in a cluster with that which would be expected on the basis of the distribution of all deaths by cause in some standard population (the citywide population in the case of this study). A PMR greater than 100 indicates that members of that cluster were more likely than average to die of that cause i.e. malaria or diarrhoea, while a PMR of less than 100 indicates that they were less likely than average to die of that cause. This gave a caution that PMRs should be interpreted with care because the proportion of deaths from the cause of interest (i.e. malaria and diarrhoea) could be affected by the relative frequency of other causes. If mortality from all causes was low in a given cluster, a high PMR from a given specific cause (in this case malaria/diarrhoea) could be expected, even if the malaria/diarrhoea rate in that cluster was lower than the national rate. As a result, an observed excess could represent a true difference, but could also simply represent a deficit of deaths from other causes.

From Table 3.7, cluster level reported mortality included all deaths reported by the existing vital registration system (VRS) and cluster level expected mortality included the total number of deaths that would be expected if the VRS were perfect. But we know that the VRS in Ghana is imperfect and therefore the reported number of deaths should be less than the actual number of deaths expected if the registration system were complete. From the data available, the fraction of cluster level deaths due to a specific cause was computed from the cluster level PMR as follows:
Let \( s = \) reported cause specific deaths in the imperfect VRS and \( S = \) expected cause specific deaths if reporting were complete, then it follows that:

\[
s = I_s \cdot S, \quad [0 < I_s \leq 1] \quad \text{………………… (1),}
\]

where \( I_s \) is a certain incompleteness (constant) factor imposed by the strength and characteristics of the existing VRS.

Also, let \( a = \) reported all-cause deaths and \( A = \) expected all-cause deaths if the VRS were perfect, then again, it follows that:

\[
a = I_a \cdot A, \quad [0 < I_a \leq 1] \quad \text{………………… (2),}
\]

where \( I_a \) has the same connotation as in equation (1) above. Note that \( I_s \) and \( I_a \) represent the same registration parameter associated with the strength and characteristics of the VRS for cause-specific and all-cause deaths respectively. So that when both \( I_s \) and \( I_a \) take the value 1, we have a perfect VRS. In reality however, \( I_s \) and \( I_a \) are strictly less than 1 because the VRS is imperfect.

Dividing (1) and (2) we obtain,

\[
 \frac{s}{a} = \frac{I_s \cdot S}{I_a \cdot A}, \quad \text{……………………… (3).}
\]

Now, assuming there were no selective death registration or if the factors that contribute to biases in death registration were independent of cause of death so that reporting incompleteness would be the same for both specific-cause and all-cause (unbiased registration by cause), then \( I_s = I_a \)

\[
I_s \simeq I_a \quad \text{and} \quad \frac{s}{a} = \frac{S}{A} = \frac{\text{City\_Specific\_Cause}}{\text{City\_All\_Cause}} \quad \ldots (4).
\]
This then means $I$ is constant and cancels out. Note however that $0 < \frac{I_a}{I_s} < \infty$. Hence $\frac{I_a}{I_s}$ may be viewed as an adjustment factor for reported health statistics (e.g. morbidity, mortality, etc.) from an inefficient VRS. Note also that $\frac{I_a}{I_s}$ represents an under- or over-estimating factor such that if $I_s < I_a$ then the VRS under-reports specific health events (selective reporting bias against specific cause) compared to all-cause morbidity, then the procedure over-estimates the specific cause morbidity. Also, if $I_s > I_a$ then the system over-reports specific cause morbidity (selective reporting bias in favour of specific cause) compared to all-cause morbidity, the procedure would under-estimate the specific cause events.

Equation (4) estimates that the proportion of reported cause-specific deaths or the fraction of deaths due to a specific cause $PMR_{fd} = \left( \frac{s}{a} \right)$. The $PMR_{fd}$ takes account of incompleteness in death reporting as $I$ cancels out in equation (4) and could be used to estimate both age- and sex-specific mortality as in Table 3.7.

3.10 Conclusion

Of the three techniques considered (wet/dry seasons mortality ratio, instantaneous death rate and the fraction of deaths due to specific cause), two (instantaneous death rate and the fraction of deaths due to specific cause) appear to fully address all the quality issues alone. Although the ratio of wet season mortality to dry season mortality although could address most of the data issues fairly robustly; it was unable to handle seasonality biases. The instantaneous death rate (IDR) and $PMR_{fd}$ both provided adequate response to death
registration incompleteness and misclassification problems, i.e. the factors responsible for variation in death registration completeness across cluster (illegal burials, relocation of aged or some old people and data losses) and all forms of misclassification would be constant in a given cluster and within each age-class. It would therefore be reasonable to assume that they would cancel out in the computation of the two summary measures. However, the PMR$_{fd}$ would be preferred over IDR because the computation of PMR$_{fd}$ was more direct and devoid of the many assumptions associated with the computation of IDR. Both techniques assumed that there was no selective registration bias by cause.

Using the PMR$_{fd}$, all-age, sex-specific and age-specific mortalities were calculated separately for each cluster and which were then employed in multiple regression analyses and other geo-statistical models to determine the nature and levels of the association between environmental and socioeconomic variables separately for malaria and diarrhoea mortality across clusters. The PMR$_{fd}$ was employed to provide spatially explicit excess mortality risk analysis. The equation describing the general linear regression model is given by:

\[ Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \ldots \beta_n X_n + e_n \]

Where $Y$ = response variable (e.g. malaria and diarrhoea mortality ratio) in equation (2) above
$X_{(1-n)}$ = the different explanatory (environmental or census variables) or exposure variables 1 to n.
$\beta_0$ = the value of mortality ratio when the value of all the environmental variables assume the value of zero
$e = \text{error term}$
n assumes the value 1, 2, 3, … 70 (number of census clusters in urban Accra)
Other analyses involved the use of the PMR_{fd} to study the spatial association between the neighbourhood urban environmental and socioeconomic conditions on the one hand and malaria and diarrhoea mortalities on the other hand.

3.11 Consent and Ethical Consideration

The data (e.g. urban environmental data) were already in public domain and there were no ethical issues surrounding their use, except a written permission of Government’s Statistician. However ethical issues associated with the mortality data (usually kept confidential) were identified and clearance sought from the Institutional Review Board (IRB) of the Noguchi Memorial Institute for Medical Research.
Chapter Four: Results and Summary of Findings

4.1 Demographics and health

It is well established that health outcomes are not evenly distributed across different populations and that different individuals in a given population differ in susceptibility to different risk factors [380-382]. A growing body of literature exists which seeks to explain the phenomenon of social and spatial disparities in health [82, 318, 323, 380, 381, 383, 384] and a range of structural, material, and socio-cultural factors have been implicated [283, 323, 380, 385-387]. On account of changes in different levels of physiological and immune statuses across sexes and with aging, inequalities in health outcomes develop among different age groups [318, 323, 381, 383]. In literature, age and sex have been widely reported to be strongly associated with some cause-specific mortalities which have been reported to differ by age and sex [323, 381, 384].

4.1.1 Cluster population structure and distribution

Figure 4.1 shows cluster level age-specific population distribution in Accra which provided an opportunity age specific distributions among the different census clusters in this analysis. Age and sex have important influence on some health outcomes such that in comparing those health events across countries or regions, the summary measure of the given health outcome under consideration has to account for sex and age differences in the different comparison groups. For instance, a given health event could be associated with age such that if the age-structure differed for the different groups, then age could play a confounding role and thus bias the results of the investigation. This is also true for sex differences in the health outcome in the different comparison groups. For this reason,
to compare malaria and diarrhoea mortality among different clusters, a useful approach was to explore the influence of both age and sex on the cause-specific mortality outcomes.

By inspection, the population distributions in the different census clusters in Accra were fairly uniform and approximately the same in the different clusters. This perhaps meant that the all-age mortality parameter computed for each cluster was unaffected by age differences (Figure 4.1).
4.1.2. Age, and sex Specific malaria and diarrhoea mortalities

Figure 4.2 shows the proportion of malaria deaths in different age groups. The overall mean fraction of deaths due to malaria was higher than the age-specific fraction of deaths due to malaria in many of the older age-groups.

![Figure 4.2: Age-specific Malaria Mortality](image)

Age group 1-4 years had the highest fraction of deaths due to malaria followed by that for age-group 5-9 years, with age-group 15-19 years tying with the overall all-age mean. Logistic regression analysis of malaria mortality variability across age showed significant differences across age groups. The results showed that malaria mortality among 1-4-year olds was approximately four times higher than that among the under-1-year olds (OR:3.5; 95%CI: 2.75-4.55; \( p < 0.01 \)). Although the proportion of malaria mortality was lower among members of 5-9-year olds compared to that among the 1-4-year olds, it was still more than double that among the under-1 year olds (OR:2.5; 95%CI:1.85-3.26; \( p < 0.01 \)).
However, there was no significant difference in the fraction of deaths due to malaria between the under-1 year olds and that for the 10-14-year olds (OR: 0.18; 95%CI: 0.75-1.48, \( p = 0.73 \)).

![Graph showing fraction of deaths due to malaria by age and sex](image)

**Figure 4.3: Sex and Age-specific Malaria Mortality**

Figure 4.3 shows sex stratified malaria mortality across age groups. There was no evidence of a difference in malaria mortality among males and females across age (\( p = 0.79 \); 95%CI: -0.059 0.045). This perhaps meant that a malaria mortality measure calculated without adjusting for differences in sex could be valid for comparison across different subpopulation groups.

On the basis of the results from the sex- and age-specific malaria mortality, a more appropriate malaria mortality classification based on age was regrouped into seven major categories as: 0-1, 1-4, 5-9, 10-14, 15-19, 20-29 and 30+ (Fig. 4.4). Malaria mortality
differed only among the lower age groups and tended to remain fairly the same at the age of 30 years and above.

Figure 4.4: Age Stratified Urban Malaria Mortality in Accra

Figure 4.4 shows urban malaria mortality classification on the basis of differences due to age. Differences in malaria mortality among the different groups could not be ignored for the lower ages i.e. 29 years and below as shown in Fig. 4.4.

Figure 4.5: Age-specific Diarrhoea Mortality
Figure 4.5 shows variation of diarrhoea mortality across age. There was a gradual decline of diarrhoea mortality with the highest mortality among the under-1 year group and dropped sharply, reaching 6.7 percent of deaths attributable to diarrhoea among the 5-9-year age group. Diarrhoea mortality rose sharply by nearly 20 percent of the deaths among the 5-9-year group to 8.0 percent of all-cause deaths among members of 10-14 year-olds. Analysis showed no evidence of a real difference between the 0<1-year olds and 1-4-year olds (OR: 0.80; 95%CI: 0.59 1.07; \( p = 0.13 \)). Nevertheless, a strong evidence of a difference was observed between groups 0<1-year olds and 5-9 year olds (OR: 0.59; 95%CI: 041 086; \( p = 0.005 \)). Although there was only moderate evidence of a difference in diarrhoea mortality between age groups 0<1 and 10-14 (OR: 0.69; 95%CI: 0.47 1.005; \( p < 0.05 \)), a strong evidence of a difference in diarrhoea mortality was observed between the members of higher age groups i.e. diarrhoea mortality among members of 0<1 age-group was approximately half of that among members of the 15-19-year group (OR: 0.51; 95%CI: 0.34 0.75; \( p = 0.001 \)). The pattern of variability in diarrhoea mortality across age made convenient re-grouping quite the same way as that for malaria mortality.

Figure 4.6 shows sex stratified age-specific diarrhoea mortality in urban Accra. Diarrhoea mortality varied non-uniformly across the different age-groups for both males and females. Although it appeared there was a difference in diarrhoea mortality between males and females especially among the lower age-groups, the evidence of this difference was not shown to be significant (\( p = 0.37 \); 95%CI: -0.010 0.26). Additionally, there were differences in age-specific malaria and diarrhoea mortalities (Figs. 4.3 & 4.6). This very much provided evidence that although diarrhoea mortality summary measure did not
account for differences in sex, it could still be valid for comparison among different groups.

![Figure 4.6: Sex and Age-specific Diarrhoea Mortality](image)

However, age-specific diarrhoea mortality showed slightly different pattern of variability compared to that for malaria mortality. The point at which diarrhoea mortality variability showed no significant differences among the age groups was observed to be much earlier in life for diarrhoea mortality than for malaria mortality. But for purposes of uniformity in age-grouping, seven categories were produced for diarrhoea as in Fig. 4.7.
As could be shown in Fig. 4.7, the seven age-groups were based on strong evidence of differences that existed for age-specific diarrhoea mortality, especially among the lower age-groups.

4.2 Urban socioeconomic conditions and urban environmental conditions

Generally, health inequalities exist among rural and urban dwellers, different incomes groups, different gender and age-groups. The dependence on cash-for-service policies in many low-income economies has increased inequalities in access to affordable health care which tend to produce disparate health outcomes among different social groups. Wide inequalities in income levels also mean uneven access to environmental services which drive environmental health inequalities across these social groups. In literature, many studies exist which highlight health problems of the urban population in the cities of Africa, Asia and Latin America [38, 39, 358, 364]. Intra-urban differentials in social, environmental and health conditions between groups in cities are now broadly understood [174, 357] and depending on the region, between 35 and 55 percent of the populations
have incomes or consumption levels below the standard poverty line [93, 155, 362, 388].

While urban poverty is rapidly exacerbating, a marginally small but numerically consequential proportion of urban residents have lifestyles and living conditions which mirror those of the very affluent countries [77, 153, 154, 358]. Several review articles have reported widening intra-urban differentials in environmental quality conditions in the poor countries [77, 88, 153-155, 174, 325, 358-360, 362, 363, 365, 389, 390]. In Ghana, such reviews and assessments reported pervasive intra-urban environmental quality differentials in the fast growing urban centers including Accra, Kumasi, Tamale, Cape Coast and Takoradi, where deprived areas exist alongside privileged areas, distinguished only by the overall area-based socioeconomic conditions [38, 71, 215, 216, 391]. In Accra, up to 46 percent of people live in the most deprived zones [71, 215, 391]. These areas are hosts to people with the lowest educational standards, the lowest incomes and the poorest facilities in terms of water, sanitation and housing [71, 215, 391].

4.2.1 Structure of urban socioeconomic status (SES) (quintiles)

Despite increased number of interventions aimed at poverty reduction, urban poverty is deepening in Ghana with 33 percent of the poor now living in the urban areas. From 1988 to 1993, while informal urban employment grew from 66 to 83 percent of total national employment, wages fell significantly in absolute terms over the same period [392]. As a result, urban poverty has reportedly increased rapidly, with some poor groups now threatened by food insecurity and malnutrition. During the same period, the number of severely malnourished children has more than doubled. The rapid decline in income levels of urban residents had direct impact on access to health, water supply, and sanitation services. The consequence of a weak urban economy has been that the urban
residents constantly face a reality of double tragedy [393]. While 14 percent of the population without potable water supply now lives in urban areas, 40 percent of the population without sanitation coverage also resides in these urban areas. The combination of unequal access to the basic services and income inequalities directly drive health inequalities in the urban areas in Ghana.

4.2.2 Differences in environmental quality across socioeconomic quintiles

Table 4.1 shows the variation in neighbourhood urban environmental quality conditions across socioeconomic classes in a typical urban setting in a low-income economy. In general, while there was very strong evidence of differences in the levels of environmental quality with respect to total waste generation ($p < 0.001$), waste collection rate ($p < 0.001$), sewer disposal rate ($p < 0.001$), non-sewer disposal ($p = 0.003$) and the proportion of households using public toilets ($p = 0.005$), only moderate evidence of a difference in the environmental quality was observed for per capita waste generation rate ($p < 0.015$) and the proportion of households with toilet/bath facilities outside own household ($p = 0.02$) across the socioeconomic classes.

In the specific case of inter-quintile variability, whereas there was no evidence of differences between the poorest class and the lower middle class for total waste generated ($p = 0.064$), per capita waste generated ($p = 0.103$) and the proportion of waste collected ($p = 0.403$), there was very strong evidence of a difference across the higher wealth quintiles.

Also, a strong evidence of differences in neighbourhood urban environmental quality conditions was observed across the wealth quintiles; i.e. the lower middle class and
middle class \((p = 0.002)\), middle class and the upper middle class \((p < 0.001)\), the upper middle class and the high class \((p = 0.004)\) for the amount of waste generated at cluster level. For per capita waste generation, the weight of the evidence of differences was equally very strong \(i.e.\) the lower middle class and middle \((p = 0.001)\), middle class and the upper middle class \((p = 0.014)\), the upper middle class and the high class \((p = 0.010)\). Similar trend was observed for waste collection rate at cluster levels. There was even much stronger evidence of a difference across the wealth quintiles for uncollected waste (deposition rate), sewer disposal rate, non-sewer disposal rate and the proportion of households relying upon facilities outside households and public toilets (Table 4.1). Although, there were differences in the levels of inter-quintile variability of the different urban environmental quality conditions, the weight of the evidence; except for the proportion of households with pit and bucket/pan latrines, was generally strong (Table 4.1), suggesting a strong link between area-based SES and urban neighbourhood environmental quality conditions.
## Table 4.1: Socioeconomic classes and environmental health inequality

<table>
<thead>
<tr>
<th>Environmental Variable</th>
<th>SES Quintile</th>
<th>Mean</th>
<th>Coef.</th>
<th>Std. Err.</th>
<th>p-value</th>
<th>95%CI</th>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Total waste generated</td>
<td>Poorest</td>
<td>2970</td>
<td>5170</td>
<td>2742</td>
<td>0.064</td>
<td>-307 - 10647</td>
</tr>
<tr>
<td></td>
<td>Lower Middle Class</td>
<td>8120</td>
<td>9156</td>
<td>2787</td>
<td>0.002</td>
<td>3588 - 14723</td>
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<tr>
<td></td>
<td>Middle Class</td>
<td>12126</td>
<td>13748</td>
<td>2787</td>
<td>0.000</td>
<td>8180 - 19315</td>
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<tr>
<td></td>
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<td>2838</td>
<td>0.004</td>
<td>2769 - 14108</td>
</tr>
<tr>
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<td>11409</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>Per cap waste generation</td>
<td>Poorest</td>
<td>0.340</td>
<td>0.067</td>
<td>0.040</td>
<td>0.103</td>
<td>-0.014 - 0.147</td>
</tr>
<tr>
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<td>0.407</td>
<td>0.139</td>
<td>0.041</td>
<td>0.001</td>
<td>0.057 - 0.220</td>
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<td>0.041</td>
<td>0.014</td>
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<td>0.010</td>
<td>0.027 - 0.194</td>
</tr>
<tr>
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<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Proportion of waste collected (%)</td>
<td>Poorest</td>
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<td>-111</td>
<td>0.044</td>
<td>0.403</td>
<td>-0.057 - 0.139</td>
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<td>-217</td>
<td>0.045</td>
<td>0.044</td>
<td>0.003 - 0.201</td>
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<td>-238</td>
<td>0.045</td>
<td>0.016</td>
<td>0.023 - 0.222</td>
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<td>-233</td>
<td>0.046</td>
<td>0.023</td>
<td>0.017 - 0.219</td>
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<td>Richest</td>
<td>0.306</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Proportion of waste uncollected (waste deposition) (%)</td>
<td>Poorest</td>
<td>0.427</td>
<td>0.041</td>
<td>0.049</td>
<td>0.015</td>
<td>-0.110 - 0.023</td>
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<td>0.000</td>
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<td>0.000</td>
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<td>-0.325 - 0.142</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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<td>Proportion households using sewer disposal (%)</td>
<td>Poorest</td>
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<td>0.193</td>
<td>0.039</td>
<td>0.000</td>
<td>-0.271 - 0.115</td>
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<td>0.040</td>
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<td>-0.333 - 0.174</td>
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<td>0.246</td>
<td>0.041</td>
<td>0.000</td>
<td>-0.327 - 0.166</td>
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<td>0.294</td>
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<td>-</td>
<td>-</td>
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<td>Proportion of households using non-sewer disposal (%)</td>
<td>Poorest</td>
<td>0.453</td>
<td>0.099</td>
<td>0.036</td>
<td>0.008</td>
<td>0.027 - 0.171</td>
</tr>
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<td>Lower Middle Class</td>
<td>0.459</td>
<td>0.112</td>
<td>0.037</td>
<td>0.003</td>
<td>0.038 - 0.185</td>
</tr>
<tr>
<td></td>
<td>Middle Class</td>
<td>0.433</td>
<td>0.137</td>
<td>0.037</td>
<td>0.000</td>
<td>0.064 - 0.211</td>
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<td>Upper Middle Class</td>
<td>0.421</td>
<td>0.131</td>
<td>0.038</td>
<td>0.001</td>
<td>0.056 - 0.206</td>
</tr>
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<td>0.322</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>Proportion of households using pit latrine services (%)</td>
<td>Poorest</td>
<td>0.032</td>
<td>0.008</td>
<td>0.011</td>
<td>0.454</td>
<td>-0.029 - 0.013</td>
</tr>
<tr>
<td></td>
<td>Lower Middle Class</td>
<td>0.024</td>
<td>0.012</td>
<td>0.011</td>
<td>0.273</td>
<td>-0.033 - 0.010</td>
</tr>
<tr>
<td></td>
<td>Middle Class</td>
<td>0.020</td>
<td>0.013</td>
<td>0.011</td>
<td>0.231</td>
<td>-0.008 - 0.034</td>
</tr>
<tr>
<td></td>
<td>Upper Middle Class</td>
<td>0.045</td>
<td>0.001</td>
<td>0.011</td>
<td>0.950</td>
<td>-0.022 - 0.021</td>
</tr>
<tr>
<td></td>
<td>Richest</td>
<td>0.031</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Proportion of households using bucket/pan latrine services (%)</td>
<td>Poorest</td>
<td>0.043</td>
<td>0.010</td>
<td>0.018</td>
<td>0.573</td>
<td>-0.025 - 0.045</td>
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<tr>
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<td>Lower Middle Class</td>
<td>0.053</td>
<td>0.020</td>
<td>0.018</td>
<td>0.278</td>
<td>-0.016 - 0.055</td>
</tr>
<tr>
<td></td>
<td>Middle Class</td>
<td>0.063</td>
<td>0.028</td>
<td>0.018</td>
<td>0.127</td>
<td>-0.008 - 0.063</td>
</tr>
<tr>
<td></td>
<td>Upper Middle Class</td>
<td>0.071</td>
<td>0.001</td>
<td>0.018</td>
<td>0.949</td>
<td>-0.035 - 0.038</td>
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<td>0.044</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Proportion of households using facility in different house (%)</td>
<td>Poorest</td>
<td>0.071</td>
<td>0.021</td>
<td>0.009</td>
<td>0.021</td>
<td>-0.039 - 0.003</td>
</tr>
<tr>
<td></td>
<td>Lower Middle Class</td>
<td>0.050</td>
<td>0.028</td>
<td>0.009</td>
<td>0.003</td>
<td>-0.046 - 0.010</td>
</tr>
<tr>
<td></td>
<td>Middle Class</td>
<td>0.043</td>
<td>0.025</td>
<td>0.009</td>
<td>0.007</td>
<td>-0.043 - 0.007</td>
</tr>
<tr>
<td></td>
<td>Upper Middle Class</td>
<td>0.046</td>
<td>0.026</td>
<td>0.009</td>
<td>0.005</td>
<td>-0.045 - 0.008</td>
</tr>
<tr>
<td></td>
<td>Richest</td>
<td>0.044</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Proportion of households using public toilet services (%)</td>
<td>Poorest</td>
<td>0.206</td>
<td>0.101</td>
<td>0.040</td>
<td>0.013</td>
<td>0.022 - 0.180</td>
</tr>
<tr>
<td></td>
<td>Lower Middle Class</td>
<td>0.149</td>
<td>0.133</td>
<td>0.040</td>
<td>0.002</td>
<td>0.052 - 0.213</td>
</tr>
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<td></td>
<td>Middle Class</td>
<td>0.186</td>
<td>0.096</td>
<td>0.116</td>
<td>0.020</td>
<td>0.015 - 0.176</td>
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<td>Upper Middle Class</td>
<td>0.155</td>
<td>0.152</td>
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<td>0.000</td>
<td>0.071 - 0.234</td>
</tr>
<tr>
<td></td>
<td>Richest</td>
<td>0.054</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
4.2.3 Association between socioeconomic conditions and urban environmental quality

In the next stage of the analysis, a key interest was in how multiple socioeconomic factors influenced the overall neighbourhood environmental quality. The desire was to assess the relationship between area-based SES and neighbourhood urban environmental quality conditions. For example, per capita solid waste generation was regarded as an important urban environmental quality measure since it was the basis for calculating the total amount of solid waste a given population generated per unit time and often the basis of waste management planning programs (e.g. size of sanitary landfills to construct, type of tipping-trucks to import, financial capital required for solid waste transport, etc.). Authors used bivariate and multiple regression techniques to assess such relationships.

There was a positive (i.e. a unit increase in population economic inactivity resulted in an increase in per capita solid waste generation rate) association between the proportion of economically inactive cluster population (economic inactivity) and per capita solid waste generation (regression coefficient = 0.276) and the amount of variation explained by economic inactivity was 3.5 percent ($R^2 = 0.0346$). Economic inactivity was defined as the number of economically inactive residents within a given self-sustaining resident urban population who were technically dependent on economically active residents for social support and this measure was computed separately for males and females.

Despite this marginal increase, there was no evidence of association between economic inactivity and per capita solid waste generation ($p = 0.13; 95\%CI: -0.079 \text{ to } 0.631$). Additionally, a sex-stratified analysis of the economic inactivity or any of the remaining SES [i.e. for male ($p = 0.50$), and for female ($p = 0.40$)] found no evidence of association with the neighbourhood urban environmental conditions. The amount of
variation in neighbourhood urban environmental quality conditions explained by variation in each of the two SES measures separately was less than 3 percent.

However, there was an inverse association (i.e. unit increase in economic activity led to a decrease in per capita solid waste generation) between economic activity and per capita solid waste generation (regression coefficient = -0.276) and the amount of variation explained by economic activity was 3.5 percent ($R^2 = 0.0346$) and essentially the same as the amount of variation explained by economic inactivity.

Further analysis showed a moderate positive (a unit increase in urban employment rate led to a slight increase per capita solid waste generation rate) association between urban employment rate and per capita solid waste generation rate (regression coefficient = 0.566) and the amount of variation in per capita solid waste generation rate that was explained by urban employment was 4.2 percent ($R^2 = 0.042$). There was no evidence of association between urban unemployment and per capita solid waste generation rate ($p = 0.09; 95\%CI: -0.093 1.224$).

Additionally, a positive (regression coefficient = 0.884) association was observed between urban employment and urban solid waste collection rate. The amount of variation explained by urban employment was 6.2 percent ($R^2 = 0.062$). A moderate evidence of association was observed between urban employment and urban solid waste collection rate ($p = 0.039; 95\%CI: 0.046 1.721$).

Figure 4.8 depicts the relationship between urban employment rate and urban solid waste deposition rate. An inverse (regression coefficient = -1.007) was demonstrated and the amount of variation in solid waste deposition rate that was explained by urban employment was 9.5 percent ($R^2 = 0.095$). As shown, a unit increase in the proportion of
urban employment resulted in a significant decrease in urban solid waste deposition rate. A very strong evidence of association was observed between urban solid waste deposition rate and the proportion of urban employment ($p = 0.01; 95\% CI: -1.764 -0.250$).

The relationship between urban employment and the proportion of households connected to the central sewer system (sewer disposal rate) showed a positive (regression coefficient = 0.841) association. The amount of variation in the proportion of households connected to the central sewer system explained by the proportion of urban employment was 6.4 percent ($R^2 = 0.064$). This meant that a unit increase in the proportion of employed cluster population resulted in a corresponding increase in the proportion of cluster households connected to the central sewer system in the Accra metropolis. Moderate evidence of association was observed between the proportion of households connected to central sewer system and the urban employment ($p = 0.036; 95\% CI: 0.058 1.624$).
However, an inverse (regression coefficient = -1.084) relationship was observed between urban employment and the proportion of households engaged in non-sewer (improper) liquid waste disposal (Fig. 4.9). The amount of variation in non-sewer liquid waste disposal explained by the urban employment was 18 percent ($R^2 = 0.181$). A very strong evidence of association was observed between non-sewer liquid waste disposal and urban employment ($p < 0.001; 95\%\text{CI}: -1.646—0.521$).
Figure 4.9: Variation of employment rate with non-sewer disposal rate

In contrast to the strong association observed between the proportion of urban households connected to the central sewer system and urban employment, no such evidence of association was observed between urban employment and such cluster hygiene conditions which included the proportion of households with water closets (WC), proportion of households with pit-latrines, proportion of households with Kumasi Ventilated Improved Pits (KVIPs) *i.e.* a locally constructed improvised community toilet, proportion of households with pan-latrines and proportion of households using public toilets at bivariate level. This was in contrast to what was observed at community level when the area-based socioeconomic factors were aggregated and categorized into wealth quintiles. Although the area-based socioeconomic factors exhibited no evidence of association with the neighbourhood urban environmental quality conditions at the household level, a strong evidence of association was observed between the area-based
socioeconomic factors and urban environmental conditions across wealth quintiles at the community level.

In further multilevel analysis, we examined the characteristics of the area-based SE-conditions in relation to their ability to drive changes in the quality of the neighbourhood urban environmental conditions. Multiple regression analysis showed no evidence of association between total waste generated and the area-based socioeconomic variables, except residents’ occupation.

In other words, educational attainment and residents’ place of work did not appear to be important factors in driving the underlying difference in the amount of wastes generated in the residential communities. Nevertheless, a few elements from residents’ occupation category showed very strong evidence of association with the amount of wastes generated in the communities i.e. administrative and managerial occupations \( (p = 0.004) \), clerical and related occupations \( (p < 0.001) \), service occupations \( (p = 0.014) \), agriculture/husbandry/forestry/fishing/hunting occupation \( (p = 0.008) \), production/transport and equipment operators and labourers \( (p = 0.028) \), and professional technical and related workers \( (p = 0.023) \). In addition, the area-based SES did not show evidence of association with the amount of waste generated per person per day (per capita waste generation rate). While educational attainment and residents’ place of work showed no evidence of association, some variables which together represented residents’ occupation category showed substantial evidence of association with waste collection rate e.g. administrative and managerial occupations \( (p = 0.004) \), clerical and related occupations \( (p < 0.001) \), agriculture/husbandry/forestry/fishing/hunting occupation \( (p = 0.021) \), production/transport and equipment operators and labourers \( (p = 0.010) \), and
professional technical and related workers \( (p = 0.044) \). Although education level did not show evidence of association with total waste generated, per capita generation rate and waste collection rate, residents’ educational attainment showed a very strong evidence of association between waste deposition rate (proportion of wastes left uncollected) \([i.e.\ no\ education\ (p = 0.005),\ pre-school\ education\ (p = 0.001),\ middle/JSS\ education\ (p < 0.001),\ secondary/SSS\ education\ (p < 0.001),\ vocational/technical/commercial\ education\ (p = 0.014)\ and\ residents\ with\ tertiary\ education\ (p < 0.001)]\). Similarly, whereas both educational attainment and residents’ place of work showed strong evidence of association with wastes deposition rate (proportion of wastes left uncollected), residents’ occupation did not. Additionally, all but one of the 16 elements representing the residents’ occupation category showed strong evidence of association with waste deposition in the communities.

On the contrary, while educational attainment and residents’ occupation only showed moderate evidence of association with the proportion of households engaged in sewer disposal, all the elements representing residents’ place of work showed very strong evidence of association with sewer disposal rate. Both residents’ place of work and residents’ education attainment showed a very strong evidence of association with households engaged in non-sewer disposal. While the proportion of households using pit-latrine services did not show evidence of association with the area-base socioeconomic variables, two of the area-based SES; namely, residents’ education attainment and residents’ occupation showed very strong evidence of association with the proportion of households using bucket/pan latrine services. Finally, whereas only a moderate evidence of association was observed between the proportion of households using sanitation
facilities in a different house and residents’ educational attainment as well as residents’ occupation and residents’ place of work showed a very strong evidence of association with the proportion of households using facilities in a different house.

4.3 Area-based urban SES and health (urban mortalities)

In epidemiological studies, two discrete measures of SES; i.e. area-based and individual level measures, exist which have been widely applied in different levels of health analysis. Whereas the individual level measures include education, occupation, income, ownership of household durable commodities, etc., the area-based indicators are largely indices based on an array of social characteristics of residential areas usually drawn from census data, aggregate income, employment rate, etc. The association between socioeconomic status and mortality, morbidity and access to health services has been well investigated at both individual and area-based levels. However, the specific manner in which individual and area-based measures of SES influence health inequalities can differ according to the specific health conditions largely on the basis of their effects on disease aetiology and the disease management process. In this section, the results of the analysis of the association between malaria and diarrhoea mortalities on the one hand and area-based measures of SES on the other hand are presented.

4.3.1 Differentiation of urban SES

In Ghana, several demographic and economic analyses undertaken over the last ten years tended to agree that urban unemployment and poverty have been, in part, consequences of urbanization [350, 351, 353, 373-375, 394 and 1984 #1200, 395, 396]. Development
experts generally agree that the dependent economic development, poor urban management, and high population growth rates of low income economies have made them more susceptible to urbanization-induced unemployment and poverty compared to their middle and high income counterparts [397-400].

The Ghanaian national economy is largely dualistic in nature, i.e. a couple-existence of a modern, urban, capitalist sector geared toward capital-intensive, large-scale production and a traditional, rural, subsistence sector geared toward labour-intensive, small-scale production [399, 400]. As would be expected, the urban economies are also dualistic in character and are often decomposed into a formal and an informal sector economies [282, 399-402]. In the informal sector, the self-employed are engaged in a remarkable array of activities, ranging from hawking, street vending, letter writing, knife sharpening, and junk collecting to selling fireworks, engaging in prostitution, drug peddling, and snake charming [399, 400, 403, 404]. Other groups include mechanics, carpenters, small-scale artisans, barbers, apprentices, and personal servants [327, 399, 400, 404]. This has meant that the informal sector residents have highly unstable and variable incomes [402, 405]. The low and often irregular incomes have strong implications on household expenditures on healthcare which need state intervention to remove systemic financial barriers to accessing healthcare, especially by the poorest of urban residents [402, 404-407].

4.3.2 Mortality outcomes across socioeconomic quintiles
The relationship between area-based SES and all-age urban malaria as well as diarrhoea mortality in Accra has been presented. Table 4.2 gives the mean of the fraction of cluster
level mortality due to a specific cause mortality and relative mortality (RM) for all-age malaria and diarrhoea across SE-quintiles. While there was strong evidence of an overall difference in the all-age fraction of deaths due to malaria across the socioeconomic quintiles ($p < 0.001$), no such evidence of an overall difference in all-age fraction of deaths due to diarrhoea was observed across the SE-quintiles ($p = 0.288$). Additionally, inter-quintile analyses revealed that while there was no evidence of a difference in all-age malaria mortality between the middle class and the lower middle class (Mean = 0.0508, 95%CI: 0.035 – 0.067, $p = 0.087$), the upper middle class and the high class (Mean = 0.026, 95%CI: 0.013 – 0.039 $p = 0.73$), a very strong evidence of a difference was observed between the middle class and the upper middle class (Mean = 0.059, 95%CI: 0.049 – 0.069, $p = 0.006$) and somewhat strong evidence of a difference between the poorest class and the lower middle class (Mean = 0.060, 95%CI: 0.043 – 0.077, $p = 0.018$). On the contrary, no evidence of a difference at all in diarrhoea mortality was observed between any of the socioeconomic quintiles i.e. the poorest class and the lower middle class (Mean = 0.041, 95%CI: 0.028 – 0.054, $p = 0.122$), the lower middle class and middle class (Mean = 0.037, 95%CI: 0.024 – 0.050, $p = 0.262$), the middle class and the upper middle class (Mean = 0.041, 95%CI: 0.033 – 0.050, $p = 0.060$) and the upper middle class and the highest class (Mean = 0.063, 95%CI: 0.015 – 0.112, $p = 0.156$) (Table 4.2).

Generally speaking on the strength of inter-quintile RMs and the inter-quintile mortality differences for urban malaria, the first three lower quintiles did not differ very much and could conveniently be grouped together as one distinct category and the upper middle class and high class merging as another distinct category (Table 4.2, column 5).
### Table 4.2: Socioeconomics and malaria/diarrhoea mortality inequality

<table>
<thead>
<tr>
<th>Health Variable (Mortality)</th>
<th>SES Quintiles</th>
<th>Mean PMR&lt;sub&gt;adj&lt;/sub&gt;</th>
<th>95% CI</th>
<th>RM</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria</td>
<td>Richest Class</td>
<td>0.0298</td>
<td>0.0104 0.0492</td>
<td>1.000</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Upper Middle Class</td>
<td>0.0261</td>
<td>0.0133 0.0389</td>
<td>0.876</td>
<td>0.725</td>
</tr>
<tr>
<td></td>
<td>Middle Class</td>
<td>0.0592</td>
<td>0.0492 0.0692</td>
<td>1.987</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>Lower Middle Class</td>
<td>0.0508</td>
<td>0.0347 0.0669</td>
<td>1.705</td>
<td>0.087</td>
</tr>
<tr>
<td></td>
<td>Poorest Class</td>
<td>0.0599</td>
<td>0.0431 0.0766</td>
<td>2.010</td>
<td>0.018</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>Richest Class</td>
<td>0.0271</td>
<td>0.0138 0.0404</td>
<td>1.000</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Upper Middle Class</td>
<td>0.0634</td>
<td>0.0152 0.1116</td>
<td>2.339</td>
<td>0.156</td>
</tr>
<tr>
<td></td>
<td>Middle Class</td>
<td>0.0411</td>
<td>0.0328 0.0494</td>
<td>1.517</td>
<td>0.060</td>
</tr>
<tr>
<td></td>
<td>Lower Middle Class</td>
<td>0.0370</td>
<td>0.0239 0.0501</td>
<td>1.365</td>
<td>0.262</td>
</tr>
<tr>
<td></td>
<td>Poorest Class</td>
<td>0.0409</td>
<td>0.0277 0.0541</td>
<td>1.509</td>
<td>0.122</td>
</tr>
</tbody>
</table>

However, while the risk of dying from malaria among members of the poorest class was approximately 2 times (RM = 2.01) that of members of the high class, the risk of dying from diarrhoea was approximately 1.5 times (RM = 1.51) in the poorest class compared to the high class. Another striking observation was that while members of the upper middle class stood a lower risk of dying from malaria (RM = 0.88) compared to their counterparts in the high class, they were approximately 2 times more likely to die from diarrhoea compared to their high class counterparts (RM = 2.34).

#### 4.3.3 Association between mortalities and the area-based measures of SES

As the interest in this analysis was not only to assess whether there was a difference in malaria/diarrhoea mortality levels across the socioeconomic classes, but to investigate the relationship between area-based socioeconomic measures and urban malaria/diarrhoea mortality, we conducted further bi-variate and multi-variate analyses.

The general observation in the case for malaria mortality was that, while only two (the proportion of residents with vocational, technical and commercial education and the proportion of electricity, gas and water supply sector workforce) of the area-based measures of SES showed evidence of association at the bi-variate level, four (the proportion of residents with vocational, technical and commercial education, the
proportion of electricity, gas and water supply sector workforce, the proportion of education sector workforce and the proportion of residents with tertiary education) variables showed strong evidence of association at multi-variate level, controlling for all other measures of SES (Table 4.3).

Table 4.3 Association between area-base SES and the fraction of deaths due to malaria and diarrhoea

<table>
<thead>
<tr>
<th>Variable</th>
<th>SES Variable</th>
<th>Coefficient</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fraction of deaths due to malaria</td>
<td>Basis (intercept)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.10</td>
<td>0.06 0.13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Proportion of residents with vocational, technical and commercial education</td>
<td>-0.49</td>
<td>-0.81 -0.18</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>Proportion of electricity, gas and water supply sector workforce</td>
<td>-2.27</td>
<td>-3.68 -0.86</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>Proportion of education sector workforce</td>
<td>0.95</td>
<td>0.47 1.43</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Proportion of residents with tertiary education</td>
<td>-0.42</td>
<td>-0.10 -0.24</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fraction of deaths due to diarrhoea</td>
<td>Basis (intercept)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.04</td>
<td>0.03 0.05</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup>, Unit increase of the disease-specific fraction of deaths per unit increase of the SES variable  
<sup>b</sup>, Confidence interval  
<sup>c</sup>, Basic fraction of deaths due to malaria  
<sup>d</sup>, None of the area-based SES variables showed evidence of association with the fraction of deaths due to diarrhoea  
<sup>e</sup>, Basic fraction of deaths due to diarrhoea

Table 4.3 shows the association between the area-based socioeconomic measures and the cluster level fraction of deaths due to malaria and diarrhoea as obtained from multiple linear regression analyses. In this analysis, we conducted a stepwise reverse removal of variables in order to detect and deal with variables that showed strong collinearity (by evaluating the VIF) in the regression model. As shown in Table 4.3, of the 39 area-based socioeconomic variables, only 4 variables emerged to be strongly associated with the cluster level fraction of deaths due to malaria. In a sharp contrast, there was no evidence of association between the area-based measures of SES and the cluster level fraction of deaths due to diarrhoea at both bi-variate and multi-variate levels.
Fig. 4.10 is a graphical illustration of the relationship between the proportion of residents with vocational, technical and commercial education and the cluster level fraction of deaths due to malaria, which showed a strong evidence of association. It was noted that a unit increase in the proportion of residents at the level of vocational, technical and commercial education led to a significant decrease (coefficient = -0.45; 95%CI: -0.75 - 0.15, \( p = 0.004 \)) in the cluster level fraction of deaths due to malaria with 14% of the variation (\( R^2 = 0.14 \)) explained by the proportion of residents at the level of vocational, technical and commercial education at bivariate level. The strength of association observed between the proportion of residents at the level of vocational, technical and commercial education and the fraction of deaths due malaria was even much stronger (coefficient = -0.49; 95%CI: -0.81 -0.18, \( p = 0.003 \)) at multivariate level. On the contrary, while no evidence of association (coefficient = 0.07; 95%CI: -1.90 1.91, \( p = 0.994 \)) was observed between the fraction of deaths due to malaria and the proportion of electricity,
gas and water supply sector workforce at bivariate level (Fig. 4.11), at multivariate level however, a very strong evidence of association (coefficient = -2.27; 95%CI: -3.68 -0.86, \( p = 0.002 \)) was observed between the two variables.

Figure 4.11: Variation of the proportion of electricity, gas and water supply sector workforce with malaria mortality
Furthermore, whereas in bivariate analysis, no evidence of association (coefficient = -0.06; 95%CI: -0.21 0.09, \( p = 0.423 \)) was observed between the fraction of deaths due to malaria and the proportion of education sector workforce (Fig. 4.12), a very strong evidence of association was observed between the two variables at multivariate level, with a unit increase in the proportion of education sector workforce leading to a corresponding increase in the fraction of deaths due to malaria at cluster level (coefficient. = 0.95; 95%CI: 0.47 1.43; \( p < 0.001 \)).

Lastly, an inverse relationship was observed between the fraction of cluster level deaths due to malaria and the proportion of urban population with tertiary education. Although a very strong evidence of association was observed between the proportion of urban population with tertiary education and urban malaria mortality (coefficient. = -0.42; 95%CI: -0.60 -0.24; \( p < 0.001 \)) at multivariate level, there was no evidence of association.
(coefficient = -0.05; 95%CI: -0.11 0.006, \( p = 0.076 \)) between the two parameters at bivariate level (Fig. 4.13).

\[ \text{Fraction of Deaths due to Malaria} \]
\[ \text{Proportion of Residents with Tertiary Education} \]

**Figure 4.13**: Association between the proportion of residents with tertiary education and malaria mortality

4.4 Neighbourhood urban environmental conditions and health (urban mortalities)

The mere lack of sanitation, poor infrastructure and deplorable living conditions in rapidly urbanizing areas are believed to increase diarrhoea risk [38, 63, 320, 326, 408-410]. The effectiveness of a city-wide sanitation intervention on diarrhoea in large urban centres in low income countries has recently been demonstrated [408-410]. This section presents the results of the analysis of the influence of neighbourhood environmental conditions on malaria and diarrhoea mortalities.

4.4.1 Mortality differentials across urban environmental zones

Life within urban spaces is a characteristic feature of modern human ecology as the global number of cities has increased exponentially and expanded rapidly over the past century [38, 134, 320, 408]. While cities are seen as sources of creativity and technology,
often conceived as the engines for economic growth, they are also in many cases especially in low income economies centers of pervasive poverty, inequality, and health hazards from environmental agents [38, 134, 320]. In an analysis to assess the influence of neighbourhood urban environmental quality on the fraction of deaths due to malaria and diarrhea, we present the means of the fraction of deaths (mean fraction) and their respective relative mortalities (RMs) in scenarios of differing urban environmental quality conditions in a rapidly urbanizing area in a low income economy.

Table 4.4: Malaria mortality in different urban environmental zones

<table>
<thead>
<tr>
<th>Urban Environmental Variable</th>
<th>Zonation</th>
<th>RM</th>
<th>Mean fraction</th>
<th>p-value</th>
<th>95%CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total waste generation in relation to cluster population</td>
<td>Extremely Deteriorated</td>
<td>1.755</td>
<td>0.050</td>
<td>0.061</td>
<td>0.031</td>
</tr>
<tr>
<td></td>
<td>Moderately Deteriorated</td>
<td>1.769</td>
<td>0.051</td>
<td>0.018</td>
<td>0.041</td>
</tr>
<tr>
<td></td>
<td>Least Deteriorated</td>
<td>1.000</td>
<td>0.029</td>
<td>-</td>
<td>0.011</td>
</tr>
<tr>
<td>Water supply and sanitation facilities</td>
<td>Extremely Deteriorated</td>
<td>2.220</td>
<td>0.054</td>
<td>0.007</td>
<td>0.035</td>
</tr>
<tr>
<td></td>
<td>Moderately Deteriorated</td>
<td>2.041</td>
<td>0.049</td>
<td>0.004</td>
<td>0.040</td>
</tr>
<tr>
<td></td>
<td>Least Deteriorated</td>
<td>1.000</td>
<td>0.024</td>
<td>-</td>
<td>0.012</td>
</tr>
<tr>
<td>Hygiene facilities</td>
<td>Extremely Deteriorated</td>
<td>0.470</td>
<td>0.024</td>
<td>0.012</td>
<td>0.011</td>
</tr>
<tr>
<td></td>
<td>Moderately Deteriorated</td>
<td>0.931</td>
<td>0.048</td>
<td>0.690</td>
<td>0.039</td>
</tr>
<tr>
<td></td>
<td>Least Deteriorated</td>
<td>1.000</td>
<td>0.052</td>
<td>-</td>
<td>0.040</td>
</tr>
<tr>
<td>Housing structure &amp; form, construction material type and arrangement</td>
<td>Extreme Slum</td>
<td>2.349</td>
<td>0.066</td>
<td>0.004</td>
<td>0.052</td>
</tr>
<tr>
<td></td>
<td>Moderate Slum</td>
<td>1.794</td>
<td>0.050</td>
<td>0.112</td>
<td>0.040</td>
</tr>
<tr>
<td></td>
<td>Non Slum (Well built)</td>
<td>1.000</td>
<td>0.028</td>
<td>-</td>
<td>0.019</td>
</tr>
</tbody>
</table>

Table 4.4 compares the fraction of cluster level deaths due to malaria across environmental zones defined on basis of four sub-components of environmental quality, namely a) total waste generated in relation to population (per capita generation), b) water supply and sanitation, c) hygiene facilities/conditions, and d) housing structure, form and construction material type. On the basis of the “population and waste generation” sub-component, there was strong evidence of a difference ($p = 0.018$) in the risk of urban malaria mortality between the least deteriorated zone (mean fraction $= 0.029$, 95%CI: 0.011 - 0.045) and the moderately deteriorated zone (mean fraction $= 0.051$, 95%CI: 0.04 - 0.059) with a relative mortality [RM] = 1.77 between the two zones. However, there
was no evidence of a difference ($p = 0.061$) in the risk of malaria mortality between moderately deteriorated zone and extremely deteriorated (mean fraction = 0.50, 95%CI: 0.031 - 0.070) zone. Regarding “water supply and sanitation”, a strong evidence of a difference in the risk of malaria mortality was observed both between the least deteriorated zone ($p = 0.004$; mean fraction = 0.024; 95%CI: 0.012 - 0.036) and the moderately deteriorated zone (mean fraction = 0.049, 95%CI: 0.040 - 0.058) on the one hand and between the extremely deteriorated zone (mean fraction = 0.054, $p = 0.007$, 95% CI: 0.035 - 0.072) and the moderately deteriorated zone on the other hand. The relative risk death due to malaria in the moderately deteriorated (RM = 2.041) and extremely deteriorated (RM = 2.221) zones compared to the least deteriorated zone was approximately the same. Whereas a strong evidence of a difference ($p = 0.012$) in malaria mortality was observed between the extremely deteriorated (mean fraction = 0.024, 95%CI: 0.011 - 0.038) and the moderately deteriorated (mean fraction = 0.048, 95%CI: 0.039 - 0.058) zones for “hygiene facilities/conditions”, no evidence of a difference ($p = 0.690$) was observed between the moderately deteriorated and the least deteriorated zones (mean fraction = 0.052, 95%CI: 0.040 - 0.062). The risk of death due to malaria in the zone of moderately deteriorated hygiene conditions (RM = 0.931) and in the zone of extremely deteriorated hygiene conditions (RM = 0.470) was remarkably different although lower in both situations compared to that in the zone of the least deteriorated hygiene conditions. The “housing structure and form, construction materials and living arrangement” component of urban environment was classified into three categories, namely, 1) “extreme slum”, 2) “moderate slum” and 3) “non slum” conditions. While we observed a strong evidence of malaria mortality ($p = 0.004$) between the zone of
moderate slum conditions (mean fraction = 0.050, 95%CI: 0.040 - 0.061) and the zone of extreme slum conditions (mean fraction = 0.066, 95%CI: 0.052 - 0.080), no evidence of mortality difference ($p = 0.112$) was observed between the zone of least slum conditions (mean fraction = 0.028, 95%CI: 0.019 - 0.037) and the zone of moderate slum conditions (Table 4.4).

Table 4.5: Diarrhea mortality in different urban environmental zones

<table>
<thead>
<tr>
<th>Urban Environmental Variable</th>
<th>Zonation</th>
<th>RM</th>
<th>Mean fraction</th>
<th>p-value</th>
<th>95%CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population, water, housing and waste generation</td>
<td>Extremely Deteriorated</td>
<td>1.258</td>
<td>0.036</td>
<td>0.411</td>
<td>0.024</td>
</tr>
<tr>
<td></td>
<td>Moderately Deteriorated</td>
<td>1.728</td>
<td>0.050</td>
<td>0.160</td>
<td>0.033</td>
</tr>
<tr>
<td></td>
<td>Least Deteriorated</td>
<td>1.000</td>
<td>0.029</td>
<td>-</td>
<td>0.016</td>
</tr>
<tr>
<td>Water supply and sanitation facilities</td>
<td>Extremely Deteriorated</td>
<td>1.010</td>
<td>0.040</td>
<td>0.986</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>Moderately Deteriorated</td>
<td>1.100</td>
<td>0.044</td>
<td>0.799</td>
<td>0.028</td>
</tr>
<tr>
<td></td>
<td>Least Deteriorated</td>
<td>1.000</td>
<td>0.040</td>
<td>-</td>
<td>0.013</td>
</tr>
<tr>
<td>Hygiene facilities</td>
<td>Extremely Deteriorated</td>
<td>1.740</td>
<td>0.067</td>
<td>0.036</td>
<td>0.016</td>
</tr>
<tr>
<td></td>
<td>Moderately Deteriorated</td>
<td>0.917</td>
<td>0.035</td>
<td>0.618</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>Least Deteriorated</td>
<td>1.000</td>
<td>0.039</td>
<td>-</td>
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<td>0.035</td>
<td>0.027</td>
</tr>
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<td>0.035</td>
<td>0.024</td>
</tr>
<tr>
<td></td>
<td>Least Slum (Well built)</td>
<td>1.000</td>
<td>0.045</td>
<td>-</td>
<td>0.038</td>
</tr>
</tbody>
</table>

Table 4.5 summarizes urban diarrhea mortality burden in the different zones. In contrast to the mortality pattern observed for malaria, the evidence of diarrhea mortality difference across the different spatially distinct environmental zones was not as striking. While there was moderate evidence of diarrhea mortality difference ($p = 0.035$) across the 3 slum grades, there was no evidence of diarrhea mortality differential in the other components of environmental quality except for a moderate evidence of difference ($p = 0.036$) between moderately deteriorated (mean fraction = 0.035, 95%CI: 0.029 - 0.042) and extremely deteriorated (mean fraction = 0.067, 95%CI: 0.016 - 0.119) zones of hygiene conditions (Table 4.5).
4.4.2 Association between urban environmental conditions and health

The environmental quality and health analysis focuses on the relationship between malaria and diarrhoea mortality on the one hand and the neighbourhood urban environmental quality conditions on the other hand using bi-variate regression analyses.

Figure 4.14 shows the association between the number of water supply sources per house and malaria mortality. There was a very strong evidence (p = 0.007; 95%CI: 0.0020 0.0129) of association between malaria mortality and the number of water supply sources depended upon by homes.
In an assessment of the relationship between solid waste generation and malaria mortality, it was observed that a unit increase in solid generation resulted in an increase in the fraction of deaths due to malaria (Fig. 4.15), with the amount of solid waste generated accounting for approximately 10 percent of the observed variation in malaria mortality ($R^2 = 0.1095$). There was strong evidence of a strong association ($p = 0.005$; 95%CI: 3.60e-07 1.96e-06) between solid waste generation and malaria mortality.

However, the relationship between malaria mortality and per capita waste generation (a quantity that measures the amount of solid generated per person per day and the basis for determination of total waste generation) showed only weak evidence of association ($p = 0.050$; 95%CI: 0.0001 0.1227).

Fig. 4.16 shows the relationship between solid waste collection rate (proportion of the amount of collected out of the total amount generated) and malaria mortality. A unit increase in waste collection rate resulted in a unit decrease in malaria mortality.
There was a very strong evidence of association ($p = 0.002; \text{ 95\%CI: } -0.1203 \text{ -0.0294}$) between waste collection rate and the fraction of cluster level deaths attributed to malaria. The amount of variation in urban malaria mortality which was explained by the variation in waste collection rate was 14 percent ($R^2 = 0.1370$).

Similarly, an increase in the proportion of households connected to the central sewer system led to a decrease in urban malaria mortality. The amount of variation in urban malaria mortality that was explained by the variation in the proportion of households connected to the central sewer system was 15 percent ($R^2 = 0.1465$). A very strong evidence of association ($p = 0.001; \text{ 95\%CI: } -0.1311 \text{ -0.0344}$) was observed between the proportion of households connected to the sewer system and urban malaria mortality.

Fig 4.17 shows the relationship between the proportion of households connected to the Water Closet (WC) and urban malaria mortality. It was observed that an increase in the proportion of households connected to the WC resulted in a decrease in urban malaria
mortality and the amount of variation in urban malaria mortality explained by the variation in the proportion of households connected to the WC was approximately 20 percent ($R^2 = 0.1966$).

![Graph showing the relationship between proportion of households connected to the WC and fraction of deaths due to malaria.](image)

**Figure 4.17:** Variation of malaria mortality with the proportion of households connected to the WC

There was a very strong evidence of association ($p < 0.001; 95\% CI: -0.1290 \text{-} 0.0443$) between urban malaria mortality and the proportion of households connected to WC. This perhaps meant that WC which was a component of environmental sanitation service was also a strong predictor of urban malaria mortality.

It was also desired to assess the influence of hygiene facilities such as public toilets, shared-bath facility, own-bath facility, public bath, shared cubicle, whether hygiene facility was in household or outside household, etc., on urban malaria mortality. Fig. 4.18 shows the relationship between the proportion of households using public toilets and urban malaria mortality.
Although the use of public toilets has not been previously reported in literature to influence malaria transmission, it was observed that an increase in the proportion of households using public toilets resulted in an increase in urban malaria mortality in this study. The amount of variation in urban malaria mortality that was explained by a unit increase in the proportion of households depending on public toilets as hygiene facilities was approximately 15 percent ($R^2 = 0.1498$). A very strong evidence of association was observed between the proportion of urban households using public toilets and urban malaria mortality ($p = 0.001; 95\%CI: \ 0.0422 \ 0.1571$).

Similarly, when an assessment of the relationship between the proportion of households without hygiene facilities and urban malaria mortality was conducted, it was established that an increase in the proportion of households without hygiene resulted in a commensurate increase in urban malaria mortality. The amount of variation in urban malaria mortality attributable to a unit increase in the proportion of households without
hygiene facilities was approximately 8 percent ($R^2 = 0.0817$) with a strong evidence of association ($p = 0.016; 95\% CI: -0.3498 - 0.0365$) between urban malaria mortality and the proportion of households without hygiene facilities. In both bi-variate and multi-variate analyses, the response of urban malaria mortality to the different hygiene variables varied widely in direction, strength, degree and levels of association. For example, while a unit increase in the proportion of households with own-bath facilities and those with shared-bath facilities resulted in a decrease in urban malaria mortality, the degree of urban mortality variability explained was approximately 13 percent ($R^2 = 0.1249$) for households with own-bath facilities and 7 percent ($R^2 = 0.0701$) for households with shared-bath facilities. Moreover, while the strength of the evidence of association between urban malaria mortality and the proportion of households with own-bath facilities was very strong ($p = 0.003; 95\% CI: -0.0807 - 0.0177$), only moderate evidence of association ($p = 0.027; 95\% CI: -0.1325 - 0.0084$) was observed between urban malaria mortality and the proportion of households with shared-bath facilities.

Fig. 4.19 and Fig. 4.20 compare the differences in the levels and strengths of association between urban malaria mortality and the proportion of households using public bath facilities on one hand and the association between the proportion of households using shared-cubicle baths and urban malaria mortality on the other hand.
In both instances, a unit increase in the explanatory variable (i.e. the proportion of households using public baths and the proportion of households using shared cubicle-baths) resulted in a corresponding increase in urban malaria mortality. However, while the amount variation in urban malaria mortality explained by the proportion of households using public bath facilities was 8 percent ($R^2 = 0.0817$), the amount of variability in urban malaria mortality that was explained by the proportion of households using shared-cubicle baths was 12 percent ($R^2 = 0.1200$). There was a very strong evidence of association ($p = 0.003; 95\% CI: 0.0561 \text{ to } 0.2694$) between urban malaria mortality and the proportion of households using shared-cubicle baths. Although the association between urban malaria mortality and the proportion of households using public baths was equally strong ($p = 0.016; 95\% CI: 0.0327 \text{ to } 0.3139$), it was slightly weaker than that between urban malaria mortality and the proportion of households using shared-cubicle baths.
Figure 4.20: Variation of malaria mortality with the proportion of households using shared-cubicle baths

Another aspect of neighbourhood urban environmental quality that was assessed to determine its influence on urban malaria mortality was housing structure, housing characteristics and living arrangements. The city of Accra has been poorly planned; composed largely of light density buildings thrown in a sprawling fashion. The form and structure of built-up areas represent a diversity of environmental archetypes of different outlook and varying levels of neighbourhood environmental quality conditions. Indeed, the influence of different building and structure types on infectious disease transmission, especially urban malaria and diarrhoea mortalities in a rapidly urbanizing low-income economy has not been previously established. In this analysis therefore, our interest did not only focus on how the specific neighbourhood environmental quality issues influenced infectious disease mortalities, but also how the different structural designs and structure types were linked to urban mortalities. Classified broadly under the heading
“housing construction material type and living arrangements” both bi-variate and multiple linear regression procedures were conducted on several variables related to the structure and form of buildings, construction material type, living arrangements i.e. number of persons per unit, etc. Under this category, of a total of fifteen variables (e.g. proportion of standalone structures, proportion of semi-detached structures, proportion flats/apartment structures, proportion of compound structures, proportion of huts, proportion of mud-brick structures, proportion concrete-brick structures, proportion of bamboo structures, etc.) were used in both bi-variate and multiple regression analyses. Out of the several variables, only three showed significant association with urban malaria mortality. The three variables that showed significant ($p \leq 0.05$) association with urban malaria mortality were: 1) the proportion of standalone structures, 2) the proportion of flats/apartment buildings and 3) the proportion of compound structures which housed multiple households.

Fig. 4.21 shows the relationship between the proportion of standalone structures and urban malaria mortality. In this investigation, it was observed that a unit increase in the proportion of standalone structures led to a large decline in the fraction of deaths due to urban malaria, with the amount of variation in urban mortality that was explained by a unit increase in the proportion of standalone structures being as large as 21 percent ($R^2 = 0.2051$). There was a very strong evidence of association between the proportion of standalone building structures and urban malaria mortality ($p < 0.001; 95\%CI:-0.1742 – 0.0618$).
Figure 4.21: Variation of malaria mortality with the proportion of standalone households

In a penultimate assessment, a unit increase in the proportion of flats/apartment structures resulted in a decrease in the fraction of deaths due to urban malaria. Approximately 7 percent \( R^2 = 0.0730 \) of the variation in the fraction of deaths due to urban malaria was explained by a unit increase in the proportion of flats/apartment building structures. Nonetheless, there was a moderate evidence of association \( (p = 0.024; 95\% CI: -0.1833 \text{ to } -0.0136) \) between the proportion of flats/apartment structures and urban malaria mortality compared to the very strong evidence of association observed between urban malaria mortality and the proportion of standalone building structures.
In a more or less extended analysis, the relationship between the proportion of compound structures and urban malaria mortality was assessed. Fig. 4.22 shows the relationship between the proportion of compound building structures and urban malaria mortality and it was observed that a unit increase in the proportion of compound structures resulted in a corresponding increase in urban malaria mortality. A little over 21 (R^2 = 0.2126) percent of the variation in urban malaria mortality was explained by a unit increase in the proportion of compound building structures. The observed evidence of association between the proportion of compound building structures and urban malaria mortality was very strong (p < 0.001; 95%CI: 0.0381 0.1045).

4.5 Mapping urban malaria/diarrhoea mortality

Using the PMR_{fd}, we explored spatial relationships and developed mortality distribution maps separately for malaria and diarrhoeal deaths in Accra.
Figure 4.23 shows the spatial distribution of the observed malaria and diarrhoeal deaths in Accra. The spatial patterns displayed represent cluster level fraction of deaths due to malaria and diarrhoea. The fraction of deaths due to each cause ranged from 0 to 0.12 for urban malaria and from 0 to 0.33 for urban diarrhoea mortality. Whereas the distribution of diarrhoea mortality was heterogeneous with high mortality ratios widely scattered around the Korle Lagoon, malaria mortalities tended to be more homogenous with only a few hot spots in Accra West, distributed roughly radial to the lagoon. By visual inspection, the observed patterns of mortality distribution showed no strong clustering for either urban malaria or diarrhoea mortality (Fig. 4.23). On account of the fact that no evidence of clustering was detected by visual analysis subsequent autocorrelation analyses were conducted, but first on a global scale using global Moran’s $I$ and then on a local scale using the Local Indicator Spatial Autocorrelation (LISA) analysis.

4.5.1 Spatial autocorrelation – Global Moran’s I

The preliminary spatial analyses on the basis of pictorial distributions showed no evidence of spatial clustering for both malaria and diarrhoea mortalities. For that reason, we conducted further spatial analysis, applying different neighbourhood weights (Inverse...
Distance, Inverse Distance squared, Queen and Rook Contiguity and $k$ Nearest Neighbours) to ensure a coverage of all possible and plausible neighbourhood relationships.

Table 4.6 shows the results of spatial autocorrelation analysis of malaria and diarrhoea mortalities, allowing for different simulations of spatial relationships to be conducted among the clusters at global (city-wide) level (i.e. at inverse distance, inverse distance squared and polygon contiguity). Both malaria and diarrhoea deaths did not show any evidence of clustering at the spatial relationships considered (Table 4.6) although both diseases showed high level of randomness (Moran’s $I = 0.0123$, $p = 0.68$ for malaria and Moran’s $I = -0.0647$, $p = 0.33$ for diarrhoea) when inverse distance was considered.

Similar pattern of random distribution was observed when inverse distance squared was considered (i.e. Moran’s $I = -0.0432$, $p = 0.85$ for the distribution of malaria deaths and Moran’s $I = -0.0605$, $p = 0.69$ for the distribution of diarrhoea deaths).

<table>
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<th>Spatial Relation</th>
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<th>Diarrhoea</th>
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<td>Inverse distance squared</td>
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<tr>
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<tr>
<td></td>
<td>Expected Index</td>
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<td></td>
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<td>Polygon contiguity first order</td>
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We considered the polygon contiguity first order as a third and final simulation of spatial relationships among the clusters. The results from the nearest neighbour analyses also showed strong evidence of randomness in the distribution of both malaria (Moran’s $I = 0.032, p = 0.52$) and diarrhoea (Moran’s $I = -0.0276, p = 0.82$) deaths (Table 4.6). In consideration of nearest neighbour analysis, no evidence of clustering was observed except for six and seven nearest neighbours (6NN and 7NN) ordering where the closest to clustering was a 5-10% likelihood that the clustered pattern was by chance. While eight and ten nearest neighbours (8NN and 10NN) ordering were somewhat clustered, the pattern was flagged to be due to random chance (i.e. Moran’s $I = 0.0505, p = 0.22$ for 8NN and Moran’s $I = 0.0500, p = 0.16$). Other nearest neighbour ordering i.e. 4NN (Moran’s $I = 0.0288, p = 0.58$), 5NN (Moran’s $I = 0.0543, p = 0.31$) and 6NN (Moran’s $I = 0.0898, p = 0.095$) that were considered did not show even subtle evidence of clustering. No evidence of significant ($p \geq 0.05$) global clustering was detected for both malaria and diarrhoea mortalities in any of the neighbourhood scenarios considered at the global (city-wide) level.

4.5.2 Spatial autocorrelation – Local Indicators of Spatial Association (LISA)

This section presents the results of further spatial analyses using univariate Local Indicators of Spatial Association (LISA) which were conducted to assess the local scale of clustering for both malaria and diarrhoea mortalities. In each case, a first order queen contiguity was utilized as neighbourhood condition. In this approach the mean Local Moran value was 0.042 for malaria and -0.041 for diarrhoea respectively. The range of the Local Moran values was from -1.27 to 1.27 for malaria with a standard deviation of
0.39 and from -2.59 to 0.60 for diarrhoea with a standard deviation of 0.35. In order to increase the power of cluster detection, spatial sampling in the order of 9,999 permutations was conducted.

The choropleths occupying the left half of both Figures 4.24 and 4.25 display those locations with a significant Local Moran statistic classified by type of spatial correlation. HH identifies those clusters with high malaria mortality lying near clusters with high malaria mortality, HL identifies those clusters with high malaria mortality lying near clusters with low malaria mortality and LL identifies those clusters with low malaria mortality lying near clusters with low malaria mortality. Thus, HH and LL clusters indicated a clustering of similar values, whereas the HL and LH clusters gave an indication of spatial outliers. Areas with plain white backgrounds were locations that showed no significant Local Moran statistic ($p \geq 0.05$). The choropleths occupying the right half of both figures show the level of statistical significance (i.e. $p$-values or the probability of the distribution). The locations with a significant Local Moran statistic were shown in different coloration, depending on the significance level. In this study, four significance levels were shown as $p = 0.05$, $p = 0.01$, $p = 0.001$, $p = 0.0001$ with dark green shade being the strongest extreme of $p = 0.0001$ and red colouration being the lowest extreme of $p = 0.05$. 
Figure 4.24 shows a spatial choropleth for malaria mortality distribution displaying those locations with a significant Local Moran statistic classified by type of spatial correlation: bright red for the high-high (HH) association, bright blue for low-low (LL), white for no clustering, and brick red for high-low (HL). One location with strong evidence ($p < 0.0001$) of clustering i.e. one hot spot (high-high location) near the Korle Lagoon, three cold spots (low-low locations), two of which were located in south-central Accra and one in the north-eastern quadrant of Accra were observed. In addition, there were two outliers in south-central Accra. A total of nine significant ($p \leq 0.05$) clusters were observed in the study area for malaria mortality. While the four HH clusters were located adjacent to one another and bordering the eastern shoreline of the Korle Lagoon ($p$-range from $< 0.001$ to $0.05$), two of the three LL clusters were observed in southern central Accra ($0.01 < p < 0.05$), one adjacent to the coast, the other one slightly north-west of the Korle Lagoon. The third LL cluster ($0.01 < p < 0.025$) was located at the north eastern outskirt of the city. Furthermore, two HL clusters ($0.001 < p < 0.01$) were observed in south central Accra and in close proximity of the coastline.

The choropleth for diarrhoea mortality distribution (Fig. 4.25) displays four cold spots (low-low locations) showing slightly lower evidence ($p = 0.05$) of clustering compared to
malaria mortality and two outliers lying opposite sides of one of the cold spots. Although the choropleths for both malaria and diarrhoea distributions showed clustering at the local level, that for malaria was slightly stronger compared to for the distribution of diarrhoea mortality. Unlike the case for malaria mortality, LISA analysis of the fraction of deaths due to diarrhoea showed evidence of six significant ($p \leq 0.05$) but weakly clustered distribution within the study area. Two of the four LL clusters which showed very strong evidence ($0.01 < p < 0.05$) of cold-spots were detected in the eastern margins of the city while the other two which were much smaller in size were observed in the west. There were also two HL clusters ($0.025 < p < 0.05$) lying to the mid-eastern stretches of the city, sandwiching one of the large LL clusters (Fig. 4.25).

![Figure 4.25: Map of Accra showing spatial clustering of diarrhoea mortality](image)

4.6 Vulnerability and Excess Mortality assessment

The results of the stepwise removal multiple regression modelling showed no significant association between diarrhoea mortality and the neighbourhood urban environmental and socioeconomic and therefore excess mortality maps were created for malaria mortality only. On the excess mortality maps, while values less than one (greenish colours) indicated locations with fewer than expected events, values larger than one (reddish
colours) indicated locations with more than the expected events. The deep red and deep green colourations represented the upper and lower extreme values respectively. Census clusters showing excess rates larger than one had a higher risk of mortality than the average risk in the whole study area for the particular explanatory variable used.

The two choropleths in Fig. 4.26 show excess malaria mortality applying the occupation of residents as potential risk factor in Accra. The choropleth to the right shows retail sector workforce as a potential risk factor for excess malaria mortality while that to the left presents administrative sector workforce as a potential risk factor for the same condition. As shown by the intensities of reddish colouration of the choropleth to the left, administrative sector workforce tended to contribute more to the observed excess malaria mortality than retail sector workforce i.e. locations with excess malaria mortality tended to occur in the western part of Accra with relatively higher proportion of administrative sector workforce (Fig. 4.26). In contrast, the choropleth to the right showed that lower than expected number of malaria deaths tended to occur in the western part of Accra, coinciding with locations which had lower proportion of retail sector workforce.
Fig. 4.27 displays the excess malaria mortality with educational status as a potential risk factor. As can be gleaned from the choropleth, only one small area close to the Korle Lagoon had excess mortality far above what was expected. The western part of Accra was observed to have elevated mortality although less pronounced compared to the case for which administrative sector workforce was considered as a risk factor and much the same pattern compared to the case for which retail sector workforce was considered as a risk factor.

Fig. 4.28: Excess malaria mortality in relation to hygiene, water and sanitation
The choropleths in Fig. 4.28 show excess malaria mortality considering the proportions of households with pipe-borne water supply source outside and those connected to WC as the underlying risk factors. Locations with excess malaria mortality were consigned to the western part of Accra when proportion of households connected to water closet (WC) was imposed as the potential risk factor (Fig. 4.28, right half). On the other hand, when we imposed the proportion of household with pipe outside as the potential risk factor, we observed fewer locations with excess malaria mortality than in the case for which the proportion of households with WC was imposed as the potential risk factor. In both cases however, locations to the eastern parts of the city recorded fewer than expected malaria mortality while several clusters in the western part of the city exhibited more than expected number of malaria deaths.

Figure 4.29: Excess malaria mortality in relation to housing type

Figure 4.29 shows an excess mortality map considering housing type as the underlying risk factor driving malaria mortality in Accra. Housing type was defined as the type of built units, i.e. compound units, single units, construction material types, etc. The
choropleth represents the proportion of separate or stand-alone structure as the potential risk factor for malaria mortality. Areas with more than expected malaria mortality and those with less than expected mortality appeared to occur randomly, although clusters with more than expected malaria mortality tended to be more common in the western part of the city while those with lower than expected malaria mortality were more ubiquitous in the eastern half of the city.
Chapter Five: Discussions

The discussion section has been structured into several parts to reflect the different aspects of the analysis conducted. In conformity with the analyses section, a discussion on cluster demographics and health is presented first and which is followed by a discussion on socioeconomics and environment analysis. A discussion on socioeconomics and health was presented after which we present a discussion on environment and health analysis. Finally, a discussion on cluster analysis and geographically weighted regression concludes the section on discussion.

5.1 Cluster population, demographics and health inequalities

The study observed that malaria mortality in Accra was lowest among the under-1 year olds and highest among members of group 1-4 years. This observation was consistent with several studies reported in literature. Maternal environment/factors provide protection against malaria until after 1 year when children would presumably begin to lose the immunity conferred by maternal factors [80, 411-413]. The consistency of the findings in this study with those reported in literature is a confirmation that the summary measure of mortality computed for the study from the routine data was a valid measure of mortality. In the study, malaria mortality was shown to be low among under-1 year olds, but increased to about 30 percent of all deaths among members of 1-4 years. This observation was attributed to immune protection conferred by maternal environment.

Despite ample evidence in literature establishing that pregnant women were at increased risk of malaria infection, newborns and infants were also reported to inherit some maternal immunity against malaria during the first few months of life [80, 318, 380, 411]. This epidemiological paradox reflected the fact that the underlying interactions between
the *Plasmodium falciparum* parasite, the mother, and the foetus were complex, incompletely understood and still needed further scientific inquiry [412]. In the case of pregnancy related vulnerability to malaria infection, the mechanisms that explain the increased susceptibility were reportedly not yet fully understood. However, new insights suggested that an initial accumulation of parasitized erythrocytes in the blood spaces of the placenta was a key feature of maternal infection with *P. falciparum* [414]. It has been reported that placental parasites expressed surface ligands and antigens that differed from those of other *P. falciparum* variants, facilitating evasion of existing immunity, and mediate adhesion to specific molecules, such as chondroitin sulfate A, in the placenta [412-414]. The polymorphic and clonally variant *P. falciparum* erythrocyte membrane protein 1, encoded by var genes, which reportedly binds to placental receptors in vitro, was suspected to be the target of protective antibodies [385, 414-416]. A suggested end-stage mechanistic path then appeared to be an intense infiltration of immune cells, including macrophages, into the placental intervillous spaces, and the production of pro-inflammatory cytokines often occurring in response to infection, and were reportedly associated with low birth weight and maternal anaemia [283, 414, 417]. Expression of alpha and beta chemokines have been ultimately reported to be responsible for the initiation or facilitation of this cellular infiltration during placental malaria [385, 414, 415]. A characteristic feature of malaria infection in pregnancy has been reported to be its strong association with maternal and foetal morbidity and mortality [411]. There have been reports that the severity of the disease during pregnancy depends on the level of pre-pregnancy acquired immunity against malaria, and the consequences of infection were more severe in non-immune women [80, 411, 413]. For instance, in highly endemic
areas, the frequency and severity of the infection have been reported to be higher in primigravida and decreases with increasing parity [80, 412, 418]. However, in non-immune women, the risk has been reportedly similar across the parity gradient and malaria has been suggested to be an important direct cause of maternal mortality [80].

The second observation was that urban malaria mortality began to decline after age four, suggesting an increased level of protection against malaria mortality with increasing age. This perhaps meant that immunity against malaria probably starts to develop after age 4 years with malaria mortality beginning to decline sharply until age 24 (Figure 4.2). This then follows prolonged age-dependent malaria mortality stabilization after 25\textsuperscript{th} year on until age 65 when age-dependent mortality levels began to increase probably due to senescence or old-age. This observation has been consistent with findings from studies in other regions in Africa [323, 381, 382, 419]. For example, in a study to examine malaria distribution in 543 villages in central Ethiopia over a 4-year period, a decrease in malaria incidence which fit a quadratic model was observed with increasing age. In addition, a significant difference in the malaria incidence density ratio (IDRs) was detected in males but not in females [380].

There is overwhelming evidence of health inequalities, especially inequalities/differences in malaria cause-specific morbidity and mortality across age, space, time, seasons and sub-population groups. Pregnant women and infants are reportedly particularly susceptible to malaria cause-specific morbidity and mortality [80, 323, 380]. The risks of morbidity and mortality associated with malaria have been reportedly characterized by spatial and temporal variation across the known transmission zones [82, 380, 381]. However, no evidence of association was found between malaria mortality and sex. The
observed malaria mortality did not differ between males and females. Although, previous studies present conflicting findings on the differences in malaria susceptibility in males and females, a general consensus has been that the case fatality rate remains fairly same in both sexes [420, 421]. This is consistent with the findings from the present study which observed no difference in malaria mortality between males and females.

On the other hand, the observed urban mortality was highest among the under-1 year olds (Fig. 4.5). Unlike the pattern shown by malaria mortality, maternal factors did not appear to confer any kind of protection against urban diarrhoea mortality for any age group. This was evident from the high levels in urban diarrhoea mortality among members of under-1 year olds with a progressive decline across the age groups as individuals begin to develop immunity with age. This observation has been consistent with findings from other studies which established that maternal environments did not confer any protection against infant diarrhoea [298, 422, 423]. There was no evidence of association between diarrhoea mortality and sex since the observed diarrhoea mortality did not differ significantly between males and females.

5.2 Urban socioeconomic conditions and urban environmental conditions

In this analysis, the association between area-based socioeconomic conditions and neighbourhood urban environmental quality conditions was assessed. Often, studies which sought to evaluate the influence of socioeconomic status on health inequalities have neglected such important intermediate variables as the physical environmental conditions (environmental media), which have direct influences on health outcomes. Poor environmental quality provided condition for insect vector breeding and ultimately influences vector-borne disease transmission (e.g. mosquito, an important agent for
malaria transmission, common housefly as a mechanical vector for many microbial diseases, including diarrhoea, enterohaemorrhagic fever, etc.).

Environmental burden (e.g. local sanitation) has been generally understood to be heavier in poor communities and declined as communities became wealthier [78]. In urban areas where consumption of goods and services per person was usually high, residual deposition (e.g. waste production) was also high in those areas. In rural communities, consumption of goods and services and waste production were generally understood to be much lower per unit compared to urban areas. However, the high consumption and high residual deposition (waste production) in the urban areas have not been backed by equitable distribution of wealth thus leaving some of the urban communities financially weak to be able to manage the waste produced. In this study, the observed varied levels of influence of the area-based SES on spatial changes in the quality of the neighbourhood urban environmental conditions were suggestive that the area-based SES did not exert the same degree of influence on the quality of the neighbourhood urban environmental conditions. While some of the area-based socioeconomic variables were important in driving changes in the quality of some neighbourhood urban environmental conditions, they did not show any perceived influence on some other components of the neighbourhood urban environmental quality conditions. For example, whereas education level did not show evidence of association with total waste generated, per capita generation rate and waste collection rate, waste deposition rate (proportion of wastes collected) was observed to be strongly associated with residents’ educational attainment.
However urban employment, urban unemployment, educational attainment, residents’ place of work and residents’ occupation have demonstrated high reliability as measures of area-based SES. The nature of the associations observed between neighbourhood urban environmental conditions on the one hand and urban employment and urban unemployment on the other hand was consistent with previously reported studies [38, 355].

Although a unit increase in urban unemployment resulted in a marginal decrease in per capita solid waste generation (regression coefficient = -0.566), there was no evidence of association between urban unemployment and per capita solid waste generation ($p = 0.09; 95%CI: -1.224 \ 0.093$). Nonetheless, a positive relationship was observed between urban employment and per capita solid waste generation rate. In this case, once in both instances no evidence of association was observed between the two area-based SES measures and per capita solid waste generation rate, urban unemployment and urban employment were probably not good predictors of waste generation. However, some studies have observed association between per capita waste generation rate and income levels (employment provides opportunities for earning incomes) [38, 354, 355, 424].

Additionally, whereas there was moderate evidence of association between urban employment and the proportion of households connected to the central sewer system, a substantially stronger evidence of association was observed between urban unemployment and proportion of households engaged in non-standard practices of liquid waste disposal. This probably meant that the implementation of Ghana’s poverty reduction strategies (GPRS) without consideration to bridge urban unemployment gaps could exacerbate the widening urban health inequalities [153, 154, 174, 181].
5.3 Area-based versus individual level SES

Socioeconomic status measures the social circumstances in which an individual lives and experiences. Such social circumstances at individual level have been measured by a wide range of proxy indicators, namely income levels, expenditures, ownership of durable personal assets, etc. These individual level measures of SES differ markedly from area-based measures ones which by definition connote the social conditions of the area in which an individual resides. Much as the individual level and area-based measures of SES differ conceptually, the outcome of their use in assessing health inequalities among different social groups also differ significantly. In a study to quantify the agreement between area-based SES measures and SES assessed at the individual level, Demissie and others observed a substantial discrepancy between area-based SES measures and SES assessed at the individual level [425].

5.4 Area-based urban SES and health (urban mortalities)

In literature, studies which have explored the relationship between socioeconomic conditions and health have varied widely in approach in relation to the nature and type of the socioeconomic variables and the type of health outcomes investigated [98, 312, 426-431]. While most documented studies have examined the role of individual level socioeconomic conditions in determining the status of human health in a variety of settings and across several health outcome measures, only few studies have explored the relationship between area-based measures of socioeconomic conditions and these health outcomes [61, 270, 432-438]. In particular, whereas the influence of social conditions on chronic diseases and their mortalities and much lesser extent for infectious diseases and their mortalities has been more widely studied, not very much has been investigated on
the influence of these condition on malaria and diarrhea mortalities [370, 439-444]. Although infectious diseases have been reported to exhibit significant spatial clustering; especially malaria and diarrhoea, of the few studies reported in literature none has explored the association between area-based measures of SES and malaria and diarrhoea mortalities [62, 63, 438, 445-448]. Moreover, a number of living standard surveys conducted in Ghana have reported a much worsened urban poverty as well as widened income and socioeconomic inequalities particularly in urban areas during the period between 1990 and 2004 [350, 375] and many of the studies have linked the increased health inequalities to widening socioeconomic inequalities [152, 268, 326, 449-452].

5.4.1 Mortality outcomes across socioeconomic quintiles

In this study, an exploration of the relationship between several area-based measures of SES and the fraction of deaths due to malaria and diarrhoea was conducted in Accra using linear multiple regression analyses and other models. A remarkable observation was an overall reduction in the inter-quintile mortality risk differentials for both malaria and diarrhoea from the 1993 study which reported five times risk of dying from both malaria and diarrhoea in the poorest class compared to that in the high class [30]. The observation provided empirical evidence in demonstration of a substantial reduction in malaria and diarrhoea mortality differentials across the socioeconomic quintiles from a baseline study in the early 1990s. Despite the deepened socioeconomic inequality gaps across the social classes in Accra, mortality inequality gaps had narrowed for both malaria and diarrhoea, but more so for diarrhoea than for malaria mortality. A plausible explanation for this observation was attributed to the implementation of a mix of health policy over the period considered could perhaps have been effective in addressing any
associated mortality inequalities due to exacerbated urban poverty and wider gaps of urban socioeconomic inequalities. The inter-quintile mortality differentials were smaller in the case of diarrhoea mortality than in the case of malaria mortality, suggesting that there was greater equalization in diarrhoea mortality inequality across quintiles than that for malaria mortality compared to the situation in the early 1990s. This could be explained by the fact that the health policy mix might have been more effective in addressing inequality gaps for diarrhoea mortality compared to malaria mortality. Alternatively, this could have also meant that the area-based measures of SES had no conceivable linear relationship with the urban diarrhoea mortality at aggregate levels. Additionally, it was observed that while many of the area-based measures of SES did not show evidence of association with urban malaria at bivariate level, a reasonable number showed very strong evidence of association with urban malaria mortality at multivariate level, very much starkly in contrast to similar studies reported in literature [61, 436, 453]. This observation probably meant that some of the area-based measures of SES had blurring effects on others for urban malaria mortality, which was suggestive that those area-based measures of SES could have important implications for urban malaria mortality at household level, but less likely so at community level. The observations also suggested that the interaction of different environmental factors could be both synergistic (i.e. reinforce one another) and antagonistic (i.e. blur the effect of each other) at aggregate level.

Moreover, there were remarkable non-uniformities in the risk of mortality for both malaria and diarrhoea among the different socioeconomic quintiles. For instance, the lower middle class and the middle class had lower risk of urban malaria mortality
compared to the high class. However, the risk of urban diarrhoea mortality in the same group was nearly four times higher than that in the high class. The highly irregular pattern of inter-quintile mortality differentials for urban malaria and diarrhoea mortality could be explained by the fact that perhaps socioeconomic factors alone were not exclusively responsible for the inter-quintile mortality gaps. An opinion put forward in relation to this observation was that other factors, such as lifestyle, differences in health seeking behaviour, traditional beliefs, taboos, etc., might have accounted for the inter-quintiles mortality anomalies observed for the middle class.

5.4.2 Association between urban mortalities area-based measures of SES

Of the large number of area-based measures of socioeconomic conditions explored, only four showed strong evidence of association with urban malaria mortality. For instance increase in the proportion of individuals with vocational, technical, commercial and tertiary education led to a decrease risk of urban malaria mortality in the given area. This could be due to the fact that urban areas with individuals in these categories were probably more likely to take malaria preventive and treatment measures more seriously compared to areas with high proportion of education sector workforce. This observation was perhaps empirical evidence in support of the inclusion of these measures of SES as potential risk factors for urban malaria mortality in future case-control studies aimed to determine city-wide distribution of risks for urban malaria mortality at both household and community levels in rapidly urbanizing settings. Finally, urban areas with high proportion of electricity, gas and water supply sector workforce tended to be associated with higher urban malaria mortality and perhaps served as evidence that those areas could constitute important hotspots and zones of vulnerability to urban malaria mortality. This
in turn provides evidence in support of appropriate targeting of scarce resources in those areas if urban malaria control programs were to be cost-effective.

5.5 Neighbourhood urban environmental conditions and health (urban mortalities)

Both solid and liquid wastes have been widely known to constitute important breeding media for insect vectors including opportunity to create stagnant pools of water for mosquito breeding. As a consequence, increased waste collection decreases habitat space and capacity for the larvae of the mosquito which is the main vector of malaria parasites - *Plasmodium falciparum*, *P. vivax*, *P. ovale* and *P. malariae*. This probably leads to a decrease in malaria transmission and therefore a reduction in mortality due to malaria infections. Moreover, non disposal of liquid waste could generally create poodles and standing water conditions which would favour the breeding of mosquito larvae in open drains systems. As a consequence, residential areas with low sewer connection rate may be noted for high mosquito breeding (high malaria transmission) while areas with high sewer connection rate (*i.e.* the proportion of households connected to the sewer system is high) low mosquito breeding (low malaria transmission). This probably accounts for the reduction in urban malaria mortality with increase in the proportion of households connected to the sewer system. This observation was consistent with findings reported in many previous studies in which the association between mosquito vector breeding and waste material deposition has been documented [192, 454-457]. Like both solid waste collection rate and the proportion of households connected to the sewer system, the higher proportion of households connected to the WC, the smaller the chance of liquid wastes left to collect as standing pools of water. This perhaps reduced the size of habitats for mosquito breeding and thus lowered urban malaria transmission. This could also be
responsible for the decrease in urban malaria mortality with an increase in the proportion of households connected to the WC. Although many studies have reported increased malaria transmission rate and intensity with deteriorating environmental quality [317, 458-464], no reports of such relationship between environmental conditions and malaria mortality have been documented. In a study to investigate risk factors for malaria, children living in houses with mud roofs had significantly higher risk of getting *P. falciparum* infection compared to those living in iron-sheet roofed houses (Odds Ratio 2.6; 95% CI: 1.4-4.7) [465]. The lack of evidence of association between the fraction of cluster level deaths due to diarrhoea and environmental conditions was perhaps a consequence of blurring of study outcomes by intermediate socioeconomic conditions more than a lack of association. There assertion was corroborated by evidence in literature which reported a strong association of diarrhoea morbidity with poor hygiene and sanitation [63, 293, 408-410]. In a study to explore how a city-wide sanitation intervention altered the magnitude of relative and attributable risks of diarrhoea determinants and the pathways by which those factors affected diarrhoea risk, the authors reported that the intervention reduced diarrhoea and also changed attributable and relative risks of diarrhoea determinants by altering the pathways of mediation [410]. In addition, the study reported that socioeconomic status was a major distal diarrhoea determinant (attributable risk: 24%) with 90% of risk mediated by other factors, mostly by lack of sanitation and poor infrastructure (53%) and only accounted for 13% of risk with only 42% mediated by other factors (18% by lack of sanitation and poor infrastructure) [410].
5.6 Mapping, vulnerability and excess urban mortality assessment

The main goal of this component of the analysis was not only to map and describe the patterns of the observed urban malaria and diarrhoea mortalities in a rapidly urbanizing area in a low income country, but also to identify the foci of excess malaria and diarrhoea mortalities in an urban area with high heterogeneity of neighbourhood environmental conditions in order to allow for comparison and efficient resource allocation.

Infectious diseases, especially malaria and diarrhoea have been widely reported to exhibit spatial clustering, generally viewed as a characteristic in regions of high infectious transmission [88, 128, 183, 311, 466-468]. In the high transmission areas, infectious diseases could either cluster with respect to socioeconomic factors or cluster based on differing levels in environmental conditions [88, 300, 327, 466, 469, 470]. Several published studies have reported excess urban diarrhoea cases in urban residential areas without access to potable water, but with poor hygiene conditions and low income neighbourhoods [128, 183, 300, 324, 466, 467, 471, 472]. Malaria has been reported to exhibit elevated incidences in areas with more breeding opportunities for the mosquito vector [183, 324, 473, 474]. In the current study, a strong evidence of clustering (i.e. hotspot of high malaria mortality) was observed in the neighbourhood of an open lagoon and two cold spots far removed from the lagoonal influences, thus suggesting that large water bodies in urban spaces could exert some amount of influence on malaria mortality, an observation which was quite consistent with findings from other studies [324, 466, 474, 475]. This observation has been viewed to have important implication for urban health, which meant that urban health policy could be strengthened if strategic objectives and program scope were broadened to encompass the larger urban structure, including
surface water bodies and other social matrices and not just the conventional approach of focusing only on hygiene, water supply and sanitation. On the contrary, the observed pattern of diarrhoea mortality was rather random, only showing strong evidence of clustering for low-low values \((i.e.\) only cold spots) in the neighbourhood of a closed freshwater lagoon in the eastern part of Accra, probably suggesting that closed freshwater lagoons could perhaps more likely protect against diarrhoea mortality than their open saline counterparts. However, currently no evidence exists in the literature that demonstrates that closed freshwater in urban environments protect against urban malaria or diarrhoea mortality, although the striking clustering of malaria and diarrhoea mortality \((i.e.\) malaria mortality hotspots near an open saline lagoon and diarrhoea mortality cold spots near a closed freshwater lagoon) could not be ignored either. Thus, the observation provides leads for further investigation into influences of urban lagoons and wetlands on infectious disease mortality \([471, 476-480]\). Such further investigations would provide additional evidence for informed decision-making on urban health policy change in rapidly urbanizing areas in countries with limited health sector budgets. Despite the obvious limitation and the failure of the study to provide conclusive explanation for the observed patterns of excess urban malaria mortality, a careful scrutiny of the study results together with the knowledge of the biology and behaviour of mosquito vector as well as lifestyle and human behaviour should aid in defining hypotheses of future research on this subject. We therefore attempt to explain the distribution of the observed urban mortalities in Accra in the context of the vector and pathogen biology which underlie their abundance and distribution in the different set of urban environmental media and socioeconomic conditions. The discussion of our findings will also draw on previous
studies relating to social and anthropogenic factors which drive changes in urban ecology and how the changes ultimately influence urban mortalities. For instance, the ideal temperature range for malaria-carrying mosquitoes is 15 - 30°C [427, 481]. Temperature exerts varied effects on survival and reproduction rate of mosquitoes. If initial temperature is high, then an increase in average temperature, associated with global warming, can decrease the survival and reproduction rate of mosquitoes [481]. Mosquitoes are also highly sensitive to changes in precipitation and humidity. Increased precipitation can increase mosquito population indirectly by expanding larval habitat and food supply. Mosquitoes are, however, highly dependent on humidity, surviving only within a limited humidity range of 55-80% [481-484].

Infectious disease; particularly malaria and diarrhoea, often accompanies extreme weather events, such as floods, earthquakes and drought [483, 485]. Outbreak of certain infectious diseases also follows certain environmental change events such as rapid urbanization and concomitant deterioration of environmental quality [484, 486, 487]. These event-dependent local epidemics occur due to loss of infrastructure, such as hospitals and sanitation services, but also because of changes in local ecology and environment. For example, malaria outbreaks have been strongly associated with the El Nino cycles of a number of countries (e.g. India and Venezuela) [488-490]. In the face of global warming, there has been a marked trend towards more variable and anomalous weather leaving no doubt about an imminent transition in infectious disease transmission [491-493]. The trend towards more variability and fluctuation in local climate conditions in endemic areas is perhaps more important, in terms of its impact on human health, than
that of a gradual and long-term trend towards higher average temperature which may tend to produce more defined and predictable disease transmission patterns [494-496].

Most mosquitoes mate shortly after emergence from pupa and after mating, sperm passed by male into the female enters the spermatheca [78, 427, 481, 497]. The sperm in the spermatheca usually serves to fertilize all eggs laid during the life time of the female; thus only one mating and insemination per female is required [481, 498]. With a few exceptions, a female mosquito must bite a host and take a blood meal to obtain the necessary nutrients for the development of the eggs in the ovaries. After a blood-meal the abdomen of the mosquito is dilated and bright red in colour, but some hours later the abdomen becomes darker red [481, 482, 499]. A gravid female mosquito searches for suitable larval habitats in which to lay eggs after full development of eggs upon completion of blood digestion [481]. Depending on the species, adult female mosquitoes lay 50-200 eggs per oviposition [481, 482, 500]. The eggs are brown or blackish, quite small (~0.5 x 0.2 mm), and most Culicinae, are elongated or approximately ovoid in shape [481, 485]. Eggs are laid singly and directly on water in *Anopheles* and *Culex* species. A unique feature of the eggs is the presence of floats on either side [485, 501]. The eggs are not resistant to drying and cannot survive desiccation, hatch within 2–3 days, although hatching may take up to 2–3 weeks in colder climates [482, 487].

In respect of larval recognition, mosquito larvae can be distinguished from those of the other aquatic insects by being legless and having a bulbous thorax that is wider than both the head and the abdomen [502, 503]. They have a well-developed head with mouth brushes used for feeding, a large thorax and a nine segmented abdomen. All mosquito larvae require water in which to develop; no mosquito has larvae that can withstand
desiccation, although they may be able to survive over limited periods in partially aquatic environments such as in wet mud [427, 502]. Mosquito larvae have well developed head, bearing a pair of antennae and a pair of compound eyes with prominent mouthbrushes which are present in most species and serve to sweep water containing minute food particles into the mouth [503]. The larvae have structures that allow survival in the water and permit efficient respiration and in most species, they breathe through spiracles located on the 8th abdominal segment and therefore must come to the surface frequently [481, 482]. However, Anopheles larvae lack a respiratory siphon and for this reason they position themselves so that their body is parallel to the surface of the water [481]. The larvae spend most of their time feeding on algae, bacteria, yeasts and numerous other microorganisms in the surface microlayer. The larvae of other mosquito species feed on decaying plant and animal material found in the water. While many browse over the bottom of habitats, some, including *Anopheles* species are surface feeders sieving out food particles from surface microlayer [500]. They dive below the surface only when disturbed and swim either by jerky movements of the entire body or through propulsion with the mouth brushes [481, 482].

The larvae develop through multiple stages, or four instars, after which they metamorphose into pupae through molting, shedding their exoskeleton, or skin, to allow for further growth at the end of each instar [481, 498]. The process from egg laying to emergence of the adult is temperature driven with average minimum time of 7 days [78, 504].

The larvae occur in a wide range of habitats but most species prefer clean, unpolluted water [500]. The habitats vary from large and usually permanent collections of water,
such as freshwater swamps, marshes, rice-fields and borrow pits, to smaller collections of
temporary water such as pools, puddles, water-filled car tracks, ditches, drains and
gulleys [481, 500]. A variety of “natural container-habitats” also provide breeding places,
such as water-filled tree-holes, rock-pools, water-filled bamboo stumps, bromeliads,
pitcher plants, leaf axils in banana, pineapple and other plants, water-filled coconut husks
and snail shells [481, 505]. Larvae also occur in wells and man-made “container-
habitats”, such as clay pots, water storage jars, tin cans, discarded kitchen utensils and
motor vehicle tyres [481, 485, 505]. Whereas some species prefer shaded larval habitats
and others like sunlit habitats, many species even cannot survive in water polluted with
organic debris with others breeding prolifically in water contaminated with excreta or
rotten vegetation [481, 500, 506]. A few mosquitoes breed almost exclusively in brackish
or salt waters, such as saltwater marshes and mangrove swamps, and are consequently
restricted to mostly coastal areas. Some species are less specific in their habitat
requirements and can tolerate a wide range of different types of breeding place [481,
504]. For this reason, the larvae of *Anopheles* mosquitoes have been found in fresh- or
salt-water marshes, mangrove swamps, rice fields, grassy ditches, the edges of streams
and rivers, and small, temporary rain pools in both rural and urbanized environments
[481]. Mosquito larvae habitat requirement is so wide to the extent that some breed in
open, sun-lit pools, while others are found only in shaded breeding sites in forests.
Almost any collection or temporary water can constitute a mosquito larval habitat, but
larvae are normally absent from large expanses of uninterrupted water such as lakes,
especially if they contain large numbers of fish and other predators [481, 497]. Larvae are
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also absent from large rivers and fast-flowing waters, except those occurring in marshy areas and isolated pools and puddles formed at the edges of flowing water [503].

Regarding pupal morphology, mosquito pupae are exclusively aquatic and comma-shaped when viewed from the side. The head and thorax are fused into a cephalothorax which carries a pair of respiratory trumpets dorsally and an abdomen which curves around underneath [481]. As with the larvae, pupae must come to the surface frequently to breathe, which they do through a pair of respiratory trumpets on the cephalothorax [502]. The pupae do not feed, spend most of pupating time taking in air and after a few days as a pupa, the dorsal surface of the cephalothorax splits and the adult mosquito or imago emerges [503].

The average time for development from egg to adult varies considerably among species and strongly influenced by ambient temperature. Mosquitoes can develop from egg to adult in as little as 5 days but usually take 10–14 days in the topics [481, 502, 503]. Like all mosquitoes, adult Anopheles mosquitoes have slender bodies with 3 sections: head, thorax and abdomen. The head is specialized for acquiring sensory information and for feeding. The head contains the eyes and a pair of long, many-segmented antennae. The antennae are important for detecting host odours as well as odours of breeding sites where females lay eggs [481, 497]. The head also has an elongated, forward-projecting proboscis used for feeding, and two sensory palps [481]. The thorax of an adult mosquito is specialized for locomotion. There are three pairs of legs and a pair of wings which are attached to the thorax with only one pair employed during flight [481, 503, 507]. The abdomen is segmented and specialized for food digestion and egg development. The segmented abdomen expands considerably when a female takes a blood meal. The blood
takes a long time to digest and serves as a source of protein for the production of eggs, which gradually fill the abdomen at complete digestion [481, 485]. Anopheles mosquitoes can be distinguished from other mosquitoes by their possession of palps, which are as long as the proboscis, and by the presence of discrete blocks of black and white scales on the wings [427, 481, 498]. Another typical feature for adult Anopheles identification is their unique resting position: *i.e.* males and females rest with their abdomens sticking up in the air rather than parallel to the surface on which they are resting [481]. Adult mosquitoes will typically mate within a few days after emerging from the pupal stage. In most cases, the males form large swarms, usually around dusk, and the females fly into the swarms to mate after which the males live for about a week, feeding on nectar and other sources of sugar before dying [481, 498, 508]. Usually, females also feed on sugar sources for energy but require a blood meal for the development of eggs. After obtaining a full blood meal, the female will rest for a few days while the blood is digested and eggs are developed. This process is temperature dependent and usually takes 2–3 days in tropical conditions [78, 481, 483]. Once the eggs are fully developed, the female lays them and resumes host seeking for another session of blood meal taking. The cycle repeats itself until the female dies. While the lifespan of adult mosquito depends on temperature, humidity, and also their ability to successfully obtain a blood meal while avoiding host defenses, most do not live longer than 1–2 weeks in nature, albeit females can live longer than a month in captivity [481]. From the standpoint of both the biology and ecology of the mosquito vector, changing environmental and climatic conditions may significantly affect either larval or pupal survival (*i.e.* vector breeding regime) and thus affect malaria transmission in such
environments. Once the disease transmission rhythm changes, other social and behavioural factors including availability of health services, access to healthcare, financial barriers and health seeking behaviour of individual all interact to change the course of progression from disease to mortality as supported by the strong association observed between urban malaria mortality and the environmental and socioeconomic conditions in Accra, Ghana. Given that our study did not include variables on the biology of the mosquito vector, an obvious limitation is presented regarding the link between mosquito vector breeding, malaria transmission and malaria mortality.

Consequently, it is imperative to discuss the results in the context of similar studies that might provide insights to guide the hypothesis that would develop from this current analysis. A Brazilian study reported that multiple players such as social policy, migration, urbanization, city water supply, garbage disposal and housing conditions, as well as community level understanding of the disease and related practices affect vector ecology \[509\]. The descriptive study used a multi-disciplinary approach to compare the effects on dengue fever transmission of irregularity in water supply in households from both under-privileged and privileged areas. It was observed that households located in more privileged blocks where irregularity in water supply was not an issue, dengue transmission was lower than in households located in underprivileged blocks where water supply was highly irregular. The study pointed out that due to irregularity in water supply in the under-privileged blocks households stored water in barrels, open tanks and pots and thus provided improved breeding opportunities and the chances of *Aedes aegypti* survival for increased dengue transmission.
To examine the relationship between terrestrial change and mosquito composition, a Peruvian study used remote sensing techniques to define spatially explicit land use categories along a gradient with low (rural), medium (peri-urban), and high (urban) anthropogenic influence in the Peruvian Amazon and reported significant differences in mosquito diversity among the land use categories [510]. The results from the study provided baseline data linking mosquito distribution to land use in the Peruvian Amazon, thus creating standardized methods to measure the impact of human influence on the environment and to predicting disease risk in rapidly changing environments. Although our study was only able to establish associations between spatial change in urban environmental conditions and mortalities rather causal relationships, both studies demonstrate that anthropogenic environmental disturbances are perhaps important factors driving mosquito community composition and malaria transmission. Malaria is especially susceptible to changes in the environment as both the pathogen (Plasmodium) and its vector (mosquitoes) lack the mechanisms necessary to regulate internal temperature and fluid levels [427, 503, 511]. Thus, there is a limited range of ecological and climatic conditions within which the parasite and vector can survive, reproduce and infect hosts and vector-borne diseases, such as malaria, have distinctive characteristics that determine its pathogenicity [502, 512]. More generally, the survival and reproduction rate of the vector, the level of vector activity (\textit{i.e.} the biting or feeding rate), the development and reproduction rate of the parasite within the vector or host all depend on availability of suitable habitats and habitat conditions such as temperature, precipitation and humidity [78, 485, 503, 504, 513].
In a study to assess the potential effects of different construction features of homes on the indoor abundance of Culicine mosquitoes in Trinidad & Tobago (TT) and the Dominican Republic (DR), a survey using xenomonitoring was conducted in different homes in both countries alongside concurrent indoor resting mosquito collection to determine which features of the homes were correlated with a greater mosquito abundance [512]. The study was conducted between June 2002 and April 2003 and data were collected from 104 and 121 homes in TT and DR respectively. While in TT, 61 (58.65%) percent of the homes were located in urban areas with 43 (41.35%) in rural areas, in the DR 81 (66.94%) of the homes were located in the urban areas with 40 (33.06%) in rural areas. Out of a total of 1,630 mosquitoes collected in TT, 77% were Culex quinquefasciatus, while 46% of the 459 mosquitoes collected from homes in the DR were Cx. quinquefasciatus. However, the mean number of Cx. quinquefasciatus mosquitoes in both countries was greater in cement homes than in either wood or other poorer quality homes (TT cement 17.43, others 14.43; DR cement 4.24, others 3.41). In the case of TT homes that had painted interiors were significantly more likely to have a high abundance of mosquitoes resting indoors compared to homes without painted interiors (OR 2.90, CI 1.09-8.72). Interestingly, having a painted interior or exterior was found to be a predictor of a high abundance of indoor resting mosquitoes in the DR (interior OR 3.13, CI 1.41-6.92; exterior OR 1.97, CI .91-4.26). Reduced adult abundance in TT was reported to correlate with homes built on stilts, with more than four people sleeping per home with a painted interior. In the DR however, reductions reportedly correlated with homes where residents slept under a bed net and rurality. Changes in construction patterns in the
Caribbean region could help prevent human-mosquito contact potentially reducing the transmission of certain vector-borne diseases in the population.

A study that assessed the effects of combined sewage overflows (CSOs) from urban streams in Atlanta, GA, on oviposition site selection by Culex quinquefasciatus under seminatural field conditions reported that CSO water quality, especially when enriched, was a more attractive oviposition substrate than nonenriched water [500]. The conclusion of the study which was consistent with the strong association observed between malaria mortality and sewer as well as non-sewer disposal practices in Accra was that environmentally sound management of municipal waste water systems had the potential to reduce the risk of Culex-borne diseases in urban areas. Many other studies have reported that ecosystem changes caused by anthropogenic activities have modified the environment in ways that have promoted the emergence of vector-borne diseases [500, 502, 510, 512]. A health facility-based survey and health care system evaluation carried out in a peripheral municipality of Abidjan (Yopougon) during the rainy season of 2002 reported that malaria infection rates in fever cases for different age groups were 22.1% (under one year-olds), 42.8% (one to five years-olds), 42.0% (> five to 15 years-olds) and 26.8% (over 15 years-olds), while those in the control group were 13.0%, 26.7%, 21.8% and 14.6%, respectively [513]. While the fractions of malaria-attributable fever were 0.12, 0.22, 0.27 and 0.13 in the respective age groups, parasitaemia was homogenously detected in different areas of the city, thus concluding that rapid urbanization had changed malaria epidemiology in more urbanized areas compared to peripheral Yopougon where endemicity was found to be moderate [513]. The conclusion of this study is consistent with the strong association observed between urban malaria mortality
and the change in socioeconomic and environmental conditions due urbanization in Accra.

By and large, whereas several reports exist in literature showing the relationship between environmental change and vector ecology, not too many studies have described the relationship between environmental modification and malaria mortality. A study that monitored oviposition activity *Aedes aegypti* as a function of demographic and environmental variables reported that the proportion of weeks of vector infestation and the total number of eggs showed strong spatial continuity and were higher in areas that had higher densities of houses and relatively closer to industrial sites compared to areas with higher human populations or higher densities of flats and far from industrial sites [485]. While many attempts have been made to quantify the burden of malaria in Africa, none has addressed how urbanization will affect disease transmission and outcome, and therefore mortality and morbidity estimates. Nevertheless, the results of a series of entomological, parasitological and behavioural meta-analyses of the few studies that have investigated the effect of urbanization on malaria in Africa showed there were 1,068,505 malaria deaths in Africa in 2000 revealing just a modest 6.7% reduction over previous iterations [484].

A bionomics study of malaria vectors carried out in riverine and non-riverine areas in the Indian city of Delhi on account of tremendous ecological changes in its topography showed that day-time resting preferences of the vector species in human dwellings and cattlesheds did not differ significantly [497]. However, the preferred larval habitats of *An. culicifacies* in riverine area had shifted to large lakes, channels and ponds and that whilst *An. culicifacies* played a role only in the northern part of the riverine area where water
pollution was at minimal level, *An. stephensi* was the main vector of malaria transmission in both areas [497]. This confirms that changes in habitat preference of the different vector species have profound implications for malaria transmission and may be the underlying reason for the observed anomalous distribution of excess malaria mortalities in the environs of surface water bodies in Accra.

Gauging from the findings of both the present and previous studies, especially relating to the ecology and biology of the mosquito vector, malaria control efforts might become more effective only if such efforts were all encompassing by way of bundling the life cycle of the vector, the parasite and the human host interactions under single intervention. The *Anopheles* mosquito is the only species known to transmit malaria. For this reason, understanding the biology and behavior of Anopheles mosquitoes will not only help in the discovery of evolving malaria transmission mechanisms, but should ultimately aid in designing appropriate control strategies. The basis for which ecological and environmental change processes affect malaria transmission relates to ability of the processes to alter habitat and survival factors such as breeding, feeding and resting conditions of the mosquito vector. In the particular case of urbanization, the resulting environmental change conditions tend to promote the survival conditions of the mosquito vector, thus favouring a sustained transmission of malaria in rapidly urbanizing endemic areas. Factors that affect the ability of the mosquito to transmit malaria include its innate susceptibility to *Plasmodium* parasite, its host choice and its longevity. Therefore in designing control programs, the susceptibility and resistance of malaria vectors to insecticide as well as the preferred feeding and resting location of adult mosquitoes should be key factors to take into consideration. Another key factor to consider in
designing malaria control programs is the resistance of the *Plasmodium* parasite itself to drugs. Parasite resistance to drugs has been widely reported in many malaria transmission areas and represents a major obstacle to control efforts. However, innovative strategies aimed to overcome the problem of drug resistance are being developed and deployed along discovery of new powerful drugs and natural plant extracts that can withstand parasite resistance and/or inhibit parasite growth and development. For instance, a recently published study reported that the hemolytic C-type lectin CEL-III from *Cucumaria echinata*, a sea cucumber found in the Bay of Bengal impaired the development of the malaria parasite when produced by transgenic *A. stephensi* [482, 514]. This could potentially be used one day to control malaria by spreading genetically modified mosquitoes refractory to the parasites, although there are reportedly numerous scientific and ethical issues to be overcome before such a control strategy could be implemented.

The survey of similar studies on the biology of the mosquito vector and a scrutiny of the excess malaria mortality maps provided clues that, the urban socioeconomic and environmental conditions were key drivers of urban malaria mortalities and that their deterioration could reinforce elevation in the risk of excess mortalities in urban spaces. The strong evidence of association observed between excess mortality and the urban environmental and socioeconomic conditions were consistent with findings from several published studies [183, 466, 467, 473, 475] and strengthened the case that disease control programs in urban centres would benefit tremendously from urban environmental management in fast urbanizing areas in low income countries.
Finally, the strong evidences of local level spatial autocorrelation and the association between excess mortality maps on the one hand and the socioeconomic and environmental quality conditions on the other hand suggest that both the socioeconomic and the environmental conditions could be important risk factors for urban malaria and diarrhoea mortalities in rapidly urbanizing areas.

5.7 Limitation of study

In general, the proportions of economically active and economically inactive populations were not shown to be valid measures of the area-based socioeconomic conditions. For instance, the positive (i.e. a unit increase in population economic inactivity resulted in an increase in per capita solid waste generation rate) association between economically inactive population and per capita solid waste generation (regression coefficient = 0.276) was obtuse. High values of the proportion of economically inactive population represented low socioeconomic status and high values of the proportion economically active cluster populations represented high socioeconomic status. However, with the understanding that the per capita waste generation rates in high socioeconomic areas have been theoretically reported to be higher than those from low socioeconomic areas [38, 355], the observed association between neighbourhood urban environmental quality conditions and the proportion of economically inactive and/or active populations somewhat did not make sense. On account of this, both the proportion of economically active and/or inactive cluster population were regarded as probably unreliable measures/proxies of area-based SES. For instance, the fact that a resident was economically active did not mean that the individual was employable and could contribute to the community’s pool of wealth. In a
similar argument, the fact that an individual was economically inactive did not mean that such individual could not generate income and/or contribute to the community’s wealth. Therefore, economically active or inactive factor did not predict community income or wealth and probably invalid proxy measure of SES. Data attributes that might affect their validity and reliability include; data completeness and coverage, misclassification and reporting biases. The Ghana Census covers the entire population and approximately 100 percent complete. In addition, Ghana’s population is fairly well defined and the variables enumerated were also fairly discretely defined without overlaps. Therefore both data completeness and misclassification did not present any perceived data limitation and therefore presented no perceived validity threats to the Ghana census data. However, it was possible that respondents to census questionnaire did not provide correct answers to census questions or might not have responded accurately to questions on the variables collected during the census. This meant that the Ghana census data might be prone to reporting bias which might have affected the results and conclusions of this study.

Finally, the PMRfdS were computed using all-cause mortality as the denominator and so if the risk of mortality for one of the components of all-cause mortality varied by cluster, then this could affect the summary/outcome measure PMRfd and therefore bias the analysis. A way to deal with this problem was to exclude such components, but unfortunately, we did not have the liberty of telling \textit{apriori} if any of the components of all-cause mortality exhibited differential mortality risk by cluster and therefore this bias could not be controlled for. Another way to deal with this bias would have been to use a specific cause of death known to have a uniform risk level across all clusters as the denominator for both the observed proportion and the expected proportion. But then
again, this also required knowledge of risk distribution by cause of death in the study area and which unfortunately was not available for the study area.
6.1 Conclusions

Environmental change is a slow process and its ultimate effects on human health can as well either be gradual or immediate, depending on the nature of the processes and the chain of links involved. Examples of environmental change events with potential adverse impacts on human health include urbanization, climate change, stratospheric ozone depletion, loss of biodiversity, changes in hydrological systems and the supplies of freshwater, land degradation and stresses on food-producing systems.

Despite emerging realities that environmental change is on-going with concomitant adverse impacts on human health, research tools required to demonstrate the association between the change events and human health are still being developed. Perhaps, this has to do with the difficulty in conceptualizing environmental change to reflect all its diverse characteristics. Unlike in the laboratory setting where most reaction conditions can be controlled, in environmental change experiments, it is hard to define and control the range of conditions forming the universe of the physical and social environment and how the conditions interact to determine the overall impacts on human health at aggregate level. Appreciation of the type, magnitude and scale of human health impacts of environmental change requires a new perspective which focuses on ecosystems and on the recognition that the foundations of long-term good health in populations rely in great part on the continued stability and functioning of the biosphere's life-supporting systems. It also brings an appreciation of the complexity of the systems upon which human health depends and how changes in the systems can produce adverse health effects on humans.
While it is well understood that large scale ecological changes affect human health, most of the phenomenal ecological changes have been largely human-induced. Human activities increasingly affect the structure and function of ecosystems. In turn, these changes also influence the entire chain of factors involved in the infectious disease transmission cycle: pathogens, vectors, reservoir species and human populations. Strictly unintended human activities can thus have serious consequences on the transmission of both communicable and non-communicable diseases. The scientific community recognizes the growing need to better understand the multi-faceted and complex linkages between environmental change (including urbanization, climate change, land and sea use change, biodiversity loss and change, socio-economic change) and human health. As the analysis evaluated the relationship between urban mortalities and several aspects of the urban environment in a spatially explicit manner, we logically present multiple conclusions to reflect the different foci of the analysis, namely demographic and health analysis, socioeconomic and environment analysis, socioeconomic and health analysis, environment and health aspect and finally disease clustering and excess mortality analysis.

In the demographic and health analysis, a key observation was that while maternal factors appeared to protect 1 year olds against malaria mortality, such a protection was absent in diarrhoea mortality. This led to a conclusion that intervention programs designed to control infant malaria and diarrhoea mortality would certainly have different strategic foci given the different levels of acquired or innate immune protection against the different disease outcomes.
In the analysis of the relationship between socioeconomic conditions and urban environmental conditions however, we observed several levels of association and remarkable findings which provided new insights for deeper understanding of how socioeconomic inequalities underpin disparate health outcomes in rapidly urbanizing areas in a low income economy.

While some of the area-based socioeconomic measures alone were not valid proxies of SES, others were valid at aggregate levels. On the whole, aggregating the area-based socioeconomic measures into a uni-dimensional attribute and generating wealth quintiles from the uni-dimensional attribute was observed to more robustly predict SES and therefore a more valid measure at community level. Strong evidence of differences in neighbourhood urban environmental quality existed across the wealth quintiles. This observation suggested that socioeconomic conditions were important drivers of change in neighbourhood urban environmental quality conditions thus providing clues that urban environmental interventions aimed at infectious disease prevention would benefit considerably from simultaneous implementation with social interventions if they were to be effective. We then concluded that widening socioeconomic inequalities (e.g., urban unemployment, urban employment, etc.) at household level could worsen the existing urban environmental health inequalities at community level. Therefore, for urban health policy change on infectious disease control, it would make sense if urban environmental interventions aimed at infectious disease prevention and control, were implemented simultaneously with complementary social interventions in order to be effective.

In addition, the analysis of the relationship between area-based measures of SES and urban malaria and diarrhoea mortalities revealed a number interesting and varying levels
of associations across different social contexts. Synthesis of the findings provided evidence for a conclusion that the health policy reforms implemented by the Ghana’s Ministry of Health to reduce urban health inequalities were perhaps more responsive to or effective in reducing urban diarrhoea than urban malaria mortality. A penultimate point is that the proportion of residents with vocational, technical and commercial, the proportion electricity, gas and water supply sector workforce, proportion of education sector workforce, proportion of residents with tertiary education are perhaps potentially important factors to consider among a range of risk factors for urban malaria mortality in future case-control studies to explore city-wide distribution of mortality risks in rapidly urbanizing low income settings. Moreover, the observed non-uniformities in the inter-quintile relative mortality differentials were perhaps more likely a consequence of lifestyle factors and inter-quintile differences in health seeking behaviour than the influence of the health policy mix.

The environment and urban mortalities component of the study showed that urban malaria mortality was more sensitive to changes in environmental conditions than diarrhoea mortality. Therefore, the conclusion drawn from the observation was that environmental management initiatives intended for infectious disease control might substantially reduce and/or lower the neighbourhood urban environmental-quality-attributable fraction of deaths due to urban malaria more than that due to urban diarrhoea in rapidly urbanising areas in a low income setting.

Finally, the geo-statistical analysis showed that the excess malaria mortalities were strongly associated with the urban socioeconomic and environmental conditions.
However, there was none at all or only weak association between urban diarrhoea mortality and the urban socioeconomic and environmental conditions.

We concluded therefore that, both the socioeconomic and the environmental conditions could be important risk factors for urban diarrhoea mortalities, and especially for urban malaria mortality. Our opinion was that salinity could also be an important factor in how surface water bodies influenced infectious disease mortality. To be able to make firm conclusion on this assertion we recommend further case-control studies to establish the range of urban mortality risk factors including those factors relating to surface water bodies within urban spaces in low income settings. This would help to direct resources appropriately at high risk areas and the most vulnerable groups as well as to provide more convincing evidence for urban health policy reforms in rapidly urbanizing areas in low income settings. Based on these findings, we support the use of small scale mapping to detect zones of vulnerability for an improved targeting of resources aimed at urban malaria and diarrhoea control in rapidly urbanizing areas in low income settings.

6.2 Eradication of malaria and diarrhoea – challenges and future direction

Undoubtedly, the cost of preventing malaria is much less than treating the disease in the long run. Whereas mosquito control is an important component of malaria control strategy, eliminating malaria in an area does not require the elimination of all *Anopheles* mosquitoes. Moreover, eradication of mosquito is not an easy task, which in itself would lead to a reduction in ecological diversity. For effective prevention of malaria, some parameters should be met such as conducive conditions in the community or a given country, improved data on the disease, targeted technical approach to the problem, behavioural change, very active and committed leadership, government’s total support,
monetary free hand, community involvement, skilled technicians from different fields as well as a well thought-out implementation strategy which incorporates the interests and views of community members. A wide range of strategies exist for achieving malaria eradication that start from simple steps to complicated strategies which may not be possible to enforce with the current tools and knowledge. Additionally, an important step towards malaria eradication will be to draw lessons from regions that have successfully eliminated malaria, regarding the strategies adopted, program delivery and implementation schedules. A combination of socio-economic improvements (e.g. houses with screened windows, air conditioning and smoothened interior walls) with vector reduction efforts and effective treatment may lead to the eradication of malaria without the complete elimination of the vector. Some important measures in mosquito control that might lead to complete eradication of malaria without eliminating the Anopheles mosquito include efforts to discourage egg laying, prevent development of eggs into larvae and adults, kill the adult mosquitoes, do not allow adult mosquitoes into places of human dwelling, prevent mosquitoes from biting human beings and deny blood meal. On the basis of the findings from this study, we advocate for the intensification of research on more targeted interventions to break the vector breeding cycle and human behavioural change, although many studies have suggested that the sterile mosquitoes might be the answer to malaria elimination. Another option might be to manipulate or modify the genetic constitution of the female Anopheles mosquito so that its internal physiological environment becomes unsuitable for the growth and development of the malaria parasite. Insecticide-based control measures (e.g. indoor spraying with insecticides, use of repellents and insecticide treated bednets) are the principal vector-based global control
strategies deployed to kill mosquitoes that bite indoors. However, after prolonged exposure to insecticides over several generations, mosquitoes, like other insects, have begun to develop counter-strategies against insecticide use. Mosquitoes are increasingly developing genetic modifications allowing them to develop resistance, a capacity to survive contact with an insecticide. Since mosquitoes have relatively short life-cycle and can have many generations per year, it therefore follows that high levels of resistance may arise very quickly over a relatively short period. It is therefore not surprising that just within a few years after the insecticides were introduced as a global control strategy, resistance of mosquitoes to some insecticides has been reported. Now, there are over 125 mosquito species with reported resistance to one or more insecticides. The development of resistance to insecticides used for indoor residual spraying is seen as a major impediment to the Global Malaria Eradication Campaign. In Ghana, mosquitoes have developed resistance to DDT. Also, chloroquine has already been removed from the list of first line drugs for malaria treatment and combination therapy has been widely adopted. There are increasing global concerns about the challenges of vector resistance, with the World Health Organization (WHO) leading a campaign for judicious use of insecticides so as to limit the development and spread of resistance. This includes controlled use of insecticides in other sectors such agriculture and fishing. While the use of insecticides in agriculture has often been reported as contributing to resistance in mosquito populations, it is possible to detect development of resistance. Thus, control programs would be well advised to conduct surveillances for this potential problem. Such surveillances would inform actions that underlie the strategic direction of malaria control efforts in endemic areas which are susceptible to developing resistance.
Unlike malaria, diarrhoea control has been successful by a larger measure in Ghana. The two main advances in managing diarrhoeal disease – formulated oral re-hydration salts (ORS) containing lower concentrations of glucose and salt, and success in using zinc supplementation remain the most effective global strategies for the control of diarrhoea recommended by the WHO. ORS in particular has been fully deployed for the management of diarrhoeal diseases in Ghana over the last decade and has proven very effective. Other strategies used in addition to prevention and treatment of dehydration with appropriate fluids include adoption of exclusive breastfeeding, continued feeding and selective use of antibiotics. The observed weak association between diarrhoea mortality and the changing urban socioeconomic and environmental conditions was probably a consequence of the tremendous success in diarrhoea treatment and management in Ghana. On the basis of fact that several instances of strong associations were observed among the environmental, socioeconomic and health variables, our first line of recommendation was therefore that, this study be repeated using the census 2010 data to confirm these associations and to evaluate how the introduction of the Ghana National Health Insurance Scheme (GNHIS) had influenced malaria/diarrhoea morbidity and mortality over the period of implementation.

Ultimately, we would recommend that case-control studies be conducted on the variables that were observed to exhibit strong associations in order to establish causal chains of events linking, socioeconomic conditions, neighbourhood urban environmental quality conditions and infectious disease mortality.
Appendices

**Appendix 1: Exploration of SES using Principal component analysis**

<table>
<thead>
<tr>
<th>SES Variable</th>
<th>Mean</th>
<th>Std dev</th>
<th>Factor score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Residents’ economic activity status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Economically inactive</td>
<td>0.610</td>
<td>0.076</td>
<td>0.500</td>
</tr>
<tr>
<td>Employed</td>
<td>0.139</td>
<td>0.041</td>
<td>0.500</td>
</tr>
<tr>
<td><strong>Residents’ educational attainment</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>No education</td>
<td>0.163</td>
<td>0.062</td>
<td>0.095</td>
</tr>
<tr>
<td>Pre-school education</td>
<td>0.044</td>
<td>0.008</td>
<td>0.448</td>
</tr>
<tr>
<td>Primary education</td>
<td>0.165</td>
<td>0.027</td>
<td>0.520</td>
</tr>
<tr>
<td>Middle/JSS education</td>
<td>0.165</td>
<td>0.027</td>
<td>0.520</td>
</tr>
<tr>
<td>Secondary/SSS education</td>
<td>0.155</td>
<td>0.032</td>
<td>0.132</td>
</tr>
<tr>
<td>Vocational/technical/commercial education</td>
<td>0.076</td>
<td>0.018</td>
<td>0.160</td>
</tr>
<tr>
<td>Post secondary education</td>
<td>0.029</td>
<td>0.009</td>
<td>-0.053</td>
</tr>
<tr>
<td>Residents with tertiary education</td>
<td>0.076</td>
<td>0.096</td>
<td>-0.451</td>
</tr>
<tr>
<td><strong>Residents’ occupation</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Administrative and managerial occupations</td>
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<tr>
<td>Clerical and related occupations</td>
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<td>0.016</td>
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<tr>
<td>Sales occupations</td>
<td>0.135</td>
<td>0.029</td>
<td>0.104</td>
</tr>
<tr>
<td>Service occupations</td>
<td>0.233</td>
<td>0.075</td>
<td>-0.490</td>
</tr>
<tr>
<td>Agriculture/husbandry/forestry/fishing/hunting occupation</td>
<td>0.122</td>
<td>0.067</td>
<td>0.356</td>
</tr>
<tr>
<td>Production/transport and equipment operators and labourers</td>
<td>0.042</td>
<td>0.047</td>
<td>-0.143</td>
</tr>
<tr>
<td>Proportion of other labourers not elsewhere classified</td>
<td>0.070</td>
<td>0.019</td>
<td>-0.415</td>
</tr>
<tr>
<td>Professional technical and related workers</td>
<td>0.237</td>
<td>0.047</td>
<td>-0.207</td>
</tr>
<tr>
<td><strong>Residents’ place of work</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Residents working in agriculture hunting and forestry</td>
<td>0.042</td>
<td>0.014</td>
<td>-0.027</td>
</tr>
<tr>
<td>Residents working in fishing</td>
<td>0.029</td>
<td>0.041</td>
<td>-0.065</td>
</tr>
<tr>
<td>residents working in mining and quarrying</td>
<td>0.018</td>
<td>0.009</td>
<td>0.020</td>
</tr>
<tr>
<td>residents working in manufacturing</td>
<td>0.169</td>
<td>0.031</td>
<td>-0.415</td>
</tr>
<tr>
<td>residents working in electricity gas and water supply</td>
<td>0.008</td>
<td>0.004</td>
<td>0.036</td>
</tr>
<tr>
<td>residents working in construction</td>
<td>0.083</td>
<td>0.041</td>
<td>0.015</td>
</tr>
<tr>
<td>residents working in wholesale/retail trade/vehicle repairers</td>
<td>0.264</td>
<td>0.081</td>
<td>-0.483</td>
</tr>
<tr>
<td>residents working in hotels and restaurants</td>
<td>0.024</td>
<td>0.009</td>
<td>-0.071</td>
</tr>
<tr>
<td>residents working in transport storage and communications</td>
<td>0.093</td>
<td>0.026</td>
<td>-0.320</td>
</tr>
<tr>
<td>residents working in banking &amp; finance</td>
<td>0.019</td>
<td>0.009</td>
<td>0.164</td>
</tr>
<tr>
<td>residents working in real estate renting and business activities</td>
<td>0.041</td>
<td>0.016</td>
<td>0.217</td>
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<tr>
<td>residents working in public administration/defence/social security</td>
<td>0.074</td>
<td>0.087</td>
<td>0.357</td>
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<tr>
<td>residents working in education sector</td>
<td>0.036</td>
<td>0.036</td>
<td>0.231</td>
</tr>
<tr>
<td>residents working in health and social services</td>
<td>0.019</td>
<td>0.032</td>
<td>0.245</td>
</tr>
<tr>
<td><strong>Residents’ marital status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>residents working in other community social and personal services</td>
<td>0.048</td>
<td>0.009</td>
<td>-0.059</td>
</tr>
<tr>
<td>residents working in private households</td>
<td>0.026</td>
<td>0.027</td>
<td>0.401</td>
</tr>
<tr>
<td>proportion of new workers seeking employment</td>
<td>0.007</td>
<td>0.008</td>
<td>0.024</td>
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### Appendices

<table>
<thead>
<tr>
<th>Marital Status</th>
<th>Mean</th>
<th>Std dev</th>
<th>Factor score</th>
</tr>
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<tbody>
<tr>
<td>Married residents</td>
<td>0.394</td>
<td>0.054</td>
<td>0.446</td>
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<tr>
<td>Residents living together but not married</td>
<td>0.043</td>
<td>0.025</td>
<td>0.446</td>
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<tr>
<td>Residents separated</td>
<td>0.018</td>
<td>0.008</td>
<td>0.269</td>
</tr>
<tr>
<td>Residents divorced</td>
<td>0.027</td>
<td>0.017</td>
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<tr>
<td>Residents widowed</td>
<td>0.016</td>
<td>0.008</td>
<td>0.410</td>
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<tr>
<td>Singles</td>
<td>0.502</td>
<td>0.076</td>
<td>-0.424</td>
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#### Residents' ethnicity

<table>
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<th>Ethnic Group</th>
<th>Mean</th>
<th>Std dev</th>
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<td>Akan group</td>
<td>0.439</td>
<td>0.106</td>
<td>0.109</td>
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<tr>
<td>Ga Dangme group</td>
<td>0.267</td>
<td>0.164</td>
<td>-0.417</td>
</tr>
<tr>
<td>Ewe group</td>
<td>0.153</td>
<td>0.076</td>
<td>0.249</td>
</tr>
<tr>
<td>Guan group</td>
<td>0.031</td>
<td>0.013</td>
<td>0.396</td>
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<td>Gurma group</td>
<td>0.011</td>
<td>0.025</td>
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<td>Mole-Dagbani group</td>
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<td>0.408</td>
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<td>Grusi group</td>
<td>0.024</td>
<td>0.012</td>
<td>0.413</td>
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<tr>
<td>Mande group</td>
<td>0.008</td>
<td>0.009</td>
<td>0.369</td>
</tr>
<tr>
<td>All other ethnic groups</td>
<td>0.013</td>
<td>0.021</td>
<td>0.298</td>
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</table>

### Appendix 2: Results of multi-variable SES included in the final PCA model.

<table>
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<th>SES Variable</th>
<th>Mean</th>
<th>Std dev</th>
<th>Factor score</th>
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<tr>
<td>Economically active</td>
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<td>0.201</td>
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<tr>
<td>Employed</td>
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<td>Pre-school education</td>
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<tr>
<td>Primary education</td>
<td>0.165</td>
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<td>0.305</td>
</tr>
<tr>
<td>Middle/JSS education</td>
<td>0.165</td>
<td>0.027</td>
<td>0.305</td>
</tr>
<tr>
<td>Residents with tertiary education</td>
<td>0.076</td>
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<td>-0.319</td>
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<td>Administrative and managerial occupations</td>
<td>0.014</td>
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<td>Clerical and related occupations</td>
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<td>Service occupations</td>
<td>0.122</td>
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<td>Agriculture/husbandry/forestry/fishing/hunting occupation</td>
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<td>0.047</td>
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<td>Proportion of other labourers not elsewhere classified</td>
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<td>Residents working in manufacturing</td>
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<tr>
<td>Residents working in wholesale/retail trade/vehicle repairers</td>
<td>0.264</td>
<td>0.081</td>
<td>0.296</td>
</tr>
<tr>
<td>Residents working in transport storage and communications</td>
<td>0.093</td>
<td>0.026</td>
<td>0.266</td>
</tr>
<tr>
<td>Residents working in public administration/defence/social security</td>
<td>0.074</td>
<td>0.087</td>
<td>-0.228</td>
</tr>
<tr>
<td>Residents working in private households</td>
<td>0.026</td>
<td>0.027</td>
<td>-0.312</td>
</tr>
</tbody>
</table>
Appendix 3: PCA output showing components produced

<table>
<thead>
<tr>
<th>Component</th>
<th>Eigenvalue</th>
<th>Difference</th>
<th>Proportion</th>
<th>Cumulative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comp1</td>
<td>5.42693</td>
<td>2.63348</td>
<td>0.3392</td>
<td>0.3392</td>
</tr>
<tr>
<td>Comp2</td>
<td>2.79345</td>
<td>0.71620</td>
<td>0.1746</td>
<td>0.5138</td>
</tr>
<tr>
<td>Comp3</td>
<td>2.07725</td>
<td>0.35214</td>
<td>0.1298</td>
<td>0.6436</td>
</tr>
<tr>
<td>Comp4</td>
<td>1.72511</td>
<td>0.55671</td>
<td>0.1078</td>
<td>0.7514</td>
</tr>
<tr>
<td>Comp5</td>
<td>1.16841</td>
<td>0.40396</td>
<td>0.0730</td>
<td>0.8244</td>
</tr>
<tr>
<td>Comp6</td>
<td>0.76444</td>
<td>0.10618</td>
<td>0.0478</td>
<td>0.8722</td>
</tr>
<tr>
<td>Comp7</td>
<td>0.65827</td>
<td>0.10866</td>
<td>0.0411</td>
<td>0.9134</td>
</tr>
<tr>
<td>Comp8</td>
<td>0.54960</td>
<td>0.26207</td>
<td>0.0344</td>
<td>0.9477</td>
</tr>
<tr>
<td>Comp9</td>
<td>0.28753</td>
<td>0.08384</td>
<td>0.0180</td>
<td>0.9657</td>
</tr>
<tr>
<td>Comp10</td>
<td>0.20369</td>
<td>0.08221</td>
<td>0.0127</td>
<td>0.9784</td>
</tr>
<tr>
<td>Comp11</td>
<td>0.12149</td>
<td>0.02328</td>
<td>0.0076</td>
<td>0.9860</td>
</tr>
<tr>
<td>Comp12</td>
<td>0.09820</td>
<td>0.03019</td>
<td>0.0061</td>
<td>0.9921</td>
</tr>
<tr>
<td>Comp13</td>
<td>0.06801</td>
<td>0.03279</td>
<td>0.0043</td>
<td>0.9964</td>
</tr>
<tr>
<td>Comp14</td>
<td>0.03522</td>
<td>0.01284</td>
<td>0.0022</td>
<td>0.9986</td>
</tr>
<tr>
<td>Comp15</td>
<td>0.02238</td>
<td>0.02238</td>
<td>0.0014</td>
<td>1.0000</td>
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<tr>
<td>Comp16</td>
<td>1.110e-16</td>
<td>0.00000</td>
<td>0.0000</td>
<td>1.0000</td>
</tr>
</tbody>
</table>
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