ASIAN JOURNAL
ISSN: 0976-3376

Available Online at http://www.journalajst.com

ASIAN JOURNAL OF SCIENCE AND TECHNOLOGY

Asian Journal of Science and Technology
Vol. 4, Issue 03, pp.037-043, March, 2013

RESEARCH ARTICLE

BURULI ULCER MORBIDITY AND SOIL ARSENIC LINKAGES IN THE AMANSIE WEST DISTRICT OF GHANA: A GEOSTATISTICAL APPROACH

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Received 16th December, 2012; Received in Revised from; 09th January, 2013; Accepted 11th February, 2013; Published online 09th March, 2013

ABSTRACT

Buruli ulcer, a disease caused by infection with Mycobacterium ulcerans, is one of the most neglected but treatable tropical diseases. The causative organism is from the family of bacteria which causes tuberculosis and leprosy but knowledge gaps exist on the exact mode of transmission. The aim of this paper is to examine the link between the Buruli ulcer morbidity and Soil Arsenic concentration in the Amansie Wes t District of Ghana using kriging method. This paper provides the application of kriging to the spatial interpolation of local disease rates on the district boundary and soil map, resulting in continuous maps of disease rate estimate. It again provides the application of kriging to arsenic sample data in Amansie West District as a covariates variable. The spatial analysis was confined to settlements located within 60 kilometers from the Amansie West District to avoid underestimating the risk of the disease incidence. Semivariogram models revealed a range of autocorrelation of 1.9 km for the Buruli ulcer disease and 17.9 km for arsenic risk. There are large patches in both southern and northeastern part of the kriged map indicating that all the soil types are susceptible to BU disease. However, the entire area has high level of arsenic concentration than recommended level by World Health Organization. The geographically weighted correlation between arsenic and Buruli ulcer estimated was 0.9 and below. The approach presented in this paper enables researchers to incorporate the pattern of spatial dependence of incidence rates into the mapping of risk values and the quantification of the associated uncertainty.

Key words: Geostatistics, kriging, Risk semivariogram, Buruli ulcer, Weighted least-square regression, Correlation.

INTRODUCTION

Buruli ulcer (BU) which is caused by Mycobacterium ulcerans has become one of the most rapidly emerging tropical disease in West Africa in recent decades (Stienstra, 2001). BU has emerged as an increasingly vital cause of human morbidity around the world, partly due to environmental change Amofah *et al*. (1993). The disease originated from the district of Uganda, where the first large numbers of cases were reported in the late 1960s and early 1970s Asiedu *et al*. (1998). Buruli ulcer incidence is highest among developing West African nations (WHO 2001), with cases in some countries exceeding those of tuberculosis and leprosy (Amofah *et al*., 1993, 2002). Up to 16% of villages are affected in Cote d'Ivoire (Marston *et al*., 1995; WHO 2001), and Benin has recorded 4000 cases since 1989 (Lagarrigue *et al*., 2000). In West Africa, nearly 25% of people infected are left permanently disabled (Johnson *et al*., 2005). There is also evidence of vast under-reporting of the disease. Ghana is the second most endemic country for buruli ulcer after Cote d'Ivoire globally, (WHO, 2012). Buruli Ulcer was first brought to public attention in Ghana in 1993 when severe cases were reported from the Amansie West district of Ashanti Region in August (MOH, 2004). Specifically the most affected town is Tontokorom, although earlier cases have been reported from the Densu and Afram plains, (Baylay 1971) and (Van der werf *et al*., 1989. The overall national prevalence rate of active Buruli Ulcer is 20.7 per 100,000 of the population but as high as 150.8 per 100,000 (Ministry of Health, Ashanti Region, 2004). The worse affected regions are Ashanti, Central, Brong Aharfo, Greater Accra and Eastern. Although there is a lot of literature on the possible causes of Buruli ulcer (BU), no one is sure where the bacterium lives in the environment and how the mycobacterium enters the human body therefore, the arguments have been purely speculative. majority of the epidemiological data and some hypothesis have associated the outbreak and emergence of the disease with an aquatic environment (Marsollier *et al*., 2002; Portaels *et al*., 1999).

Most investigators have implicated insects, airborne, trauma and human to human as possible modes of transmission. Foci of the disease appear to develop after some form of environmental disturbance such as flooding or the formation of new dams or water storages, sand winning, where excavation have left behind large sheets of stagnant water Aiga *et al*. (2004) . Veitch *et al*. (1997) reporting a large outbreak of the disease on Philips Island, Australia associated the source of infection to an irrigation which lay in the midst of the cluster of cases. Number of cases reported from the community reduced after the irrigation site was modified and

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limited from the public. Scot *et al*. (2004) noted that cases of Buruli ulcer are associated with tropical wetlands of west and central Africa ,and cases have increased rapidly in these areas since the 1980's, particularly after irrigation and dam construction. Apart from associating aquatic environment with BU infection, it has also been observed that high levels of Arsenic (As) concentrations prevail in such environment. According to (Duker *et al*., 2004) arsenic may play a vital role in the spatial distribution of BU. Areas where BU is a serious health threat, concentration of As in surface and ground water has been higher than average. Coincidentally, in Ghana, the Amansie West District, which accounts for most of the BU cases happens to have the highest levels of As, possibly released into rivers and lakes and ground water by intensive gold mining activities (Aidoo *et al*., 2007). Human activities in the Amansie West District have elevated arsenic contamination in the environment (Bell, 1998). High levels of arsenic in drinking water have been detected in the water bodies with concentrations frequently exceeding the World Health Organization level of (MCL) of 10 µg/L (Smedley *et al*., 2002). In a separate study in an arsenic enriched environment, Sarkodie *et al*. (1997) alsos found that the peak period in which subsistence crops and fern contained the highest concentration of both species of arsenic (As^{3+}, As^{5+}) was the beginning of the dry season.

Hence, arsenic accumulates in soil, contaminates both surface and groundwater is taken up by the community (Lloyd-Smith and Wickens, 2000). Arsenic occurs naturally in groundwater from dissolution of arsenic-bearing aquifers, with concentrations typically ranging from <1−1000 µg/L. Elevated levels of arsenic are cause for concern because it is associated with a number of adverse health outcomes, including several types of cancer, vascular diseases, dermatological ailments, diabetes, respiratory diseases, cognitive decline, and infant mortality (Chen *et al*., 1995). This paper is therefore aimed at examining the spatial relationship between soil arsenic concentration distribution and the distribution of Buruli ulcer in the Amansie West district of Ghana, using Poisson Kriging method. Kriging is a group of geostatistical techniques to interpolate the value of a random field (e.g., the elevation, *z*, of the landscape as a function of the geographic location) at an unobserved location from observations of its value at nearby locations. The theory behind interpolation and extrapolation by kriging was developed by the French mathematician Georges Matheron based on the Master's thesis of Danie G. Krige, the pioneering plotter of distance-weighted average gold grades at the Witwatersrand reef complex in South Africa (Webster *et al.* 1994).

Kriging belongs to the family of linear least squares estimation algorithms. As illustrated in Figure 1, the aim of kriging is to estimate the value of an unknown real-valued function, f, at a point, x^*, given the values of the function at some other points, $x_1,....,x_n$. A kriging estimator is said to be *linear* because the predicted value $\hat{f}(x^*)$ is a linear combination that may be written as

$$\hat{f}(x^*) = \sum_{i=1}^{n} \lambda_i(x^*) f(x_i)$$

The weights $\lambda_i(x^*)$ are solutions of a system of linear equations which is obtained by assuming that f is a sample-path of a *random process,* $F(x)$ and that the error of prediction

$$\varepsilon(x) = \sum_{i=1}^{n} \lambda_i(x) F(x_i) - F(x)$$

is to be minimized in some sense. For instance, the so-called simple kriging assumption is that the mean and the covariance of $F(x)$ is known and then, the kriging predictor is the one that minimizes the variance of the prediction error (Mohammad *et al*., 2006). Although kriging was developed originally for applications in geostatistics, it is a general method of statistical interpolation that can be applied within any discipline to sampled data from random fields that satisfy the appropriate mathematical assumptions. To date kriging has been used in a variety of disciplines, including public health. Kriging as a geostatistical technique has a lot of advantages: It is an exact interpolator (that is if the control point coincides with the grid mode), relative index of the reliability of estimation in different regions, good indicator of data geometry, small nugget (or sll) gives a smaller kriging variance, minimizes the mean square error and a robust technique (i.e small changes in kriging parameters equals small changes in the results) (Gandhimathi *et al*., 2012).

METHODOLOGY

The Amansie West District falls within latitudes 6º 35 and 6º 51 North and Longitudes 1º 40 and 2º 05. It is located in the south-western part of Ashanti Region in the forest zone of Ghana. It shares boundaries with the Amansie East District in the west, Atwima Mponua District in the east, Atwima Nwabiagya District in the north and Amansie Central in the South. The District covers an area of about 1,364 sq. km. and forms about 5.4 percent of the total land area of the Ashanti Region (AWDP, 2005). The district lies entirely in the rainforest belt. It exhibits most semi-deciduous characteristics. The district is very rich in forest resources, such as timber, herbs of medicinal value and fuel wood. It also abounds in different species of tropical hardwood, notably Odum, Mahogany and Sapale. There are four main forest reserves in the district. These are: Oda River Forest Reserve, Apamprama Forest Reserve, Gyeni River Forest Reserve and Jimira Forest Reserve. The dominant soil type in the district is the ochrosols soils that are suitable for a number of crops such as plantain, cocyam, cassava, maize, legumes, oil palm, cocoa, coffee, citrus and pear. The district covers an area of 136,400 square kilometers. The year 2000 population census put the estimated population of the district at 108,273 with a population density of 62.8 persons per square kilometer. The district can be classified as predominantly rural. It has about 310 settlements fairly distributed within the district. Of the 310 settlements, only 19 have populations above 1000 and shows a large proportion constituting small settlement of farming communities.

Incidence of BU for each settlement over the period 1999-2011 was obtained from the Amansie West District Health Directorate and was entered as attributes for each settlement. The demographic data of the area was obtained from the 2000

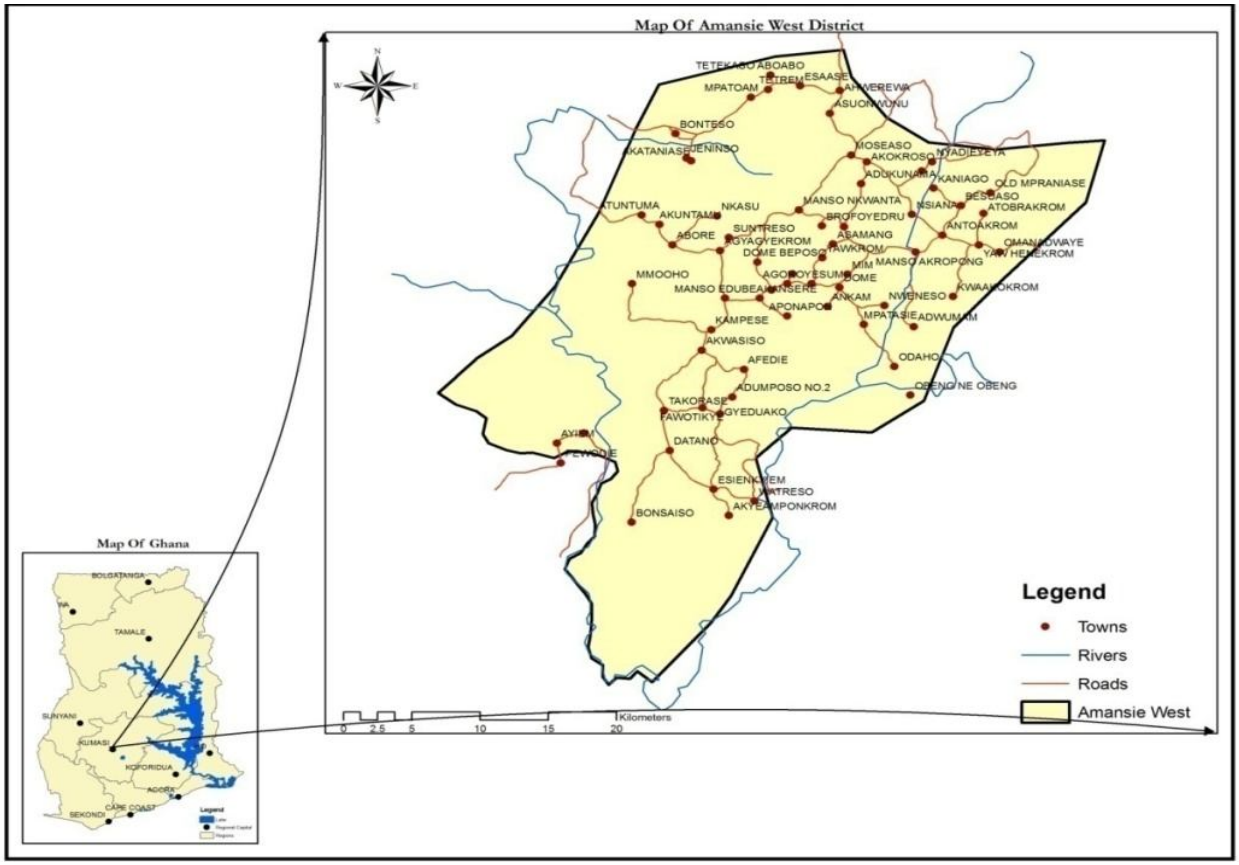


Figure 1. Map of the Amansie West District

Ghana Population and Housing Census Data. Spatial data were obtained by exacting the coordinates from the digitized boundary map of the Amansie West District using the topographic map obtained from the Survey and Mapping Division Accra, Ghana. Population from 1999-2011 was extrapolated based on the 1984 and 2000 population growth rates computed using data from Ghana Statistical Service Accra, Ghana. Buruli ulcer incidence per (100) people of the population was then computed for each settlement. Data were collected from Forty (40) Wells made of 14 sample points of Leptisols of average arsenic level 178 µg/L, Fluvosols 12 sample points of arsenic average of 628.3 µg/L and Acrisols of 16 sample of average arsenic 469.81 µg/L. Grids of nodes of interval 100 meters were generated on the soil map to predict the arsenic distribution on the study. Webster *et al*. (1994) looked at the effect of geological formation and land use on topsoil concentrations and found much smaller concentrations for most metals on the Argovian formation. The soil map with sampled arsenic data will thus acted as our source of areal information. This map is made up of 3 polygons that belong to one of the three soil formations. In the present application, no independent calibration of the soil maps existed and, for the purpose of illustration, the mean arsenic concentration within each formation was simply computed as the weighted average of all samples collected on that formation. The weight is the area of influence of each sample (that is, Thiessen polygon) in order to account for data clustering. In a situation where a soil map is available, areal data would simply be identified with concentrations recorded on representative profiles for each mapping unit (Kery *et al.*, 2004).

Disease mapping

The data used in this study is Buruli ulcer data, and the BU count data is discrete. To model these spatial data, the standard kriging algorithm like simple kriging is not right for the discrete distribution. It is essential to take into account the binomial or Poisson nature of the count data. The methodology for estimating a spatial Poisson distribution was introduced by Kaiser *et al*. (1997). They developed the spatial "auto-models" based on the Poisson distribution to be used to incorporate spatial dependencies among the variables. Monestiez *et al*. (2006) developed Poisson kriging to model spatially heterogeneous observation effort. The approach applied by Monestiez is similar to binomial co-kriging proposed by Oliver *et al*. (1998) except that count data followed a Poisson distribution. Poisson kriging was then generalised by Goovaerts (2005) to analyse cancer data under the assumption that all geographic units are the same in size. Spatial epidemiological studies have proven useful for understanding the geographical distribution and landscape-drivers of many diseases, including Puumala virus, Lyme borreliosis disease, malaria, and Human African Trypanosomiasis, among others (Linard, *et al.*, 2007). Disease mapping demands the interpolation of BU rate data to the nodes of a grid covering the study area.

Spatial Prediction of Arsenic levels

The arsenic concentrations levels in the soil were modeled by simple kriging with local mean (SKLm). This was based on a study by Goovaerts *et al*. (2010). He used ancillary data to

improve prediction of soil and crop attributes in agriculture using one of the following approaches: simple kriging, cokriging and Kriging with an External Drift. In this research, we used simple kriging with local mean (SKLm) by modeling the local average arsenic concentration levels (Z) on each soil type. The kriging estimates were then expressed as linear combination of the neighbouring primary Z- data and the local mean estimated at the various (n) geographical locations and the specific location u being predicted:

$$Z_{SKLm}(\mu) = \sum_{\alpha=1}^{n} \lambda_\alpha(\mu)[Z(\mu_\alpha) - m^*(\mu)] + m^*(\mu) \qquad \text{Equation 1}$$

$$= \lambda_\alpha(\mu) r(\mu_\alpha) + m^*(\mu)$$

Where $r(u_\alpha) = Z(u_\alpha) - m^*(u_\alpha)$ are referred to as residuals. The kriging weights were achieved by solving the following simple kriging system:

$$\sum_{\beta=1}^{n} \lambda_\beta(u) C_R(u_\alpha - u_\beta) = C_R(u_\alpha - u) \quad \alpha = 1,2,3 \ldots\ldots\ldots\ldots\ldots\ldots n, \qquad \text{Equation 2}$$

Where $C_R(h)$ is the covariance function of the residuals random function. R (u), not that of the variable Z itself.

Geostatistical analysis of Buruli ulcer rates

We took settlements with BU at Amansie West District to be N and BU incident at each given settlement was expressed as d(uα). Given that population at each geographical position (x_α, y_α) is n (u_α). We therefore, computed morbidity rate as z (u α) = d (u α)/n (u α).

Poisson Kriging

Poisson kriging was developed by Pierre Goovaerts and successfully applied to cancer data in USA (Goovaerts *et al*., 2005). We employed this approach based on the characteristics of BU count data. The 71 settlements used for this analysis can be modeled as the combination of the risk of contracting BU infection and a random (error term) because of spatially changing population size $n(u_\alpha)$:

$$Z(u_\alpha) = R(u_\alpha)(u_\alpha) + \varepsilon(u_\alpha) \qquad \alpha = 1 \ldots N \qquad \text{Equation 3}$$

The estimated number $d(u_\alpha)$ of BU cases at a geographical position has a fixed function R (u_α). This follows a Poisson distribution with one parameter (Number of BU cases) as product of the population size $n(u_\alpha)$ by the risk R (u_α). According to Goovaerts (2005) the following relations are met:

$$E[\varepsilon(u_\alpha)] = 0 \qquad \text{and Var } \varepsilon(u_\alpha)] = R(u_\alpha)/n(u_\alpha) \qquad \text{Equation 4}$$

$$E[Z(u_\alpha)] = E[R(u_\alpha)] = m \text{ and Var } [Z(u_\alpha)] = \text{Var}[R(u_\alpha)] + \text{Var}[\varepsilon(u_\alpha)] \qquad \text{Equation 5}$$

The probability of getting infected with BU disease at any given location u_α is computed as linear combination of K neighbouring data (Ali *et al.*, 2006)

$$\hat{r}pk(u_\alpha) = \sum_{i=1}^{K} \lambda i(u_i) z(u_i) \qquad \text{Equation 6}$$

Where $z(u_i)$ is the rate observed at location u_i. The kriging weights are obtained by finding solution to the following system of (K+1) linear equations:

$$\sum_{j=1}^{K} \lambda i(u_\alpha)\left[C_R(u_i - u_j) + \delta_{ij}\frac{m^*}{n(u_i)}\right] + \mu(u_i) = C_R(u_i - u_\alpha) \quad i = 1, \ldots\ldots, k$$

$$\sum_{j=1}^{K} \lambda_j(u_\alpha) = 1 \qquad \text{Equation 7}$$

Where δ_{ij}=1 if u_j and 0 otherwise, $n(u_i)$ is the population size at u_i, and m^* is the population- weighted mean of the set of N rates computed as :

$$m^* = \frac{\sum_{\alpha=1}^{N} n(\mu_\alpha) z(\mu_\alpha)}{\sum_{\alpha=1}^{N} n(u_\alpha)} \qquad \text{Equation 8}$$

The quantity $m^*/n(u_i)$ is error variance term that denotes the variability as a result of population size and is calculated directly under the Poisson model for the counts (Goovaerts., 2005). The addition of this term zero distance paved way to assign smaller kriging weights to rates that were calculated from smaller populations and deemed less reliable. The term $\mu(u_i)$ is Lagrange parameter that results from the reduction of the estimation variance subject to the unbiasedness constraint on the estimator. In order to solve the kriging system (Equation 5), one requires to have a model of the spatial covariance of the unknown risk, $C_R(h)$, or equally its semivariogram $\gamma_R(h)$= C_R (0)-C_R(h). The experimental variogram is computed using the following estimator developed by Monestiez *et al*., (2006).

$$\hat{r}\gamma_R(h) = \frac{1}{2\sum_{\alpha=1}^{N(h)} \frac{n(u_\alpha)n(u_\alpha+h)}{n(u_\alpha)+n(u_\alpha+h)}} \sum_{\alpha=1}^{N(h)} \left\{ \frac{n(u_\alpha)n(u_\alpha+h)}{n(v_\alpha)+n(u_\alpha+h)} [z(u_\alpha) - z(u_\alpha+h)]^2 - m^* \right\}$$

Equation 9

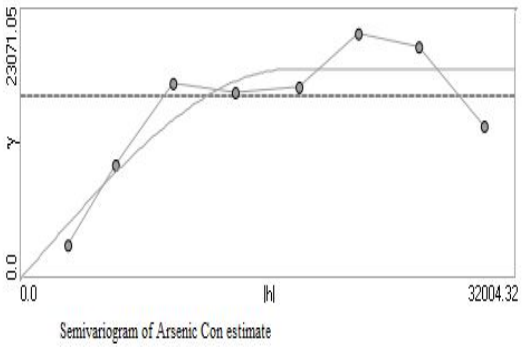
Where N (h) is the number of pairs of settlements separated by vector h

The different spatial increments $[z(u_\alpha)-z(u_\alpha+h)]^2$ are weighted by a function of their respective population sizes, $n(u_\alpha)n(u_\alpha+h)/(n(u_\alpha)+n(u_\alpha+h))$, a term which is inversely proportional to their standard deviation. Preference was given to pair data with small standard deviations. A permissible model $\gamma_R(h)$, was then fitted to the experimental semivariogram in order to obtain the semivariogram. In this work, the modeling was performed using the weighted least-square regression.

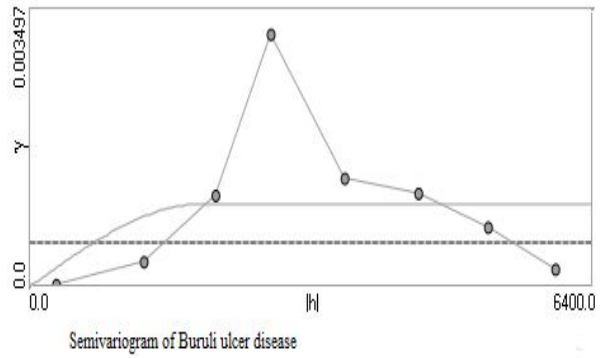
RESULTS AND DISCUSSIONS

The residuals of arsenic concentration of 40 sample data were used to compute experimental semivriogram model (equation 3). This residual semivariogram was estimated based on 8 lag count of size 4000 metres, Figure 1. The spatial spread of arsenic concentration was modeled using equation 1. The soil map and the two main rivers were overlaid on the

arsenic concentration estimate. The arsenic levels were very high at the basin of the two main rivers. This is where artisanal mining activities are of ascendency. There was large spatial autocorrelation of 17975.34m in soil arsenic concentration in Amansie West District. The Spherical model which was fitted through the residual arsenic data has 0.122 MSS errors. The nugget variance which is the unexplained error in the data was $5.144*10^{-6}$. Experimental risk semivariogram was computed using BU incidence rates from the 71 settlements of Amansie West District, using the population-weighted estimator (Equation 8). There were no observed differences between directional semivariograms, therefore, the spatial variability was considered isotropic and only the omnidirectional semivariograms is shown in Figure 3.1. The semivariogram for BU was estimated using 8 lags of a size 800 metres. We used weighted least-square regression to model the BU disease where the weights account for both the number of data pairs and the semivariogram value.



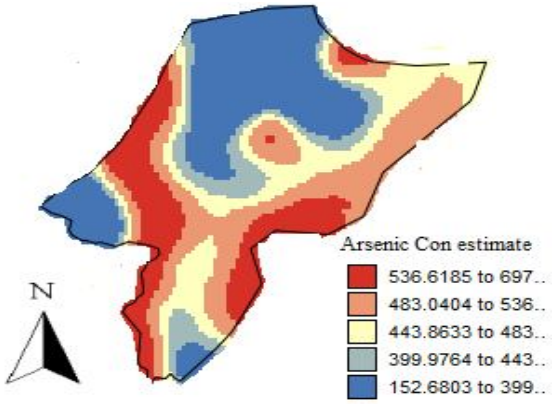
A



B

Figure 3.1. Semivariogram model for Arsenic concentration (A) and Semivariogram model for Buruli ulcer disease (B)

Buruli ulcer semivariogram is a spherical model with practical range of 1946.06 metres. The arsenic estimate has large spatial autocorrelation than that of the BU disease spread in the same area. This large range of spatial autocorrelation in arsenic concentration model shows a better spatial structure which may be speculated to be related to exposure of arsenic through human activities such as mining, agriculture and constructional activities into water bodies used by the community. This is supported by Duker *et al*., (2006) that the influence of arsenic on gold activities enhances the growth of the Mycobacterium and this may contribute to the spread of BU. The BU incidence estimate was overlaid on the soil map of the area to indicate spatial relation between the soil and the disease Figure 3.2. The boundary of the colour classes correspond to the deciles of the histogram of the risk estimates. The Buruli ulcer estimates created by the Poisson kriging (Figure 3.2**)** shows a large area of high incidence in the western and eastern part of the study area, where the mining activities is intensive. These regions have large patches of arsenic risk (Figure 3.2) and may relate to the BU disease.



A

Figure 3.2. Maps of Arsenic concentration on District boundary created by Poisson Kriging

Co-incidentally, these regions are drained by the two main rivers, Figure 3. The southern part has relatively higher risk of BU disease estimate and is underlain by Fluvosols which has the highest patches of arsenic level, Figure 3.3. This region has large patches of arsenic risk and may be related to BU disease. These areas are very close to the two main rivers in the study area where the artisanal mining activities are vigorously pursued. On the Leptisols and Acrisols soils, the arsenic risk levels are very low and this may partly account for the low level Buruli ulcer incidence in these areas.

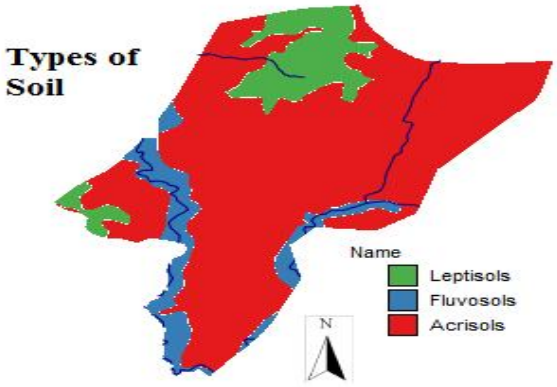


Figure 3.3. Map of soil with Arsenic concentration and the two main rivers of Amansie West District

The study found that the major consideration for the location of settlements in the study area is based primarily on proximity to and availability of water for drinking and other domestic purposes. As a result, many settlements are located within the distance of about 150 m from drainage basins. This finding is supported by Duker *et al*. (2004) who concluded in their study in the Amansie West that siting of rural settlements in the study area is based primarily on proximity to and availability of water for drinking and other domestic purposes.

Consequently, many settlements are located within the optimum buffer distance of 100 m from drainage channels (Duker *et al*. 2004). The water which is abstracted from the drainage basin is enriched in arsenic. The perpetual ingestion of the arsenic-enriched water through drinking and cooking is likely to enhance the growth and development of the Mycobacterium, the disease causing organism. In a related

Figure 3.4. arsenic enrich polluted river in the Amansie West District

development, Amofah *et al*. in 1993 studied 90 BU patients and found that 52 used surface water as the source of their drinking water. The result of the kriging analysis corroborates this observation as the research indicated that BU prevalence is highest where the inhabitants have ready access to domestic water supplies from arsenic-enriched surface drainage. Again, food crops that are grown on the plains may take up these high concentrations of arsenic and when consumed will make the individuals susceptible to several kinds of diseases including BU.

Conclusions

In Amansie West District, Poisson risk map has never been used to depict areas of high Buruli ulcer incidence and to help control this disease. In addition to BU incidence, arsenic risk level has been used as covariate variable to explain spatial relationship between BU disease and arsenic consumptions. Our risk map for Buruli ulcer and arsenic may be vital for identifying putative factors of increased disease risk and for assisting health officials take remedial actions. The Simple Kriging with Local mean (SKLm) was also used to characterize the arsenic risk as a covariate variable to BU disease incidence. This was based on the three main soil types in the area where sample were taken from each for analysis. There is a positive correlation between Arsenic concentration and BU disease incidence in most part of the study area. However, there are some places where correlation is negative indicating that BU may also be high but Arsenic may not be so. Poisson kriging was applied to Amansie West District BU incidence data as filter to produce more accurate spatial mapping because it takes population sizes into consideration. The BU disease is fast spreading through the boundaries especial Amansie Central and the other parts. Arsenic levels for the entire are is higher than the recommended WHO level. There is a need to check the movement of people and the activities they engage in. Further investigation looking into vegetation cover of the study area will help to identify high incidence risk of Buruli ulcer.

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