## Where are the Mycobacterium Ulcerans? Mapping the Risk and Vulnerable Areas of Mycobacterium Infection in the Amansie West District of Ghana

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**Abstract** *Mycobacterium Ulcerans* (MU) is the bacterium that causes Buruli ulcer (BU), a neglected tropical disease whose epidemiology has proven hard to pin down till date. This study was undertaken to map out the high and low risk areas of the disease in the Amansie West District, the most endemic constituency in Ghana. The disease affects people who live on less than a dollar a day and scourges mainly women and children. It is unclear where the bacterium lives in the environment and how it enters the human body. These slits in knowledge necessitated the study. Semivariograms were computed to determine the strength and the spatial dependency of the pattern of the disease. Kriging was chosen in the variogram modeling. The BU datasets exhibited a highly positively skewed histogram with possible outlying. The kriged map showed large patches of BU infections in the southern, eastern and western parts of the study area. Coincidentally, these areas are drained by the two major rivers, Oda and Offin and also characterized by intense mining and agricultural activity. The paper is of the view that the environmental characteristics and intense economic activity may be responsible for the high concentration of MU in the identified areas.

Keywords: variogram, kriging, spatial patterns, Buruli ulcer, Mycobacterium ulcerans

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## **1. Introduction**

It is well documented in literature that the wealth and health of the population in developing countries in general are interwoven with the surrounding environment in ways that make them both particularly valuable as custodians of environmental resources and also vulnerable to environmental health [1]. In these regions, the environment provides livelihood support systems for a greater proportion of the population in the area of agriculture, mining, forest resources and tourism attraction among others. Indeed, these sectors provide the muchneeded income and employment opportunities in fragile economies of developing countries. In the meantime, the environment is implicated as being home to and providing solace for disease causing organism that make the population highly vulnerable to poor health and poverty resulting in premature death and illness due to major environmental risks which account for one-fifth of the total burden of diseases [2]. In Sub-Sahara Africa (SSA), environmental factors such as pollution, poor sanitation, lack of drinking water, poor housing, and transport are a root cause of nearly 35% of the total burden of diseases (ibid). This is manifested by different spatial patterns of morbidity and mortality due to infectious diseases (respiratory diseases, malnutrition and diarrhea) on one hand, and neglected, but preventable tropical diseases on the other hand, at the same time [3].

Though medically diverse, neglected tropical diseases form a group by virtue of the fact that they flourish in impoverished environments and thrive best in tropical regions, where they tend to coexist [4]. Unfortunately, neglected tropical diseases have traditionally ranked low in national and international health efforts. They can cause massive but hidden and silent suffering, but the mortality rate is low as compared to well known HIV/AIDS, tuberculosis or malaria. Neglected tropical diseases have their breeding grounds in places challenged by socioeconomic progress, substandard housing, lack of access to safe water and sanitation and filthy environments [5].

Indeed, one of such preventable tropical diseases that continues to blight the lives of many rural folks in Ghana and threatens many more, is Buruli ulcer (BU). The disease is caused by *Mycobacterium ulcerans* (MU) which produces mycolactone, the toxin responsible for the extensive destruction of skin and soft tissue. It eventually leads to the formation of large ulcers, usually on the legs or arms [4]. BU represent some of the most common infections of the poorest people living in Ghana because they primarily badly affect the disenfranchised poor as well as selected indigenous populations [5]. It is the second most widespread *Mycobacterium* infection after tuberculosis [6]. The disease usually begins as a painless nodule and may gradually progress to massive skin ulceration [7]. The higher number of cases and the complications associated with the disease as well as its long-term socioeconomic impact could have a substantial effect on the national economy [8]. The disease is mostly found in rural areas located near wetlands and slowmoving rivers and may also be associated with the flooding and rapid environmental change [9].

The incubation period, the time between infection with Mycobacterium ulcerans and clinical presentation of Buruli ulcer is usually under three months [10]. The disease can affect any part of the body, but predominantly affects the limbs [11]. The most vulnerable groups are women and children under 15 years of age [11]. The clinical features of Buruli ulcer have been clearly defined by the World Health Organization [12]. BU starts as a papule when the localized swelling in the skin is less than one centimeter in diameter. The papule develops into a nodule when the swelling is one to two centimeters in diameter, attached and under the skin (subcutaneous). The nodule then develops further to become a plaque when the ulcer has irregular edges and is more than two centimeters in diameter [13]. Eventually, the plaque develops into ulcers with undermined edges whose real size becomes difficult to estimate visually. The base of the ulcer is filled with dead (necrotic) tissue [13]. Death due to Buruli ulcer is rare. The disease may, however, result in joint deformities (contractors) from excessive scarring, making movement of joints difficult. Loss of or severe damage to vital organs such as eyes, breast, or genitalia may occur. The main form of treatment is wide excision surgery, including amputation of limbs, which requires prolonged hospitalization, though indigenous knowledge of treatment has been acknowledged [14].

Globally, Buruli ulcer has been reported in more than 33 countries, mainly those with tropical and subtropical climates [4]. In 2013, 2630cases were reported from 14 countries globally and 2543 of them were from the African Region [15]. Ghana is the second most endemic country after Cote d'Ivoire, worldwide [7]. The overall national prevalence is 22.7 cases per every 100,000 inhabitants. The [16] reports that the disease is endemic in all the ten regions, but the Ashanti Region is the most endemic accounting for over 60% of all cases. The Amansie West District is the most endemic in the Region with a prevalence of 151 cases per 100,000 inhabitants (GHS, 2012).

Table 1. Trend of Buruli ulcer diseases in the Ashanti Region, 2008-2010

Nodules         Ulcer         Other           2008         235         24         36         164         0           2009         177         15         22         129         46	Year	Nou	Decument	Clin	nical Forn	18
2009 177 15 22 129 46	i ear	INEW	Recurrent	Nodules	Ulcer	Others
	2008	235	24	36	164	0
2010 Half Vear 251 5 72 180 47	2009	177	15	22	129	46
2010 Hall Teal 251 5 72 180 47	2010 Half Year	251	5	72	180	47

Source: Ghana Health Service, 2012.

The continued prevalence of BU in the Ashanti Region has become a source of worry, a situation which indicates that the extensive public education on prevention and treatment have not yielded the needed results. Buruli Ulcer drew national attention in 1993 when several cases were reported from Tontokrom in the Amansie West District [17], although earlier cases had been reported from the Densu and Afram plains, [18].

To the best of our knowledge, the origin of the Mycobacterium ulcerans is evasive, though most epidemiological data and some hypothesis have associated the outbreak and emergence of the pathogen and the disease with an aquatic environment [19]. Similarly, the natural reservoir and precise mode of transmission have escaped scientific lenses. What is more, early detection of cases has also proven to be difficult, although a 2014 WHO report indicates that researchers have devised a rapid diagnostic test using thin-layer chromatography (TLC) to detect mycolactone, the toxin that causes tissue damage in Buruli ulcer. The researchers used boronateassisted fluoroscent thin-layer chromatography (F-TLC) to selectively detect mycolactone when visualized with ultraviolet light. This scientific feat is yet to become operational worldwide [15].

In this paper, we seek to map out the high and low risk areas of BU in Amansie West District of Ghana using kriging interpolation method belonging to the geostatistics family. With this method, we believe that besides the visual representation of the risk areas, we will be able to ascertain some of the environmental factors that predispose humans to infection. The study will eventually add to the local understanding of the disease epidemiology. After the literature review, the methodology is given the maximum prominence in the next section. This is followed by the results and analysis. The conclusion and recommendations are presented in the final section.

## 2. Methodology

### 2.1. Study Area

The Amansie West District falls within latitudes 6° 35` and 6° 51` North and Longitudes 1° 40` and 2° 05`. It is located in the southwestern part of the 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.

The District lies entirely in the rainforest belt. It exhibits most semi-deciduous characteristics. The district is drained by the Oda and Offin rivers in the north with their tributaries such as the Pumpin, Emuuna and Jeni. These rivers have, however, been polluted by numerous mining activities in the district.

The year 2000 population census puts 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 the small settlement of farming communities. The economy is basically an agrarian with the majority of the people drawing their incomes from farming, mining and petty trading.

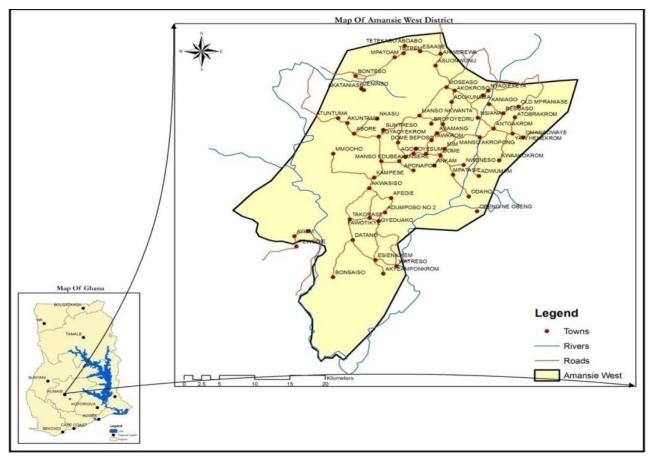


Figure 1. Map of the Amansie West District (Source: field work)

In spite of the government's efforts to provide potable water for the communities, access to potable water is a major problem in several of the communities in the Amansie West District. The main sources of water supply in the district are rivers and streams, hand dug, wells, boreholes with pumps and pipe-borne water.

The major health facility in the district is the St. Martin's Catholic Hospital at Agroyesum which also doubles a specialized center for the treatment of Buruli ulcer in the Ashanti region and a general Hospital for the people of the Amansie west district. Additionally, there are twenty-two other health centers, including four maternity homes [16]. This seriously affects health delivery in the district.

### 2.2. Dataset and Assumptions

As a starting point, we digitized a topographic map of the study area which was obtained from the Survey Department, using Arc-GIS version 9.2. The map was Geo-referenced by defining the X and Y coordinates of corner points of the map into a War Office coordinate system. The boundary map of the study area was digitized as a polygon feature and the communities were digitized as point features. Again, we compiled the total number of reported cases of BU from 1999 to 2012 which was obtained from the district health directorate of Amansie West. The data were entered as attributes of the point features (settlement).

### 2.3. Geostatistical Analysis of BU

The second stage of the methodological process involves a standard geostatistical analysis, which includes exploratory data analysis, semivariogram analysis of the spatial structure, surface interpolation, and display of the results. Geostatistics is a collection of techniques aimed at modeling spatially correlated data, and using the selected model for estimating values in localities not visited during the sampling [20]. The population is viewed as a stochastic process in space, and the data gathered is regarded as one sampling realization of that process, which is then used to estimate it. A given number N of entities (town) denote the number of recorded mortality cases by d( $x_{\alpha}$ ) and the size of the population at risk n( $x_{\alpha}$ ), where  $x_{\infty}$  is the size of the risk entities at  $\infty$ . Following [21] entities are referenced geographically by their centroids with the vector of spatial coordinates  $u(x) = (x_{\alpha}, y_{\alpha})$ , which means that the actual spatial support (i.e. Size and shape of the towns) are ignored in the analysis. The empirical or observed mortality rates are then denoted as:

$$z(x_{\alpha}) = \frac{d(x_{\alpha})}{n(x_{\alpha})} \tag{1}$$

Generally at each place x, where x represents the spatial coordinates of the place, a value Z(x) is given as:

$$Z(x) = m(x) + \varepsilon(x)$$
<sup>(2)</sup>

Where m(x) is some function of x, known as the drift, and  $\varepsilon(x)$  is a random variable with variance defined by:

$$\operatorname{Var}\left[\varepsilon\left(x\right)+\varepsilon\left(x+h\right)\right]=E\left\{\left[\varepsilon\left(x\right)-\varepsilon\left(x+h\right)\right]^{2}\right\}=2\gamma\left(h\right) (3)$$

In the above expression, h denotes a vector and the lag, that separates the two places x and x+h in both distance and direction. In some instance, m(x) is constant, at least locally and therefore can be replaced by  $\mu$ . The variance expression for equation (2) becomes

$$\operatorname{Var}\left[Z(x) - Z(x+h)\right] = E\left\{\left[Z(x) - Z(x+h)\right]^{2}\right\} = 2\gamma(h)$$
(4)

The quantity  $\gamma(h)$  is the semivariance, and the function that relates  $\gamma$  to h is the variogram. In situations where the mean and variance remain constant over a whole region, the semivariance is equivalent to the autocovariance. The covariance at lag h is given by:

$$C(h) = E\left\{ \left[ Z(x) - \mu \right] \left[ Z(x+h) - \mu \right] \right\}$$
(5)

The semivariance is related to the covariance at lag h simply by:

$$\gamma(h) = C(0) - C(h)$$

Where C(0) is the covariance at zero lag or the priori variance of the process. The variogram always exists, and this makes it more generally useful, and in fact it is central to geostatistics.

#### 2.4. Basic Model

The rates recorded at N=77 towns can be modeled as the sum of the risk of developing Buruli ulcer and a random component (error term) due to spatially varying population size,  $n(x_{\alpha})$ :

$$Z(x_{\alpha}) = R(x_{\alpha}) + \varepsilon(x_{\alpha}) \quad \alpha = 1, \dots N$$
(6)

Conditionally, to a fixed risk function, the counts  $d(x_{\alpha})$  follows a binomial distribution with parameters R0 and n0. In other words, there are two possible outcomes: having Buruli ulcer or not, with  $R(x_{\alpha})$  being the probability of having the disease within a township with independent risk set  $n(x_{\alpha})$ .

# **2.5.** Geostatistical Analysis of BU: Statistical Relation

Some assumptions were made in this analysis:

$$E[\varepsilon(x_{\alpha})] = 0$$
  
and  $Var[\varepsilon(x_{\alpha})] = R(x_{\alpha}) * \{1 - R(x_{\alpha})\} / n(x_{\alpha})$  (7)

From (6) and (7) we get the following expression for expectations and variance:

$$E[Z(x_{\alpha})] = E[R(x_{\alpha})] = \mu$$
  
and  $Var[Z(x_{\alpha})] = Var[R(x_{\alpha})] + Var[\varepsilon(x_{\alpha})]$  (8)

For estimation purposes and following with Oliver *et al.* (1998), the variance of the error term can be approximated as:

$$\operatorname{Var}\left[\varepsilon(x_{\alpha})\right] = \mu x(1-\mu) / n(x_{\alpha}) \tag{9}$$

where the mean parameter  $\mu$  is estimated by the population-weighted average of rates,  $\overline{Z}$ . The risk over a given entity with centroid  $x_{\alpha}$  is estimated from  $s(x_{\alpha})$  neighboring observed rates as:

$$\overline{R}(x_{\alpha}) = \sum_{i=1}^{s(x_{\alpha})} \lambda_i(x_{\alpha}) z(x_{\alpha})$$
(10)

The kriging weights are solution of the following system:

$$\sum_{j=1}^{s(x_{\alpha})} \lambda_j(x_{\alpha}) C(x_i - x_j) + \mu(x_{\alpha})$$

$$= C_R(x_i - x_{\alpha}) \quad \text{for } i = 1, \dots, s(x_{\alpha})$$
(11)

Where;

$$\sum_{j=1}^{s(x_{\alpha})} \lambda_j(x_{\alpha}) = 1.$$
(12)

# **2.6. Estimating the Variogram of the Frequencies**

In geostatistics analysis, variogram is a basic tool to measure the spatial correlation of the studied attributes. Therefore, the experimental variogram was computed to measure the spatial correlation of the BU cases using the following formula:

$$\gamma(h) = \frac{1}{2M(h)} \sum_{i=1}^{M(h)} \left[ Z(x_i) - Z(x_i + h) \right]^2 \quad (13)$$

The solid is the model that was fitted to the experimental variogram of the risk by weighted leastsquares approximation using the program ArcGIS. The function is Exponential model given by:

$$\gamma(h) = c_0 + c_1 \left\{ 1 - \exp\left(-\frac{h}{a}\right) \right\}$$
(14)

Where  $\gamma(h)$  is the semivariance, *h* the lag, *a* is the range,  $C_0$  the variance and  $C_0+C_1$  the sill.

### 2.7. Kriging Algorithm

$$\hat{Z}(x_0) = \sum_{i=1}^n \lambda_i z(x_i)$$
(15)

Where *n* is the number of sample points,  $\lambda_i$  the weights of each sample point. Where the estimate is unbiased, the weights are made to sum up to 1. That is  $\sum_{i=1}^{n} \lambda_i = 1$ .

The prediction of the variance is given by:

$$\sigma^{2}(x_{0}) = \sum_{i=1}^{n} \lambda_{i} \gamma(x_{i}, x_{0}) + \phi$$

Where  $\sigma^2$  is variance,  $\gamma(x_i, x_0)$  the semivariance between samples point  $x_i$  and unvisited point  $x_0$  and  $\phi$ the Langrange multiplier.

### **3. Results**

Our results show that the histogram has exhibited a strongly positive skewness (Figure 2A). Furthermore,

possible outliers can also be identified. The strongly skewed distribution and outliers can cause problems leading to biased conclusions in statistical and geostatistical analyses.

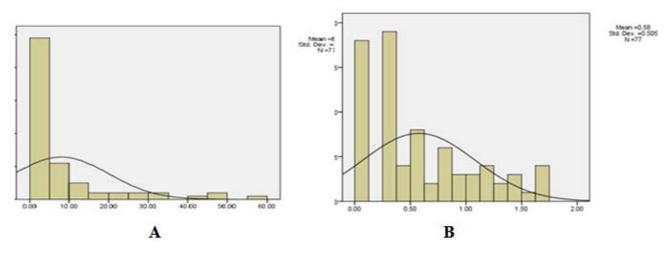


Figure 2. A: Sample data of BU cases; B: Log transformed data of BU cases

Kurtosis which should be near 3 for the normal distribution is, however, 10.514 (Figure 2A) indicating that the BU data set is heavily skewed. The BU cases had large variation, it ranged from 1 to 77 cases which means that the BU cases in the study area were not evenly distributed (see Table 2).

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Statistics	Untransformed data	Transformed data
Count	78	78
Minimum	1	0
Maximum	76	4.3307
Mean	8.8846	1.3674
Std. Dev	14.14	1.2036
Skewness	2.7028	0.70097
Kurtosis	10.514	2.5147
1 <sup>st</sup> Quartile	2	0.69315
Median	3	1.0986
3 <sup>rd</sup> Quartile	8	2.0794

Since the probability distribution of the raw data sets was heavily skewed, it was necessary to carry out data transformation prior to further statistical and geostatistical analyses. All the raw data on BU cases were strongly positively skewed, with the skews much higher than 0, implying that there were some extremely high values in the data sets. The kurtoses were also very sharp, caused by the fact that the majority of samples is clustered at relatively low values.

The isotropic variogram of BU (Figure 3) exhibits a very good structure, which can be well fitted with exponential model. This means that the direction (angle) has no influence on the practical range (autocorrelation). The nugget effect is very small (C0=0.000), showing that the sampling density is adequate to reveal the spatial structures (see Table 3). Furthermore, the range of 35090.4 feet implies that the length of the spatial autocorrelation is much longer than the sampling interval of 14282 feet (Table 3). The sampling design is therefore appropriate for this study and it is expected that a good spatial structure will be shown on the interpolated map.

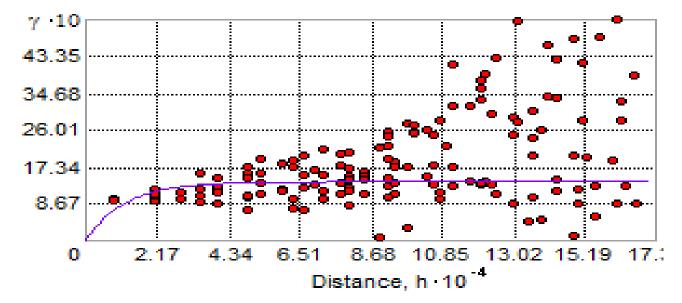


Figure 3. Isotropic Variogram Model [1.3785\*Exponential (3509)]

Table 5. Dest litted semivariogram model parameter of De	
Model	Exponential
Partial Sill (C0 +C)	0.26502
Nugget (C0)	0
Lag size	14282
Number of lag	12
Major range	35090.4
Angle tolerance	45°
Angle direction	0 °

Table 3. Best fitted semivariogram model parameter of BU data

#### 3.1. Risk Map of Buruli Ulcer

The isotropic variogram model was then used to develop risk maps at the district level using ordinary kriging, see Figure 4. In all cases, the estimation was based on the K=32 closest observations, which were selected according to the population- weighted districts, for ordinary kriging. The krigged map of Buruli ulcer risk (B) was smoother than the map for the raw rates (A) because the noise, due to the population size was filtered.

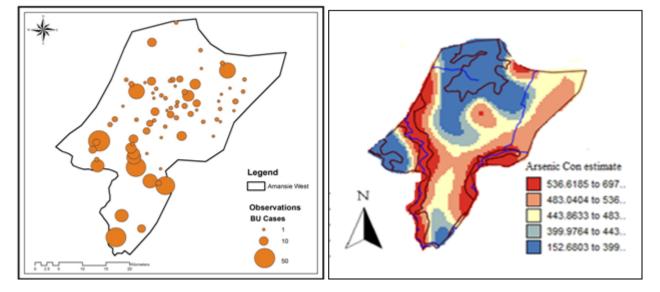


Figure 4. (A) Risk Map of BU without Water Bodies and Soil; (B) Risk Map of BU with Water Bodies and Soil

It can be observed from Figure 4 that the spatial dependence as evident in the variogram comes up because there are variations in the risk patches. The communities closer to the south generally appear to have the largest frequencies and apparent risk than communities in the central parts of the study area where the majority of the settlements is located.

## **3.2.** Predicting Errors for Ordinary Kriging in the Training Dataset

We also predicted the errors for the ordinary krigging in the data set and our results showed that the value of the mean prediction error (0.6078) was close to zero, showing that the predicted values were unbiased (Table 4). The mean standardized prediction error (-0.0533) was also close to zero, indicating that the prediction error was unbiased. The average standard error (22.59) was greater than the root-mean-square of predicted-errors (13.09) (Table 4).

Mean:	0.6078
Root-Mean- Square:	13.09
Average- Standard Error:	22.59
Mean Standardized:	-0.0533
Root-Mean- Square Standardized:	0.8123

The results indicate that the model slightly overestimates the variability of BU cases. Finally, the rootmean-square standardized (0.8123) prediction error is less close to one, and thus corresponds to a good estimate (Table 4).

#### **3.3. Predicting Errors in Validation Dataset**

The research further showed that the value of the mean prediction error value of -0.432 was less bias (see Table 5). The average standard error (9.41) is lower than root-mean-square of predicted-errors (14.79), which shows that the model over-estimates the variability of BU cases. Prediction errors for validation has better root-mean-square standardized of 0.71 which was very close to 1 indicating that the model was very good as in Table 5.

Table 5. Prediction Errors For Validation Dataset			
Mean:	-0.432		
Root-Mean- Square:	9.41		
Average- Standard Error:	14.79		
Mean Standardized:	-0.089		
Root-Mean- Square Standardized:	0.71		

### 4. Discusions

The results of the study have once again demonstrated the nexus between the environment and human health. The results of the study indicate that the origin of MU and the risk of infection in the Amansie West district may be associated with the aquatic environment. This is evidenced by the patches on the risk map. The larger patches on the risk map represent a high risk and vulnerable areas. The smaller patches represent low risk and less vulnerable areas. Coincidentally, the high risk areas (Eastern and Western portion of the risk map, figure 4) are drained by the two major rivers and are also characterized by intensive agricultural activity, intense artisanal mining and high rate of deforestation. Again the high risk areas are common in arsenic-rich soils and also experiences periodic flooding. These characteristics have been hypothesized in literature as being responsible for the occurrence and re-occurrence of BU in most endemic areas of the world [6,22].

BU in the Amansie West District is common in settlements where intense artisanal mining activities are prevalent. Artisanal mining has been observed to be responsible for the higher levels of Arsenic (As) concentrations in such environment [22]. According to [6], Arsenic may play a vital role in the spatial distribution of MU. This is because communities where BU is a serious health threat, concentration of (As) in surface and ground water has been higher than average. Mining, which is the second contributor to the District's economy, is an activity which dates back to the pre-colonial era. The sector currently employs about 22 per cent of the labour force in the District, mainly the male population. Artisanal and small-scale mining is a significant sector that provides a means of livelihood for the local community and produces a sizeable proportion of the world's extractive commodities, but it is also associated with serious negative social, environmental, and security consequences [14]. Even as Artisanal and small-scale mining has potential to contribute positively to social and economic development and can provide much-needed income in fragile rural economies, the sector is closely linked to human health and poverty. The problem has therefore been how to strike a balance between livelihood and MU infections. The usefulness of these rivers/streams to the District is limited due to the level of pollution from mining activities. The pollution of the water bodies in the District poses a great health threat to the communities that use these rivers as sources of drinking water and other domestic purposes.

Apart from the artisanal mining, environmental change which is due to deforestation might be responsible for the high risk in the communities near the two major rivers. Thus the recent marked increase of prevalence rate in the endemic areas is attributed to environmental changes taking place at an alarming rate. Supporting this hypothesis, [23] revealed that in Benin, the ratio of infection was 100 per 100,000 people in communities with environmental changes where as in those areas without environmental changes, the ratio was about 20 per 100,000 people. A similar situation prevails in the most endemic areas in the Amansie West district. The Amansie West District is home to the Datano-Essienkyiem Forest Reserve and the Apiaprama Forest Reserves. These forest reserves are located between Tontokrom and Watreso are fast being depleted because of the intense agriculture activity through the felling of trees and tilling of the land. These physical activities on the natural environment have the potential of elevating MU onto the surface of the land thereby increasing the rate of infection.

Intense agricultural activity on the flood plains of the two major rivers might also be another reason for the high incidence of BU in the settlements in the southern part of the study area. The agricultural activities lead to the alteration of the natural environment and thus have the potential to elevate the *Mycobaterium oceans* onto the surface of the soil. Farmers are therefore highly exposed to the risk of MU infections. These findings confirm the

studies of [6], which found that most farmlands are situated close to the two major rivers and food crops are mainly cultivated in the flood plains because they are fertile and easy to irrigate during dry periods. The flood plains themselves comprise mainly recent sediments transported down stream and deposited on the adjacent land during flood events. If the suspended load includes arsenic bearing minerals, they are also deposited on the flood plains during flood events. However, the high arsenic locations are more restricted in spatial extent and therefore influence only part of the agricultural land and food crop consumption [6]. More importantly, high arsenic might be a measure or reflection of more general contamination (e.g., from mining), which has rendered land unsuitable for agriculture. James et al., [24], also identified three risk areas in Benin according to origin of patients reporting at hospitals with Buruli ulcer and noted that most of them were coming from Laguna areas of coastal Benin, marshy inland areas where market crops and rice are cultivated, and river valley areas.

The prevalence of BU corresponds to the quality of the natural environment as well as the socioeconomic quality of life. Apart from the physical damage that it inflicts on the human body, it drains the scarce resources that could have been used to improve the socioeconomic conditions of the District. For instance, literature on the disease in Ghana, (see [25,26] indicate that on average, treatment costs (direct) are estimated at \$780 (US) per patient. Outpatients who are seeking early treatment could pay about \$20-30 and the cost may even escalate with limited hospitalization. However, in a poverty stricken district where the average daily income is less than \$2 a day and where family members are often forced to stay away from home for long periods in order to give care to their afflicted relative for more than 120 days, the overall socioeconomic impact, both on the local economy and community development may even be devastating.

### 5. Conclusion

The geostatistical analysis applied in this research has led to an optimal estimation and mapping of the risk areas and the environmental factors that may contribute to the disease prevalence. The study has also shown that the risk is higher in the settlements where artisanal mining and farming is prevalent. The prediction map which is based on kriging interpolation and kriging standard deviation provides useful information for risk assessment and decision support. We recommend that as a first strategy, an alternative source of potable water could be provided for the communities in the high risk zones to minimize the frequency of contact with the rivers and streams in order to reduce the rate of infection. Secondly, whiles we recognized that health and well being of members of the communities cannot be separated from the natural environment (physical, chemical and microbiological), it is always important that interventions for livelihood support systems designed for the affected communities strike a balance with the environment in a mutually inclusive manner so that the over dependence on the environmental resources, which also predisposes the population to infection is minimized considerably.

## References

- Siegrist, J., Marmot, M. Health inequalities and the psychosocial environment-two scientific challenges. *Social Science & Medicine*, 58:1463-1473, 2004.
- [2] World Health Organization. Report of the World Health Organization 7th Advisory Group Meeting on Buruli ulcer, Geneva, Switzerland. Geneva, Switzerland: World Health Organization, 8-11 March 2004.
- [3] McGranahan, G., Songsore, J. and Kjellen, M. Sustainability, Poverty and Urban Environment Transitions. Sustainability, the Environment and Urbanization, London: *Earthscan*, pp. 21-43, 1996.
- [4] World Health Organization. Resolution WHA57.1. Surveillance and control of Mycobacterium ulcerans disease (Buruli ulcer), In: 57th World Health Assembly. Geneva, Switzerland: World Health Organization, 2010.
- [5] Hotez, P.J., Molyneux, D.H., Fenwick, A., Kumaresan, J. and Ehrlich, S. Control of neglected tropical diseases. *New Eng J Med* 357: 1018-1027, 2007.
- [6] Duker A. A., Carranza E.J.M. and Hale M. Spatial dependency of Buruli ulcer prevalence on arsenic-enriched domains in Amansie West District, Ghana: implications for arsenic mediation in Mycobacterium ulcerans infection. *Int J Health Geogr* 3: 19, 2004.
- [7] World Health Organization. Buruli ulcer Diagnosis of Mycobacterium Ulcerans Disease. Geneva, Switzerland: World Health Organization, 2012.
- [8] Chauty A. The role of antibiotics in the management of Buruli ulcer at the Pobe Center in Benin, in Proceedings of the 7th WHO Advisory Group Meeting on Buruli Ulcer, WHO, Geneva, Switzerland, (2004).
- [9] Merritt R.W., Walker, E.D.and Boakye, D.A. Ecology and transmission of Buruli ulcer disease: A systematic review. *PLoS Negl. Trop. Dis.*, 4:911-911, 2010.
- [10] Bone, E. Geospatial Modelling of Buruli ulcer Prevalence in Amansie West District, Ghana. Unpublished thesis, 2010.
- [11] Asiedu K., Scherpbier, R. and Raviglione, M. Buruli Ulcer: Mycobacterium ulcerans Infection. World Health Organisation, Global Buruli Ulcer Initiative, pp. 87-92, 2000.
- [12] Marston B.J., Diallo, M.O., Horsburgh, C.R., Diomande, I., Saki, M. Z., Kanga, J. Emergence of Buruli ulcer disease in the Daloa region of Cote D'ivoire. *J Trop Med. Am*, 52: 219-224, 1995.

- [13] Amofah G.K., Bonsu, F., Tetteh, C., Okrah, J., Asamoa, K. Buruli ulcer in Ghana: Results of a National Case Search. *Emerging Infectious Diseases* 8: 167-170, 2002.
- [14] Owusu-Sekyere E. Managing the Buruli ulcer morbidity in the Amansie West District of Ghana: Can Indigenous Knowledge Succeed? *International Journal of Medicine and Medical Sciences* 4: 180-185, 2012.
- [15] World Health Organization Accelerated detection of mycolactone production and response to antibiotic treatment in a mouse model of *Mycobacterium ulcerans* disease. DASH at Harvard University, 8(1):e2618, 2014.
- [16] Ghana Health Service (GHS). Strategic Objectives and New Paradigm of the Ministry of Health. Ashanti Regional Health Directorate in 2012.
- [17] Ministry of Health (MOH). Public Health Division, Annual Report, 2004.
- [18] Van der Werf, T.S., van der Graaf, W. T. A., Groothuis, D. G. and Knell, A. J. Mycobacterium ulcerans infection in Ashanti Region, Ghana. *Trans Roy Soc Trop Med* 1018. 974, 1989.
- [19] Marsollier L., Sévérin T., Aubry J. and Merritt R. W. Aquatic snails, Passive Hosts of Mycobacterium ulcerans. *Appl Environ Microbiol* 70: 6296-6298, 2002.
- [20] Cesaroni. G. Individual and area-based indicators of socioeconomic status and childhood asthma. *The European Respiratory Journal*, 22:619-624, 2003.
- [21] Oliver, M. A. Binomial cokriging for estimating and mapping the risk of childhood cancer. *IMA Journal of Mahematics Applied in Medicine and Biology*, pp. 279-297, 1998.
- [22] Owusu-Sekyere E., Bonya, E., and Harris, E. Buruli Ulcer Morbidity and Soil Arsenic Linkages in the Amansie West District of Ghana: A Geostatistical Approach. *Asian Journal of Science and Technology* Vol. 4, Issue 03, pp. 037-043, March, 2013
- [23] Aiga, H., Amano, T., Cairncross, S. Assessing water-related risk factors for Buruli ulcer: a case-control study in Ghana. *Am J Trop Med Hyg* 71: 387-92, 2004.
- [24] James K., Attipou K.K. and James Y.E. Buruli ulcer in Togo: A hospital study. *Sante* 13(1): 43-7, 2003.
- [25] Asiedu K. and Etuaful, S. Socioeconmoic implications of Buruli ulcer in Ghana: a three-year review. Am J Trop Med Hyg, 59(6):1015-1022, 1998.
- [26] Oliver M.A. and Webster R. Kriging: A method of interpolating for geographical information systems. *In J Geogr information Syst* 4: 313-323, 1990.