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Research article

MODELING AND SIMULATION TO MONITOR HETEROGENEOUS INFLUENCES ON PARTIAL DEPOSITION OF PATHOGEN IN LATERITIC AND SILTY FORMATION, SAGBAMA, BAYELSA STATE OF NIGERIA

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Abstract

Pathogenic depositions were investigated on heterogeneous formation, the study were able to monitor the behaviour of pathogen in heterogeneous deposition influencing partial concentration of pathogen. Lateritic and silty formation were observed to express partial deposition base on its fluctuated deposit on void ratio and permeability, such formation developed vacillation on these parameters consequently generated partial deposition of pathogen in the study location. Monitoring the rate of partial concentration deposition was possible through mathematical modeling approach, the system were developed base on these parameters from predominant formation characteristics in study area, these generated derived model through the developed governing equation, simulating these model generated theoretical values that were compared with experimental results, both parameters generated best fits validating the model, experts will definitely applied this concept in monitoring and evaluation of pathogen deposits in the study area.

Keywords: modeling, heterogeneous, pathogen, lateritic and silty formation

1. Introduction

Anthrax is known to be acute bacterial infection of primarily herbivores, which is infectious to humans. The etiologic agent, *Bacillus anthracis*, is a gram-positive spore developing rod shaped microbes. Animals become contaminated by ingesting spores or perhaps by being bitten by flies that have fed on an infected animal or carcass (Ebedes, 1976). Contaminated animals are normally found dead as death can happen within 24 hours. (Whitford and

Hugh-Jones, 1994). Anthrax can be established universal affecting wildlife, livestock, and humans. During epidemics in 1959/60 and 1970 in the Kruger National Park, South Africa, anthrax deaths numbered in the thousands (De vas 1976). Livestock luggage's are identified to contribute to human cases through the cutaneous gastrointestinal and inhalation route. In 2000, the first gastrointestinal cases were reported in the United States after the family ate beef from an infected carcass (Dragon and Rennie 2001: Pamala, 2002).Although environmental contamination with *B. anthracis* spores occurs because of wildlife and domestic livestock cases, the degree or level of pollution from each case is unknown. Anthrax spores are known to persevere in the surroundings for years and are opposed to ecological factors (Turnbull 1996). Spores may be found in soil contaminated by diseased animals or in diseased animal products such as hair, wool, hides, and bones (Beyer, et al 1995, Lindedeque and Turnbull 1994; Eluozo, and Afiibor,2013). Very little research has been done on anthrax spore survival under natural conditions. Eight hundred and eighty four million people were estimated from WHO to lack access to enhanced water sources, and estimated 2.6 billion populace do not have access to enhanced sanitation (UNICEF and WHO 2010a UNICEF and WHO 2010b UNICEF and WHO 2010c). In 2000, it is confound that Water Supply and Sanitation joint Council (WSSCC), a universal multi-partner organization intended at enhancing entrance to safe water and hygiene, established three precise targets for water supply and sanitation: 1) decrease the amount of people lacking access to hygienic sanitation facilities by one half by 2015, 2) decrease the amount of people without access to a sustainable source of quality drinking water by one half by 2015 (where superiority water is defined as assembly the WHO guidelines for safe drinking water), and to provide water, sanitation and hygiene for all by 2025 where sanitation was defined as full coverage of hand washing, safe disposal of feces, as well as safe water handling and storage (UNICEF and WHO 2000). eight goals has been confound to have been set by united nations to achieve this Millennium Development Goals (MDGs) the aim is to increase equality and decrease poverty universal, and among these, was the goal is to decrease the number of people who do not have access to safe water and improved sanitation by half by 2015 (UN 2011). Ever since 2000, exponential coverage of 7 and 10% worldwide for superior sanitation and water access respectively has occurred. However, if radical improvements towards the MDGs are not prepared, then in 2015 an predictable 2.7 billion people were confirm have access to enhanced sanitation, more so 672 million will be lacking better drinking water sources, reaching the MDG for water access and missing the sanitation target by 13% (UN 2010; UNICEF and WHO 2010c). The load of lack of access to secure water sources and enhanced hygiene falls heavily on people in developing nations and is even a more ordinary trouble for people living in rural areas compared to those living in urban environments (UNICEF and WHO 2010c). Rural populations account for around 84% of the people lacking access to improved water sources and sanitation services (UNICEF and WHO 2000). The WHO defines enhanced drinking water sources as those with knowledge that is most likely to deliver safe water to persons, such as family relations to piped water, public standpipes, boreholes, protected wells, and rainwater catchments (WHO 2004). It is significant to note that insecure wells, springs, water sold from vendors and tanker trucks fall under the heading of "unimproved water sources." (WHO 2004).Pathogens are frequently multiply in low concentrations into water supplies making them hard and costly to detect. But some microorganisms can be used to indicate pathogen existence in water; however the association is not always a straight connection (Ashbolt et al. 2001; EPA 2009). Ashbolt et al. (2001) describe three types of microbial indicators: 1) process

indicators, 2) fecal indicators, and 3) index organisms. Microbes are deposited in intestines of warm-blooded mammals and are discarding into the environment in excreta (Ashbolt et al. 2001; EPA 2010). Total coliform bacteria may occur in human intestines; these sources of contaminants are found in animal excreta, soil, and from other man made activities (EPA 2010). Total coliforms are considered process indicators and used for drinking water analysis confirmed to notice the presence of pollutants; however they do not precisely compare to pathogen pollution. The existence of total coliforms in treated drinking water indicates incomplete treatment, treatment failure, or post-treatment contamination. Fecal coliforms and *E. coli* are more closely linked to fecal contamination from warm-blooded mammals than total coliforms, although both can be found in the environment from non-fecal sources (Ashbolt et al. 2001; EPA 2010). Fecal coliforms and *E. coli* are less useful as environmental indicators of water quality due to the possibility of nonfecal origins, but they are generally good indicators of fecal contamination in drinking water (EPA 2010). *E. coli* is not only recommended as an indicator of fecal contamination, but can also be used as an index organism along with Enterococci (a fecal streptococci bacteria), because their presence often occurs with *Vibrio cholerae*, *Salmonella*, *Cryptosporidium parvum*, and other water-borne bacteria shed into the environment along with excreta (EPA 2010; NRC 2004, Stephen 2008).

2. Developed Governing equation

$$K\frac{hA}{L}\frac{\partial c}{\partial t} = \Delta V\frac{\partial^2 c}{\partial z^2} + h_{(x)}\frac{\partial c}{\partial z} + \Delta\phi\frac{\partial^2 c}{\partial z^2} \quad \text{.....} \quad (1)$$

Nomenclature

h	=	Fluid flow at vertical level
K	=	Permeability
A	=	Cross sectional area
L	=	Length
T	=	Time
Q	=	Porosity
c	=	Concentration
V	=	Velocity
z	=	Depth
$h_{(x)}$	=	Fluid at short distance

$$K\frac{hA}{L}\frac{\partial c}{\partial t} = [\Delta V + \Delta\phi]\frac{\partial^2 c}{\partial z^2} + h\frac{\partial c}{\partial z} \quad \text{.....} \quad (2)$$

$$K\frac{hA}{L}\frac{\partial c}{\partial t} = [\Delta V + \Delta\phi]\frac{\partial^2 c}{\partial z} \quad \text{.....} \quad (3)$$

$$K\frac{hA}{L}\frac{\partial c_1}{\partial t} = h\frac{\partial c}{\partial z} \quad \text{.....} \quad (4)$$

$$[\Delta V + \Delta\phi]\frac{\partial^2 c_3}{\partial z^2} = -h\frac{\partial c_3}{\partial z} \quad \text{.....} \quad (5)$$

The solution is of the form $c = (t, z) = c_1(t, z) + c_2(t, z) + c_3(t, z)$

Let $c = T, Z$ ..... (6)

$$\frac{\partial c_1}{\partial t} = T^1 Z \quad \text{.....} \quad (7)$$

$$\frac{\partial c}{\partial z} = TZ^1 \quad \text{.....} \quad (8)$$

$$\frac{\partial^2 c}{\partial z^2} = TZ^{11} \quad \text{.....} \quad (9)$$

Consider (3)

$$K\frac{hA}{L}T^1 Z = \left[\Delta V + \Delta\phi\right]TZ^{11} = \beta^2 \quad \text{.....} \quad (10)$$

$$K\frac{hA}{L} = \beta^2 \quad \text{.....} \quad (11)$$

$$\int \frac{dT}{T} = \int \frac{\beta^2}{K\frac{hA}{L}} dt \quad \text{.....} \quad (12)$$

$$Ln\, T = \frac{\beta^2}{K\frac{hA}{L}} + c \quad \text{.....} \quad (13)$$

…….. …… (14)

$$\boxed{T = A\ell^{\frac{\beta}{K\frac{hA}{L}}t}}$$

Considering this expression again $\left[\Delta V + \Delta\phi\right] = \beta^2$

$$\left[\Delta V + \Delta\phi\right]Z^{11} = \beta^2 \quad \text{.....} \quad (15)$$

$$c = B\ell^{\frac{\beta^2}{\Delta V + \Delta\phi}Z} + D\ell^{\frac{\beta^2}{\Delta V + \Delta\phi}Z} \quad \text{.....} \quad (16)$$

Combine (14) and (16) gives

$$c_1(t, z) = \left(B\ell^{\frac{\beta}{\Delta V + \Delta\phi}Z} + D\ell^{\frac{\beta}{\Delta V + \Delta\phi}Z}\right)A\ell^{\frac{\beta^2}{K\frac{hA}{L}}t} \quad \text{.....} \quad (17)$$

Consider equation (4)

$$K\frac{hA}{L}\frac{\partial c_2}{\partial t} = h\frac{\partial c_2}{\partial z}$$

$$K\frac{hA}{L}T^1Z = hZ^1T$$

$$K\frac{hA}{L}\frac{T^1}{T} = h\frac{Z^1}{Z} = \gamma \quad \text{.....} \quad (18)$$

$$h\frac{Z^1}{Z} = \gamma \quad \text{.....} \quad (19)$$

$$\int\frac{dT}{T} = \frac{\gamma}{K\frac{hA}{L}}\int dt \quad \text{.....} \quad (20)$$

$$Ln\,T = \frac{\gamma}{K\frac{hA}{L}} + \varphi \quad \text{.....} \quad (21)$$

$$T = C\ell^{\frac{\gamma}{K\frac{hA}{L}}t} \quad \text{.....} \quad (22)$$

Considering $h\frac{Z^1}{Z} = \gamma$

$$\int\frac{dz}{z} = \int\gamma v\,dz \quad \text{.....} \quad (23)$$

$$Ln\,z = \gamma hz + b \quad \text{.....} \quad (24)$$

$$z = \Delta\ell^{\gamma h} \quad \text{.....} \quad (25)$$

Combine (22) and (25), gives;

$$c_2 = (t,z) = ab\ell^{\left(\frac{1}{K\frac{hA}{L}}+h\right)t} \quad \text{.....} \quad (26)$$

Consider equation (5)

$$\left[\Delta V + \Delta\phi\right]Z^{11}T = -hZ^1T$$

$$[\Delta V + \Delta\phi]\frac{Z^{11}}{Z} = -h\frac{dz}{dz} = \theta^2 \quad \text{.....} \quad (27)$$

$$[\Delta V + \Delta\phi]\frac{d^2z}{dz^2} = \theta^2 \quad \text{.....} \quad (28)$$

$$Z = E\,Cos\frac{\theta}{\sqrt{\Delta V + \Delta\phi}}z + F\,Sin\frac{\theta}{\sqrt{\Delta V + \Delta\phi}}z \quad \text{.....} \quad (29)$$

Also $h\frac{dz}{dz} = +\theta^2$

$$\int\frac{dz}{dz} = h\theta^2\int dz \quad \text{.....} \quad (30)$$

$$Ln\,z = h\theta^2 z + d \quad \text{.....} \quad (31)$$

$$z = D\ell^{h\theta^2} \quad \text{.....} \quad (32)$$

Combining (29) and (30) yield

$$c_3 = (t, z) = \left(E\,Cos\frac{\theta}{\sqrt{\Delta V + \Delta\phi}}z + F\,Sin\frac{\theta}{\sqrt{\Delta V + \Delta\phi}}z\right)G\ell^{h\theta^2} \quad \text{.....} \quad (33)$$

Therefore, combined equations (17), (26) and (33) give

$$c(t, z) = c_1(t, z) + c_2(t, z) + c_3(t, z)$$

$$c_1(t, z) = \left(B\ell^{\frac{\beta}{\Delta V + \Delta\phi}Z} + D\ell^{-\frac{\beta}{\Delta V + \Delta\phi}Z}\right)A\ell^{\frac{\theta^2}{K\frac{hA}{L}}t} +$$

$$ab\ell^{\left(\frac{1}{K\frac{hA}{L}}+h\right)}\gamma + \left(E\,Cos\frac{\theta}{\sqrt{\Delta V + \Delta\phi}}z + F\,Sin\frac{\theta}{\sqrt{\Delta V + \Delta\phi}}\right)G\ell^{h\theta^2}z \quad \text{.....} \quad (34)$$

3. Materials and method

Standard laboratory experiment where performed to monitor the rate of pathogen concentration using column experiment at different formation, the soil deposition of the strata were collected in sequences base on the structural deposition at different locations, this samples collected at different location generate variation at different depth

producing different migration of pathogen concentration through pressure flow at different strata, the experimental result are applied to compared with the theoretical values to determined the validation of the model.

4. Results and Discussion

Results and discussion are presented in tables including graphical representation of pathogen concentration bellow:

Table: 1 Theoretical values of Pathogen concentration at Different Depths

Depth [M]	Concentration
3	1.97E-12
6	3.74E-12
9	5.92E-12
12	7.89E-12
15	9.87E-12
18	1.18E-11
21	1.37E-11
24	1.57E-11
27	1.77E-11
30	1.96E-11
33	2.16E-11
36	2.36E-11

Table: 2 Theoretical values of Pathogen concentration at Different Time

Time per Day	Concentration
10	1.97E-12
20	3.74E-12
30	5.92E-12
40	7.89E-12
50	9.87E-12
60	1.18E-11
70	1.37E-11
80	1.57E-11
90	1.77E-11
100	1.96E-11
110	2.16E-11
120	2.36E-11

Table 3: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

Depth [M]	Theoretical Values Conc. mg/l	Experimental values mg/l
3	1.97E-12	1.87E-12

6	**3.74E-12**	**3.55E-12**
9	**5.92E-12**	**6.11E-12**
12	**7.89E-12**	**7.56E-12**
15	**9.87E-12**	**9.66E-12**
18	**1.18E-11**	**1.09E-11**
21	**1.37E-11**	**1.24E-11**
24	**1.57E-11**	**1.45E-11**
27	**1.77E-11**	**1.66E-11**
30	**1.96E-11**	**1.90E-11**
33	**2.16E-11**	**2.09E-11**
36	**2.36E-11**	**2.26E-11**

Table 4: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

Time per Day	Theoretical Values Conc. mg/l	Experimental values mg/l
10	**1.97E-12**	**1.67E-12**
20	**3.74E-12**	**3.66E-12**
30	**5.92E-12**	**5.88E-12**
40	**7.89E-12**	**7.77E-12**
50	**9.87E-12**	**9.77E-12**
60	**1.18E-11**	**1.11E-11**
70	**1.37E-11**	**1.31E-11**
80	**1.57E-11**	**1.51E-11**
90	**1.77E-11**	**1.72E-11**
100	**1.96E-11**	**1.91E-11**
110	**2.16E-11**	**2.09E-11**
120	**2.36E-11**	**2.24E-11**

Table: 5 Theoretical values of Pathogen concentration at Different Time

Time Per Day	Concentration
4	**1.31E-12**
8	**2.62E-12**
12	**3.93E-12**
16	**5.25E-12**
20	**6.55E-12**
24	**7.87E-12**
28	**9.19E-12**
32	**1.05E-11**
36	**1.18E-11**
40	**1.31E-11**

44	**1.44E-11**
48	**1.57E-11**
52	**1.70E-11**
56	**1.96E-11**
60	**2.36E-11**

Table: 6 Theoretical values of Pathogen concentration at Different Depths

Depth [M]	Concentration
2	**1.31E-12**
4	**2.62E-12**
6	**3.93E-12**
8	**5.25E-12**
10	**6.55E-12**
12	**7.87E-12**
14	**9.19E-12**
16	**1.05E-11**
18	**1.18E-11**
20	**1.31E-11**
22	**1.44E-11**
24	**1.57E-11**
26	**1.70E-11**
28	**1.96E-11**
30	**2.36E-11**

Table 7: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

Depth [M]	Theoretical Values	Experimental Values [mg/l]
2	**1.31E-12**	**1.37E-12**
4	**2.62E-12**	**2.44E-12**
6	**3.93E-12**	**3.88E-12**
8	**5.25E-12**	**5.11E-12**
10	**6.55E-12**	**6.33E-12**
12	**7.87E-12**	**7.55E-12**
14	**9.19E-12**	**9.22E-12**
16	**1.05E-11**	**1.09E-11**
18	**1.18E-11**	**1.18E-11**
20	**1.31E-11**	**1.24E-11**
22	**1.44E-11**	**1.34E-11**
24	**1.57E-11**	**1.48E-11**
26	**1.70E-11**	**1.64E-11**

28	**1.96E-11**	**1.87E-11**
30	**2.36E-11**	**2.22E-11**

Table 8: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

Time Per Day	Theoretical values	Experimental Values
4	**1.31E-12**	**1.34E-12**
8	**2.62E-12**	**2.55E-12**
12	**3.93E-12**	**3.88E-12**
16	**5.25E-12**	**5.31E-12**
20	**6.55E-12**	**6.41E-12**
24	**7.87E-12**	**7.74E-12**
28	**9.19E-12**	**9.22E-12**
32	**1.05E-11**	**1.10E-11**
36	**1.18E-11**	**1.16E-11**
40	**1.31E-11**	**1.29E-11**
44	**1.44E-11**	**1.38E-11**
48	**1.57E-11**	**1.45E-11**
52	**1.70E-11**	**1.64E-11**
56	**1.96E-11**	**1.88E-11**
60	**2.36E-11**	**2.21E-11**

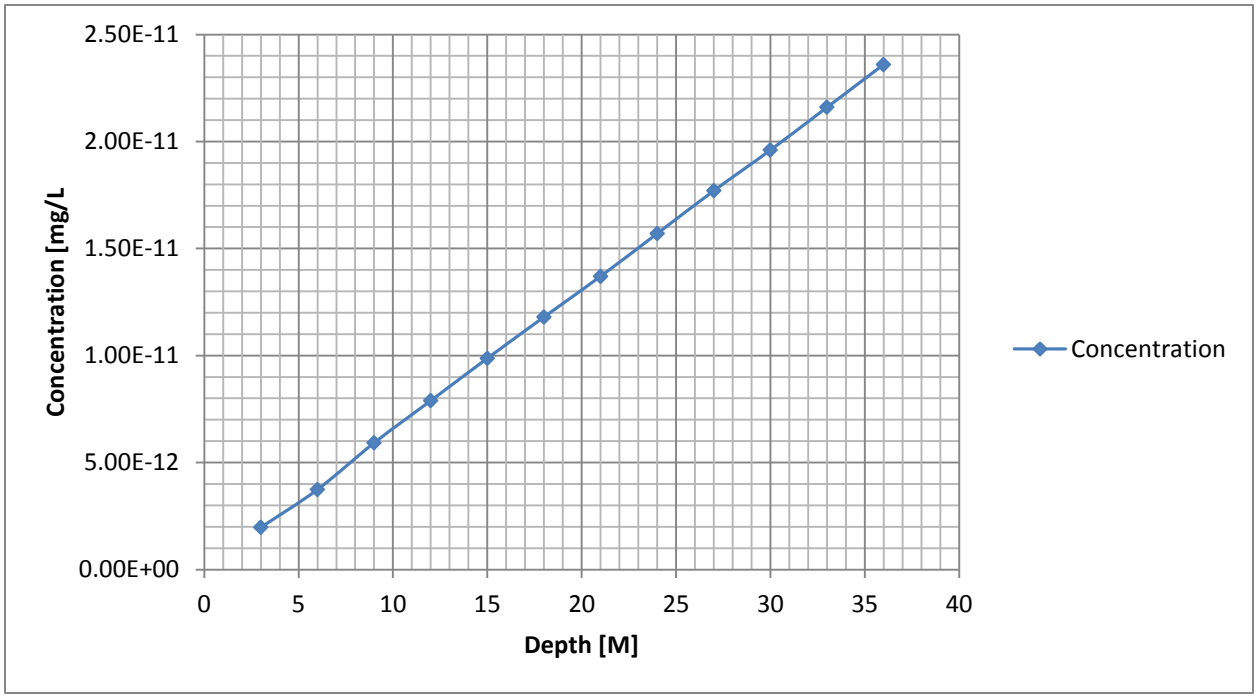

Figure: 1 Theoretical values of Pathogen concentration at Different Depths

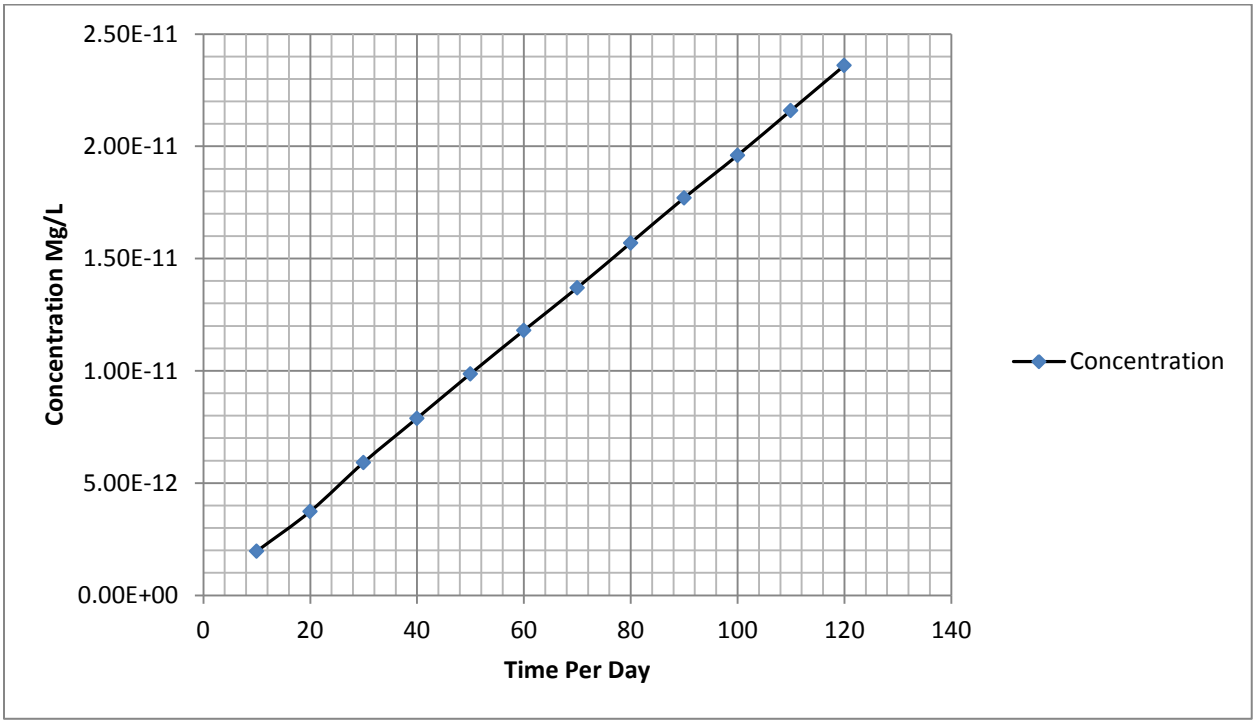

Figure: 2 Theoretical values of Pathogen concentration at Different Time

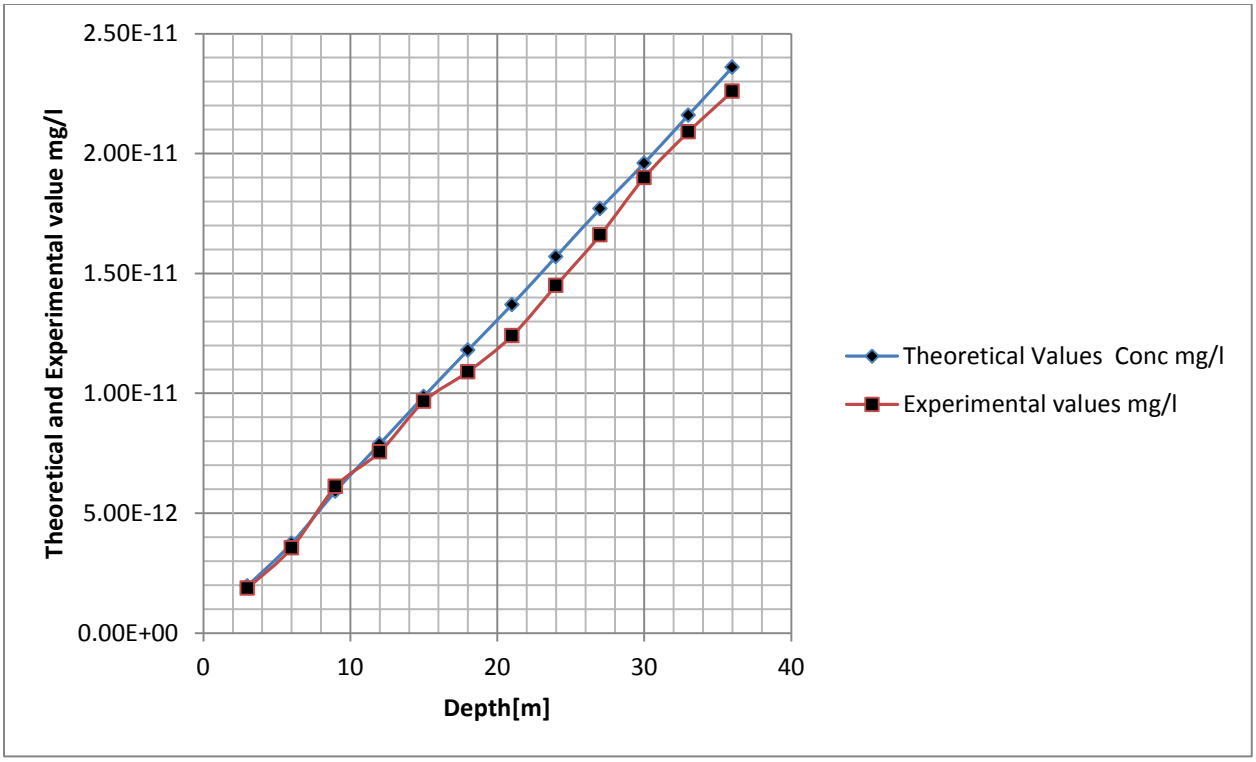

Figure 3: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

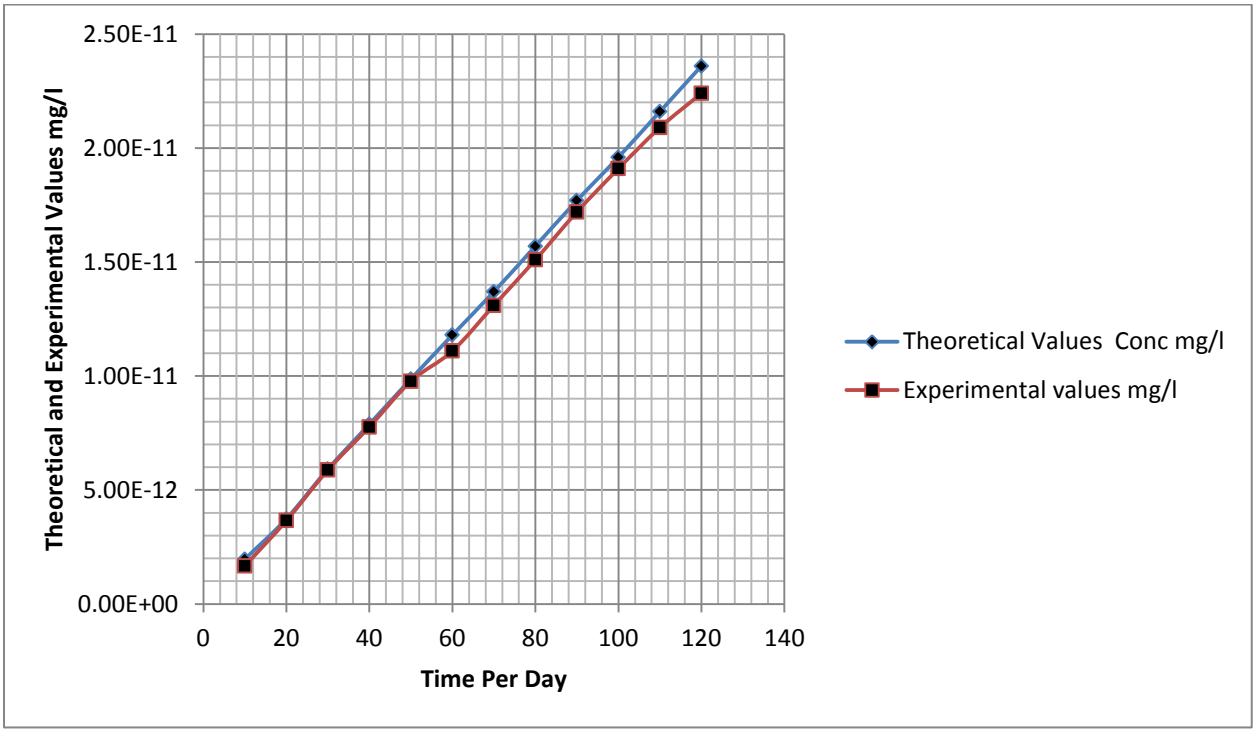

Figure 4: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

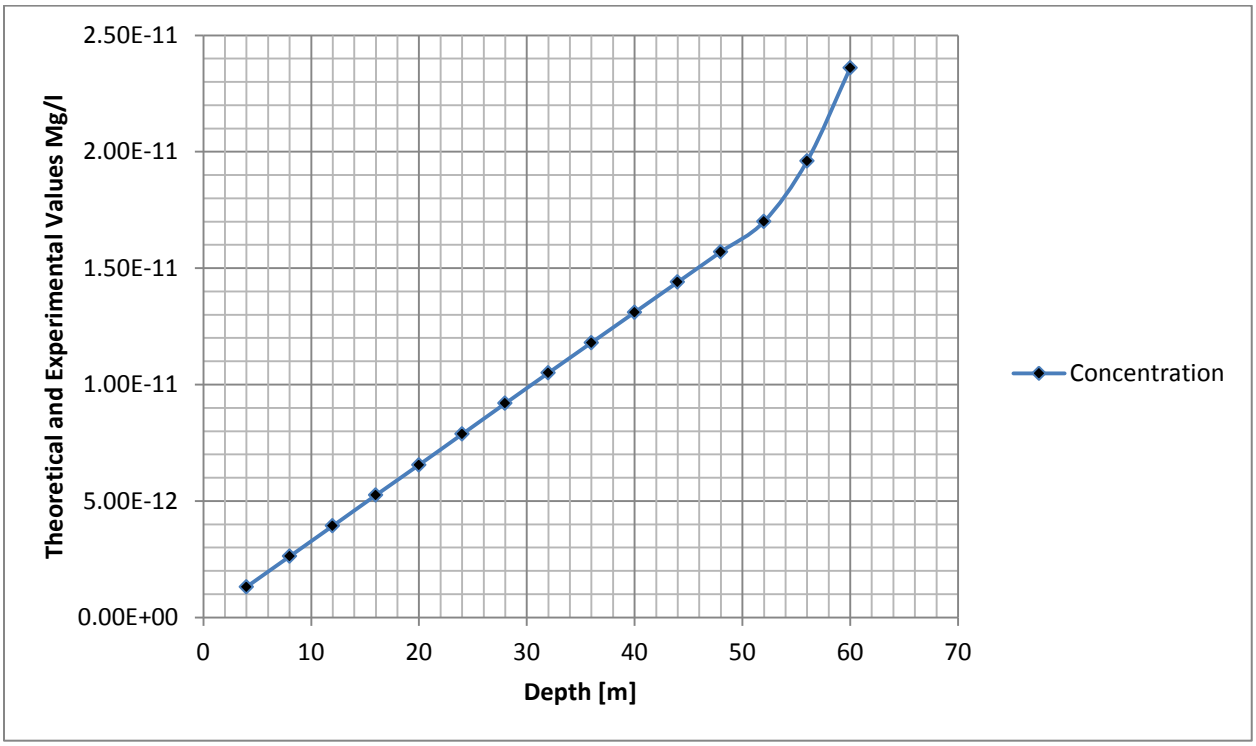

Figure: 5 Theoretical values of Pathogen concentration at Different Depths

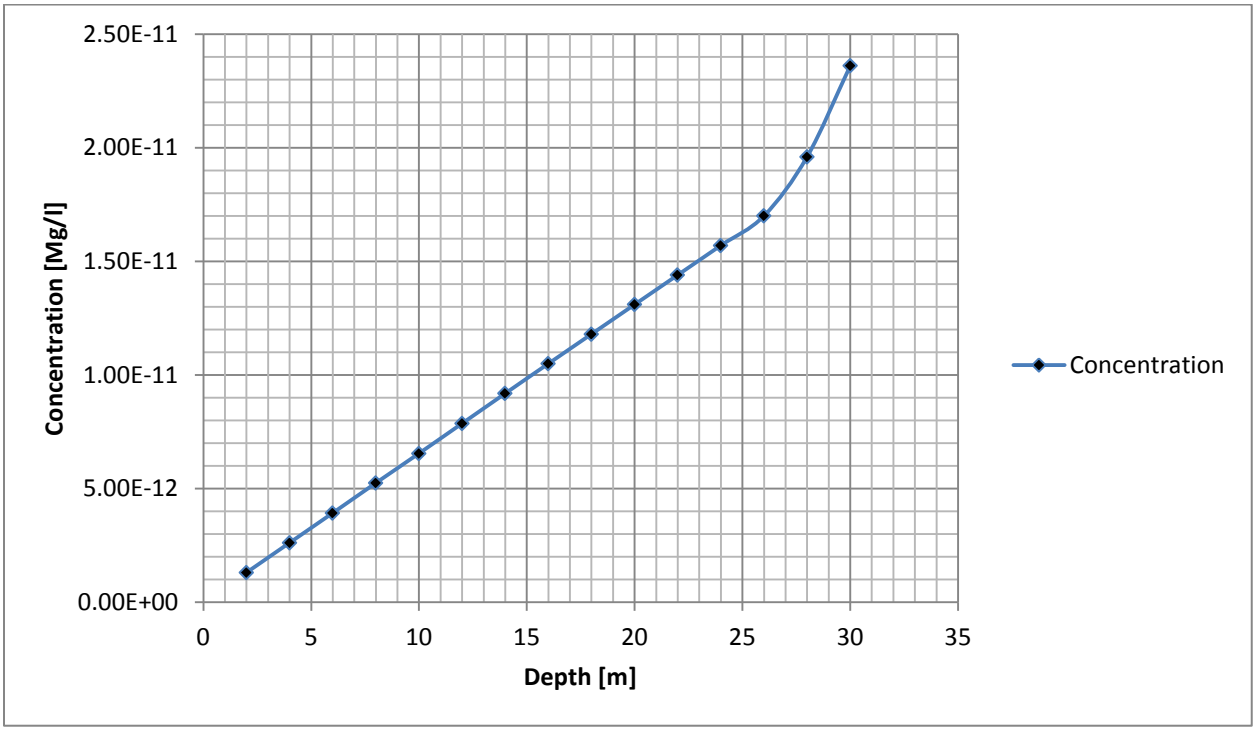

Figure: 6 Theoretical values of Pathogen concentration at Different Depths

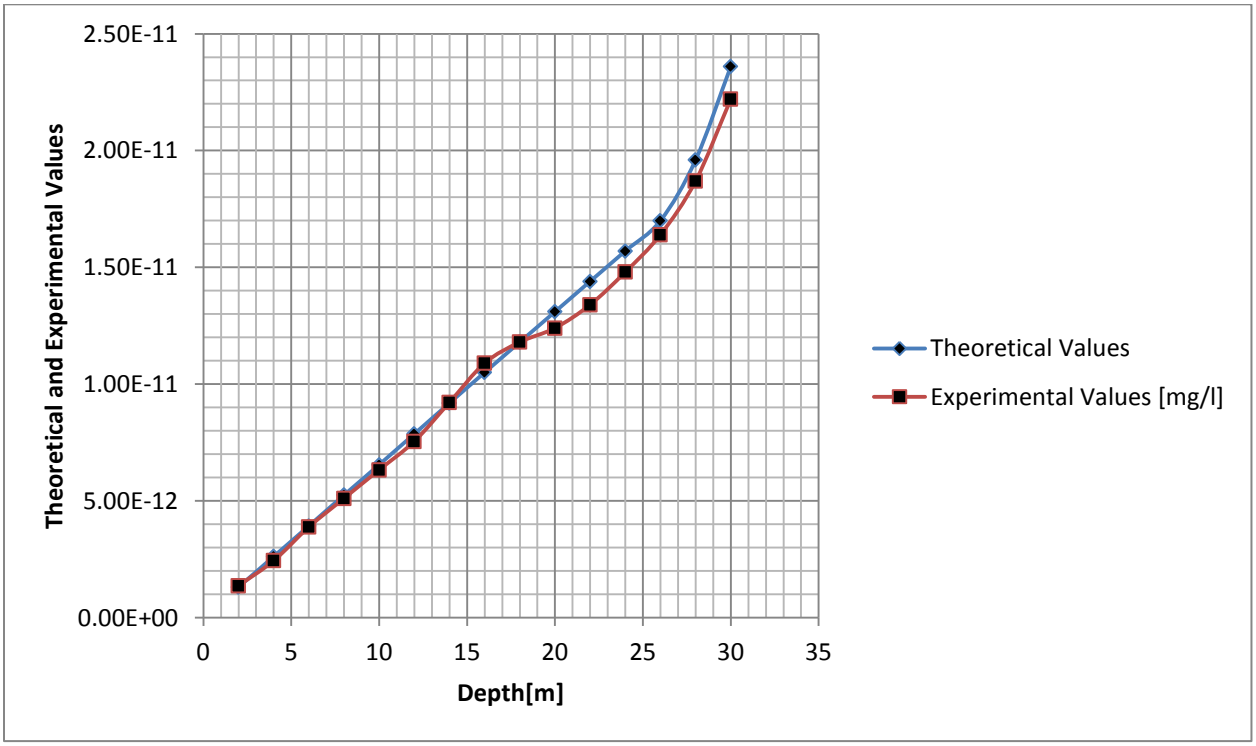

Figure 7: Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

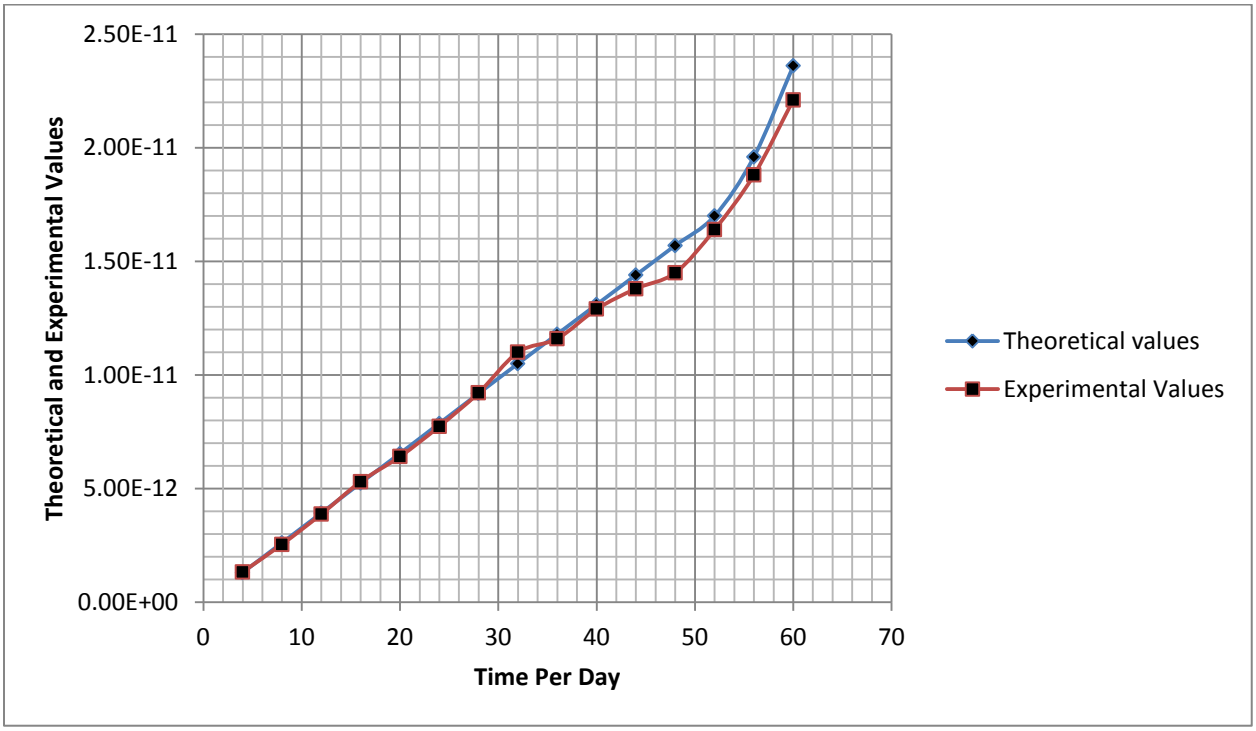

Figure 8 Comparison of Theoretical and Measured Values of pathogen Concentration Different Depth

Figure one to four express the behaviour of pathogen concentration to be in linear direction under exponential phase in the transport system, the deposition of pathogen in those locations express homogeneous stratification of the formation. The rate of migration from the theoretical values shows that the partial deposition may have been hinder by low permeability in lateritic soil formation, but due to constant increase of saturation from high rain intensities the concentrations find it way to the silty formation and migrate partially to unconfined bed. While four and five to eight express similar condition as linear direction predominantly influences the transport system, but experiences slight fluctuation from twenty four to thirty metre at the duration of fifty to sixty day respectively, partial deposition of pathogen in linear direction on migration were able to establish uniformity deposition on pore distribution of the grain size generating homogeneous void ratio. This condition may have influences the velocity experiencing linear direction on the migration of pathogen, the express figure from the simulation generated values that have been compared with experimental values, both parameters developed a best fit validating the developed model for the study.

4. Conclusion

Partial deposition of pathogen were subjected to thorough investigation, the study were able generated better results that should assure that its deposition partially will be harmless to human through consumption ground water in the study area, the deposition of pathogen in lateritic and silty formation establish fluctuation on the permeation and pore distribution of grain size sediments, these resulted to heterogeneous void ratio and permeability, lateritic formation were observed to have slightly accumulate pathogen concentration base on its low void ratio and

permeability, but due to constant high rain intensities in the area, pathogen migrated slightly to silty formation were higher void ratio and permeability were deposited. The velocity at those formation pressure the concentration migrating to unconfined bed, if the deposited formation establish heterogeneity, it will definitely result to increase in concentration through an accumulation of the pathogen in those formation that may experience low void ratio and permeability. Continuity of the deposition in heterogeneity will definitely be in long term generates accumulation that has increase the concentration which will become severely harmful to human consuming ground water through long time effect.

References

[1] Ashbolt NJ. 2004. Microbial contamination of drinking water and disease outcomes in developing regions. Toxicology 198:229-238.

[2] Ashbolt NJ, Grabow WOK, and Snozzi M. 2001. Indicators of microbial water quality. World Health Organization

[3] EPA. 2009. Drinking Water Pathogens and their Indicators: A Reference Resource. U.S. Environmental Protection Agency.

[4] EPA. 2010. Monitoring and assessment: fecal bacteria. Environmental Protection Agency.

[5]NRC. 2004. Indicators for waterborne pathogens. Washington DC: The National Academies Press
.
[6] UN. 2010. The Millennium Development Goals Report 2010. New York: The United Nations.

[7] UN. 2011. Millennium Development Goals. Geneva: The United Nations.

[8] UNICEF, and WHO. 2000. Global Water Supply and Sanitation Assessment 2000 Report. In: Joint MonitoringProgramme for Water Supply and Sanitation WHO, editor.

[9] UNICEF, and WHO. 2009. Diarrhea: Why children are still dying and what can be done.

[10] UNICEF, and WHO. 2010a. Estimates for the use of Improved Drinking water Sources. In: Sanitation JMPfWSa, editor.

[11] UNICEF, and WHO. 2010b. Estimates for the use of Improved Sanitation Facilities: Mexico. In: Sanitation JMPfWSa, editor

[12] UNICEF, and WHO. 2010c. Progress on Sanitation and Drinking Water: 2010 Update. WB. 2007. Mexico 2006-2012: Creating the foundations for equitable growth. Washington DC: The World Bank.

[13] WHO. 2004. Access to improved drinking water and to improved sanitation: indicator definitions and metadata. WHO Statistical Information System (WHOSIS

[14] Stephen Crabbe 2008 An Analysis of Water Quality in Small Water Treatment Plants and Households in the Yucatán Peninsula, Mexico

[15]Ebedes, H. 1976. Anthrax epizootics in wildlife in the Etosha National Park, South West Africa, p. 19-526. *In* L. A. Page (ed.), Wildlife Diseases. Plenum Press, New York.

[16]**de Vos, V.** The Ecology of Anthrax in the Kruger National Park, South Africa. Turnbull P.C.B. Salisbury Medical Bulletin 68 Special Supplement, 19-24. 1990.Wiltshire England, Salisbury Medical Society. 4-11-1989

[17]Dragon, D. C. and R. P. Rennie. 1995. The ecology of anthrax spores: tough but not invincible. Can.Vet.J. **36**:295-301.

[18] Turnbull P.C.B. 1996. Stubborn contamination with anthrax spores. Environmental Health 106:171-173

[19] Pamala R. 2002 Coker bacillus anthracis spore concentrations at various carcass sites Dissertation Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy

[20] Eluozo. S.N 2013 modeling exponential phase migration of Edwardsiella in homogeneous silty and fine sand formation aquifers in coastal area of Buguma, rivers state of Nigeria World Journal of Science and Technology Research Vol. 1, No. 2, April 2013, PP: 23 – 33

[21] Eluozo, S. N and Afiibor, B.B **2013;** carbon and salmonellae model transport on vertical and horizontal deposited gravel formation in coastal area of Borikiri of port Harcourt metropolis, Niger delta of Nigeria Open Journal of Environmental Science and Development Vol. 1, No. 1, November 2013, PP: 20 -29