



Modeling of pheratic aquifers on E, coli transport influenced by preconsolidation and compressibility of soil

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Abstract

The rates of pollution on soil are the major problem of ground water contamination in the study area. A study to determine the influence from preconsolidation and compressibility on E.coli transport has been carried out. This is to determine the rate of influence on E.coli transport in pheratic aquifers, mathematical model were developed to monitor the influence on the preconsolidation and compressibility of soil on microbial transports, experimental analysis were also carried out through a standard column experimental analysis, ten samples were collected in a drilling site at interval of three metres each for several location, the effluent discharge from the lower end of the column were collected and subjected to a thorough analysis, the theoretical values were compared with experimental values, and both values compared favourably well. This study has explained the transport of E.coli base on the level of concentration generate from aquiferous zone under the influence of preconsolidation and soil compressibility. The study has reveal the variation of concentration of E.coli on the influence of soil preconsolidation and it compressibility, therefore exploration of ground water should be design with its criteria's to prevent the wild spread of water pollution emanating from the E.coli transport in the study areas, because the preconsolidation and compressibility of soil were found to be insignificant on the transport of the microbes, the preconsolidation and its rate of compressibility could not reduce the transport of E.coli on a natural state.

Keywords: modeling, E.coli Transport, preconsolidation and compressibility of soil.

1 Introduction

Testing for bacteria” is the most frequent superiority check for well water. “No bacteria” is the favorite result. There are many diverse types of bacteria that might occur in your well water. This research gives some background studies about these minute single-cell organisms. (Just one, is a bacterium, but they are usually referred to in the plural, bacteria). Most are nontoxic to humans and many are essentially beneficial for our existence. Microfossils in ancient rocks show bacteria that were around 3.2 billion years ago. Bacteria were doubtless accountable for creating the first oxygen that appeared on Earth, about 2 billion years ago. Some bacteria develop and increase by using energy (food) obtained from minerals in ground water such as sulfur (“sulfur bacteria”) or iron (“iron bacteria”). Some bacteria thrive in oxygen rich environments (aerobic) and others in oxygen short (anaerobic) conditions. They have three basic shapes spherical (coccus), rod-like (bacillus) and curved (vibrio, spirillum, spirochete). Most bacteria are extremely little (about 1 micron long). If they were lined up side by side, 25,000 bacteria would take up about an inch. a good number bacteria replicate by splitting into two. If situation are suitable, bacteria can reproduce very quickly, completing one division every 20 to 30 minutes. Bacteria are very elastic, remaining inactive when conditions are not ideal. Dried, but living bacteria can even be carried in the air. Bacteria can excrete toxins or carry them inside their cell wall until they die and disintegrate. Some bacteria may invade a specific organ of the body, for example the brain, throat or bone. Bacteria may also produce enzymes, some of which are responsible for illness. Coliform bacteria are the bacteria most commonly associated with well water. The United States Environmental Protection Agency (EPA) standard for drinking water is a total coliform count of zero. While in developing nation like Nigeria more precisely Rivers State are having a lots of pollution from microbes. Coliform bacteria are a large group of various rod-shaped species and strains of bacteria. The group includes bacteria that occur naturally in the intestines of warm-blooded animals (fecal coliform) and no fecal coliform. Non-fecal coliform bacteria are extremely familiar and are found virtually everywhere on soil particles, insects, plants, animals, walls and furniture in homes and on your skin and clothes. Fecal coliform can include disease causing (pathogen species) and non-disease causing species. Over 200 types of non-disease causing bacteria have been

found in human digestive tracts. Most arrive on the food and drink we consume. Many yogurt cultures include coliform bacteria. *Lactobacillus acidophilus* is the most common bacteria strain used in commercial yogurts and some studies show it creates an acidic environment that inhibits harmful bacteria in the digestive tract. *Escherichia coli* (E. coli), often listed in water quality analyses, is one species of fecal coliform bacteria. A single E. coli is 2 microns long and about 0.5 microns in diameter. There are hundreds strains of E. coli bacteria that differ only in the type of toxin or enzyme that they produce. Despite the fact that they originate in the digestive system of a warm-blooded creature, most E. coli strains are not harmful to humans E. coli can be easily cultured in a laboratory and therefore, they are a good indicator species for bacterial contamination in water tests. Its presence in a water sample indicates that sewage material may be present and that if sewage is present, more harmful disease-causing organisms may also be present, for example *Vibrio cholerae* that causes cholera. (AMERICAN WELL OWNER, 2002). Since E. coli is found in human and animal waste, it is often used as an indicator to detect the presence of these wastes in the water. Many different laboratory methods exist to detect and count E. coli Most of them are based on collecting a sample of water, and passing it through a 0.45µm membrane filter to capture any bacteria in the water. The filters are then placed on selective growth media, which are incubated for about 24 hours to produce colored colonies. The colonies can then are counted by hand using a filter grid or by using a routine automated colony counter. To isolate E. coli O157:H7 from human or animal feces, which usually contains many other strains of E. coli, distinct characteristics of E. coli O157:H7 are considered. It does not break down certain sugars as rapidly as 95% of other E-coli strains. Therefore, when using the sugar as a growth medium, E. coli [1, 2, 3].

Five classes of Diarrheagenic *Escherichia coli* (DEC) have been described: enteropathogenic (EPEC), enterotoxigenic (ETEC), enteroinvasive (EIEC), Shiga-toxin-producing (STEC), and enteroaggregative (EAEC). Diffuse adhering *E. coli* (DAEC) may represent a sixth category, but this has not been clearly Diarrheagenic pathotype could only be identified on the basis of O:H serotyping. In the past 20 years, however, the tools for identifying EPEC have been refined as the molecular basis of EPEC pathogenesis has begun to be elucidated and specific virulence genes have been discovered [8 10, 20]. The central mechanism of EPEC pathogenesis is a lesion called attaching and effacing (A/E), which is characterized by microvilli destruction, intimate adherence of bacteria to the intestinal epithelium, pedestal formation, and aggregation of polarized actin and other elements of the cytoskeleton at sites of bacterial attachment. The ability to induce A/E lesions is encoded by genes located on a 35-kb pathogen city island called the locus of enterocyte effacement (LEE), which contains the genes encoding intimin, a type III secretion system, secreted proteins (Eps), and the translocated intimin receptor (Tir). Homologues of LEE are also found in STEC and in animal A/E *E. coli* strains [5, 6, 11, 14, 15, 16, 17, 18]. Intimin, a 94-kDa outer-membrane protein encoded by the gene *eae*, is responsible for the intimate adherence of bacteria to enterocyte membranes [11]. The intimin protein is highly variable between different EPEC and STEC serotypes and at least five distinct antigenic variants have been identified [4]. Differentiation of intimin alleles represents an important tool for STEC and EPEC typing in routine diagnostics as well as in pathogenesis, epidemiological, clonal, and immunological studies. The C-terminal end of intimin is responsible for receptor binding, and it has been suggested that different intimin may be responsible for different host-tissue cell tropism. The 5' regions of *eae* genes are conserved, whereas the 3' regions are heterogeneous. This observation led to the construction of universal PCR primers and allele-specific PCR primers, which has made it possible to differentiate between 21 variants of the *eae* gene that encode 21 different intimin. [4, 6, 7, 8 11,12,15, 21].

2 Materials and method

Column experiments were also performed using soil samples from (7) different borehole locations, the soil samples were collected at intervals of three metres each (3m). An E.coli solute was introduced at the top of the column and effluents from the lower end of the column were collected and analyzed for E.coli, and the effluent from the lower end of the column were collected at different days for analysis, velocity of the transport were monitored at different days. Finally, the results were collected to be compared with the theoretical values, for model validation.

Developed mathematical model

$$TC(x) \frac{\partial V(x)}{\partial t} = \frac{U \partial C(x)}{\partial t} \quad (1)$$

$$\frac{U \partial C(x)}{\partial t} = -TC(x) \frac{\partial V(x)}{\partial t} \quad (2)$$

$$\frac{U \partial C(x)}{\partial t} = -TC(x) \frac{Vt}{\partial t} \quad (3)$$

$$\left(\frac{U}{U(x)}\right) \frac{\partial C(x)}{\partial(x)} = -\frac{T \partial t}{t} \tag{4}$$

$$\frac{U}{U} = \int \frac{1}{C(x)} \partial C(x) = -T \int \frac{\partial t}{\partial t} \tag{5}$$

$$\frac{U}{U(x)} \left[\text{Ln } C(x) = -\text{Ln } \frac{t}{t_0} \right] \tag{6}$$

$$\text{Ln } \frac{C(x)}{C(x)_0} = \left(\frac{t}{t_0}\right) - \frac{TU(x)}{U} \tag{7}$$

$$\frac{C(x)}{C(x)_0} = \left(\frac{t}{t_0}\right) - \frac{TU(x)}{U} \tag{8}$$

$$\frac{C(x)}{C(x)_0} = \ell^{-T \text{Ln} \left(\frac{t}{t_0}\right) \frac{Ux}{U}} \tag{9}$$

$$C(x) = C(x)_0 \ell^{-T \text{Ln} \frac{Ux}{t_0}} \tag{10}$$

$$C(x) = \beta \ell^{-T \text{Ln} \frac{1}{t_0}} \tag{11}$$

Where

$$\boxed{\beta = C(x)_0 \ell^{\frac{Ux}{U}}} \tag{12}$$

$$\beta = C(x)_0 \ell^{\frac{Ux}{U}} \cdot C_1 \cdot \frac{e_1 - e_2}{\text{Log } \frac{\partial_2}{\partial_1}} \tag{13}$$

$$\text{Let } A = C_1 \cdot \frac{e_1 - e_2}{\text{Log } \frac{\partial_2}{\partial_1}} \tag{14}$$

$$\Rightarrow \beta = AC(x)_0 \ell^{\frac{Ux}{U}} \tag{15a}$$

$$C(x) = \frac{\beta}{A} \ell^{-\frac{Ux}{U}} \tag{15b}$$

$$C(x) = \frac{\beta}{A} = \beta \left(\frac{1}{A}\right) \tag{16}$$

$$C(x) = C_0 \ell^{\frac{Ux}{U}} \tag{17}$$

Where C_0 is preconsolidation compressibility of soil and concentration of the microbes is Take Laplace of equation (17).

$$C(x) = \beta \ell^{\frac{Ux}{U}} \quad (18)$$

$$C(o) = \beta \ell^{\frac{Ux}{U}} \quad (19)$$

i.e. $\frac{\beta}{U+S}$ (20)

$$C(o) = [U+S] = \beta \quad (21)$$

i. e. $C(o) = U + C(o) S - \beta = 0$ (22)

Applying quadratic formula (20) we have expression of this form.

$$C(x) = \frac{-S \pm \sqrt{S^2 U^2 + \beta U}}{2U} \quad (23)$$

Now $S = U$ that equation (22) we have expression of this form

$$C(x) = \frac{-U \pm \sqrt{U^2 + 4\beta U}}{2U} \quad (24)$$

The general solution of equation (23) is

$$C(x) = A \exp\left[\frac{-U \pm \sqrt{U^2 + 4\beta U}}{2U} t\right] + \beta \exp\left[\frac{-U \pm \sqrt{U^2 + 4\beta U}}{2U} t\right] \quad (25)$$

Subjecting equation (24) to the following boundary condition values.

$$X = 0 \quad C(o) = 0 \quad \text{and} \quad t = 0$$

We have $\beta = -1$ and $A = 1$

So that particular solution

$$C(x) = \exp\left[-\frac{U + (U^2 + 4\beta U)^{1/2}}{2} t\right] - \exp\left[-\frac{U - (U^2 + 4\beta U)^{1/2}}{2} t\right] \quad (26)$$

But $e^x e^{-x} = \sin$

Therefore, the expression (25) can be expressed in this form

$$\boxed{C(x) = 2 \sin\left[U + (U^2 + 4\beta U)^{1/2}\right] t} \quad (27)$$

Coefficient of consolidation

3 Results and discussion

Table 1: Theoretical values and figures are presented bellow at various depths

Depth	Theoretical values Conc at constant velocity
3	0.13
6	0.26
9	0.39
12	0.52
15	0.62
18	0.78
21	0.9
24	1.02
27	1.12
30	1.22

Table 2: Theoretical values and figures are presented bellow at various depths

Depth	Theoretical values Conc at Different velocity
3	0.03
6	0.05
9	0.02
12	-1.69
15	-0.95
18	-1.62
21	-8.00E-03
24	1.02
27	0.78
30	0.86

Table 3: Theoretical values and figures are presented bellow at various depths

Depth	Theoretical values Conc at Different velocity
3	0.09
6	0.06
9	0.02
12	0.04
15	-1.92
18	1.23
21	1.76E+00
24	1.02
27	0.84
30	0.86

Table 4: comparison of theoretical and experimental values at various depths

Depth	Theoretical values Conc at constant velocity	Experimental Values
3	0.13	0.15
6	0.26	0.23
9	0.39	0.41
12	0.52	0.53
15	0.62	0.64
18	0.78	0.77
21	0.9	0.89
24	1.02	1.04
27	1.12	1.14
30	1.22	1.23

Table 5: comparison of theoretical and experimental values at various period

Time	Theoretical values Conc at constant velocity	Experimental Values
10	0.13	0.15
20	0.26	0.23
30	0.39	0.41
40	0.52	0.53
50	0.62	0.64
60	0.78	0.77
70	0.9	0.89
80	1.02	1.04
90	1.12	1.14
100	1.22	1.23

Table 6: comparison of theoretical and experimental values at various depths

Depth	Theoretical values Conc at various velocity	Experimental Values
3	0.03	0.04
6	0.05	0.03
9	0.02	0.05
12	-1.69	-1.65
15	-0.95	-0.97
18	-1.62	-1.65
21	-8.00E-03	-7.78E-03
24	1.02	1.05
27	0.78	0.81
30	0.86	0.84

Table 7: comparison of theoretical and experimental values at various period

Time	Theoretical values Conc at Constant velocity	Experimental Values
10	0.03	0.04
20	0.05	0.03
30	0.02	0.05
40	-1.69	-1.65
50	-0.95	-0.97
60	-1.62	-1.65
70	-8.00E-03	-7.78E-03
80	1.02	1.05
90	0.78	0.81
100	0.86	0.84

Table 8: comparison of theoretical and experimental values at various depths

Depth	Theoretical values Conc at Constant velocity	Experimental Values
3	0.09	0.07
6	0.06	0.05
9	0.02	0.05
12	0.04	0.06
15	-1.92	-1.89
18	1.23	1.21
21	1.76E+00	1.74E+00
24	1.02	1.06
27	0.84	0.86
30	0.86	0.83

Table 9: comparison of theoretical and experimental values at various period

Time	Theoretical values Conc at Constant velocity	Experimental Values
10	0.09	0.07
20	0.06	0.05
30	0.02	0.05
40	0.04	0.06
50	-1.92	-1.89
60	1.23	1.21
70	1.76E+00	1.74E+00
80	1.02	1.06
90	0.84	0.86
100	0.86	0.83

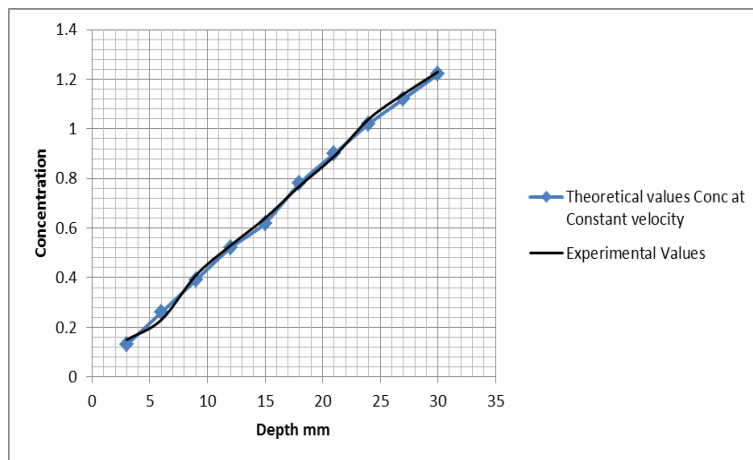


Figure 1: comparison of theoretical and experimental values at various depths

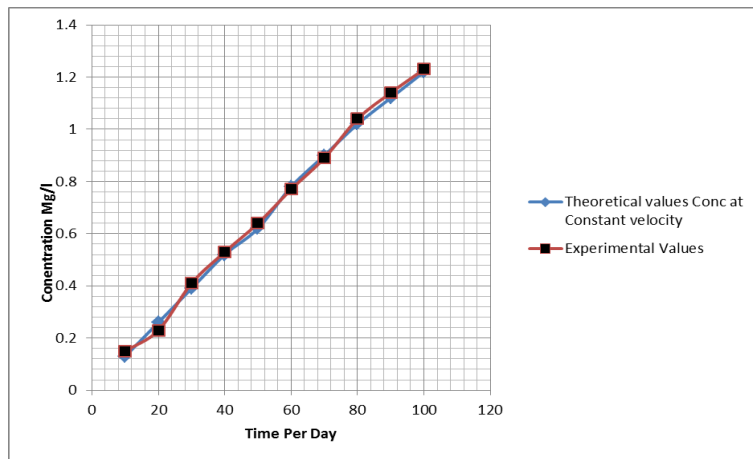


Figure 2: comparison of theoretical and experimental values at various period

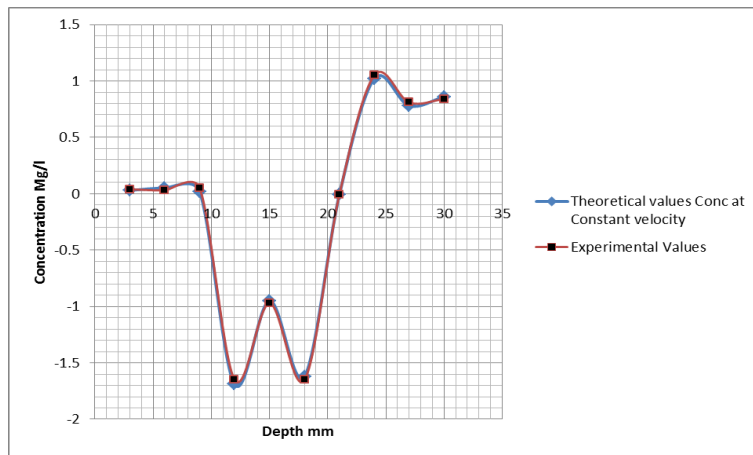


Figure 3: comparison of theoretical and experimental values at various depths

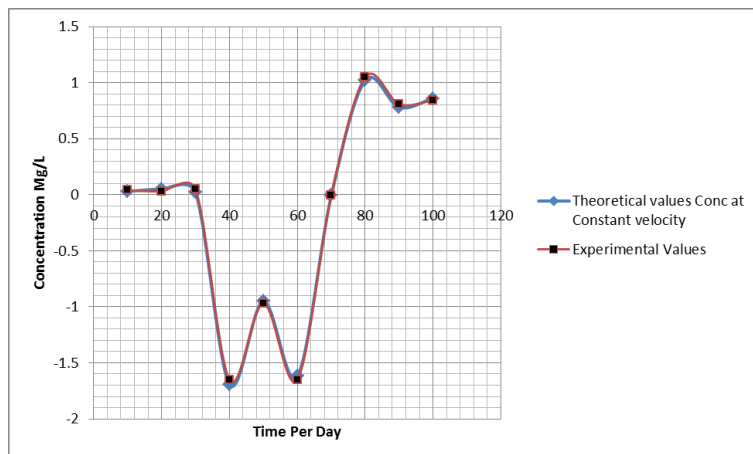


Figure 4: comparison of theoretical and experimental values at various period

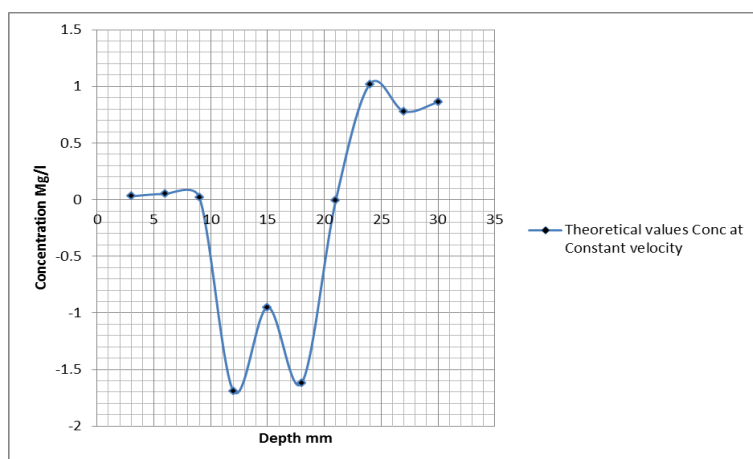


Figure 5: comparison of theoretical and experimental values at various depths

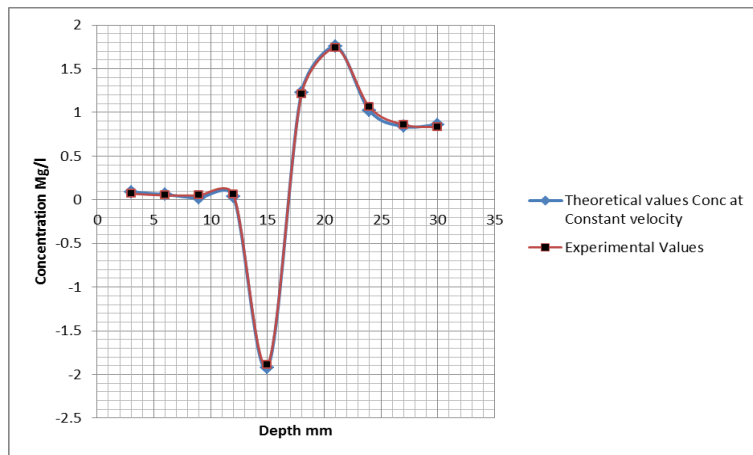


Figure 6: comparison of theoretical and experimental values at various depths

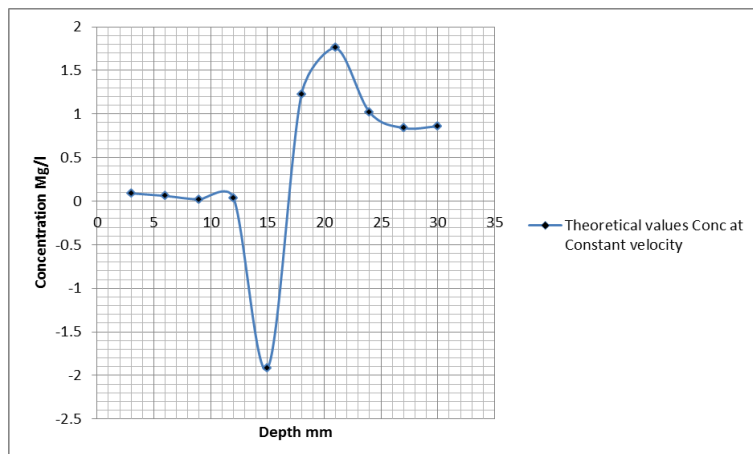


Figure 7: comparison of theoretical and experimental values at various depths

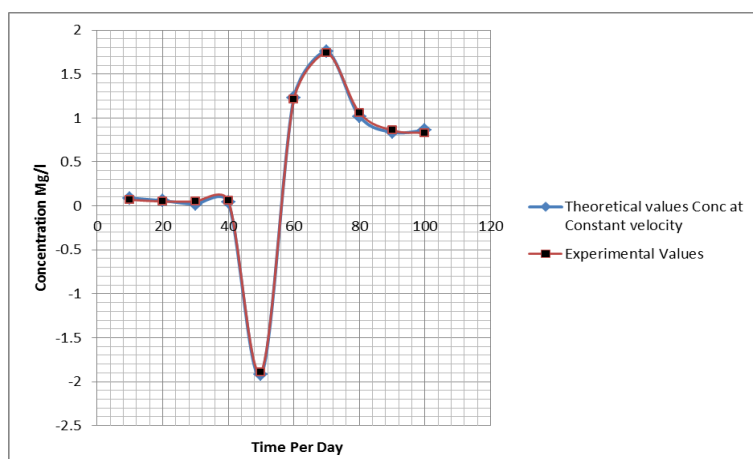


Figure 8: comparison of theoretical and experimental values at various depths

4 Results and discussion

Modeling of microbial process in soil and water on porous medium is essential, the biodegradation of contaminant on E.coli transport to ground water aquifers influenced by preconsolidation and compressibility of soil matrix; the study. were carried out through the development of mathematical model. The study is to determine the influence of

preconsolidation and the rate compressibility of soil, this were found not to decrease the microbial transport, in figure One and two the microbes gradually increase to a point where the optimum values were recorded at thirty metres at the period of hundred days, while figure three and four gradually increase between three to nine metres, suddenly the microbes were found to maintained a lag phase between twelve to twenty one metres at the period of forty to seventy days, and a rapid increase were experienced on the migration were substrate deposited at twenty four metres, it also experience its optimum values between twenty four to thirty metres at the period of eighty to hundred days. Figure five to eight were found to experiencing slight fluctuation between three to twelve metres, lag phase condition were also experienced at fifteen metres at the period of fifty days, unexpectedly, a rapid increase were experienced were the optimum increase were recorded at twenty one metres at the period of seventy days, it finally develop slight decrease between twenty four and thirty metres at the time of eighty to hundred days. The theoretical values compared favourably well with the experimental values, the results were found to develop fast migration of the microbes even with high level of preconsolidation and compressibility of the soil. Definitely it may be attributed to the level of soil structural deposition and other environmental influence, base on the condition of the study location (deltaic environment). therefore transport of E.coli were found to develop contaminant at aquiferous zone, this condition explain the compulsory remediation of the soil and regulate the waste dump to avoid regeneration of the microbes as the preconsolidation and compressibility level of the soil were found to be insignificant in reducing concentration of the microbes at the unconfined bed for human consumption.

5 Conclusion

The influence of preconsolidation and compressibility of soil on of E.coli transport in unconfined bed has shown insignificant effect, base on the level of microbial growth in pheratic aquifers. This condition can be attributed to the level of man made activities, including the soil structural deposition that could not develop any significant effect to reduce the increase of e.coli concentration in deltaic environment. The study is imperative because most people may conclude that the level of soil on physical observation may belief that there is no need for thorough design of ground water system, due to there physical observation on consolidation of the soil observed physically, such idea may definitely generate failure as this will produce more polluted groundwater, it will also increase the rate of water related diseases in the study area or even outside the deltaic environment. Preconsolidation and compressibility of soil structural deposition may be higher in the study location, the results has proof that such level of preconsolidation and high rate of compressibility were found to be insignificant in pheratic aquiferous zone, the contaminant as compared with word health organization is high and the water from such aquiferous zone will not be good for human consumption, the paper has stress the insignificant level of preconsolidation and compressibility of the soil that were found to have less influence on the degradation of the microbes in the study location.

References

- [1] Coliform, E. Coli Bacteria American Ground Water Trust was originally published in THE AMERICAN WELL OWNER, 2002, Number 2.
- [2] E. Coli and Other Waterborne Pathogens" Great Lakes Beachcast.http://www.great-lakes.net/beachcast/bw_waterborne.html (Accessed December, 2006).
- [3] Farmer, J.J. 3rd and Davis B. R., "H7 antiserum-sorbitol fermentation medium..". *Journal of Clinical Microbiology*. (2010); 22 (4) :620-625.
- [4] Adu-Bobie J, Frankel G, Bain C, Goncalves AG, Trabulsi LR, Douce G, Knutton S, Dougan G Detection of intimins α , β , γ , and δ , four intimin derivatives expressed by attaching and effacing microbial pathogens. *J Clin Microbiol* (1998) 36:662-668.
- [5] Blanco JE, Blanco M, Alonso MP, Mora A, Dahbi G, Coira MA, Blanco J Serotypes, virulence genes and intimin types of Shiga toxin (verotoxin)-producing *Escherichia coli* isolates from human patients: prevalence in Lugo (Spain) from 1992 through 1999. *J Clin Microbiol* (2004) 42:311-319.
- [6] Bernárdez MI, Blanco J Serotypes, virulence genes and intimin types of Shiga toxin (Verotoxin)-producing *Escherichia coli* isolates from cattle in Spain: identification of a new intimin variant gene (2004) (*eae*- α). *J Clin Microbiol* 42:645-651.
- [7] Blanco M, Podola NL, Krüger A, et al. Virulence genes and intimin types of Shiga-toxin-producing *Escherichia coli* isolated from cattle and beef products in Argentina 2004. *Int Microbiol* 7:269-276 9.
- [8] Blanco M, Schumacher S, Tasara T, Zweifel C, Blanco JE, Dahbi G, Blanco J, Stephan R Serotypes, intimin variants and other virulence factors of *eae* positive *Escherichia coli* strains isolated from healthy cattle in Switzerland. Identification of a new intimin variant gene (*eae*-2). (2005) *BMC Microbiol* 5:23 10.
- [9] Clarke SC, Haigh RD, Freestone Clarke SC, Haigh RD, Freestone PPE, Williams PH (2003) Virulence of enteropathogenic *Escherichia coli*, a global pathogen. *Clin Microbiol Rev* 16:365-378.
- [10] Frankel G, Phillips AD, Rosenshine I, Dougan G, Kaper JB, Knutton S Enteropathogenic and enterohaemorrhagic *Escherichia coli*: more subversive elements.(1998) *Mol Microbiol* 30:911-921.
- [11] Garrido P, Blanco M, Moreno-Paz M, Briones C, Dahbi G, Blanco JE, Blanco J, Parro V STEC-EPEC oligonucleotide microarray: a new tool for typing genetic variants of the LEE pathogenicity island of human and animal Shiga toxin-producing *Escherichia coli* (STEC) and enteropathogenic *E. coli* (EPEC) strains.(2006) *Clin Chem* 52:192-201.

- [12] Jenkins C, Lawson AJ, Cheasty T, Willshaw GA, Wright P, Dougan G, Frankel G, Smith HR Subtyping intimin genes from enteropathogenic *Escherichia coli* associated with outbreaks and sporadic cases in the United Kingdom and Ireland. (2003) *Mol Cell Probes* 17:149-1.
- [13] Jerse AE, Yu J, Tall BD, Kaper JB (1990) A genetic locus of enteropathogenic *Escherichia coli* necessary for the production of attaching and effacing lesions on tissue culture cells. *Proc Natl Acad Sci* (1990) USA 87: 7839-7843.
- [14] Kaper JB, Elliott S, Sperandio V, Perna NT, Mayhew GF, Blattner FR Attaching and effacing intestinal histopathology and the locus of enterocyte effacement. In: Kaper JB, O'Brien AD (eds) *Escherichia coli* O157:H7 and other Shiga toxin-producing *E. coli* strains. ASM Press, Washington, DC, (1998) pp 163-182.
- [15] Oswald E, Schmidt H, Morabito S, Karch H, Marchès O, Caprioli A Typing of intimin genes in human and animal enterohemorrhagic and enteropathogenic *Escherichia coli*: characterization of a new intimin variant. *Infect Immun* (2000) 68:64-71.
- [16] Perna NT, Mayhew GF, Posfai G, Elliott S, Sonnenberg MS, Kaper JB, Blattner FR Molecular evolution of a pathogenicity island from enterohemorrhagic *Escherichia coli* O157:H7. *Infect Immun* (1998) 66:3810- 3817.
- [17] Voss E, Paton AW, Manning PA, Paton JC Molecular analysis of Shiga toxin-producing *Escherichia coli* O111:H- proteins which react with sera from patients with hemolytic uremic syndrome. *Infect Immun* (1998) 66:1467-1472.
- [18] Zhu C, Agin TS, Elliot SJ, Johnson LA, Thate TE, Kaper JB, Boedeker EC Complete nucleotide sequence and analysis of the locus of enterocyte effacement from rabbit diarrheagenic *Escherichia coli* RDEC-1. *Infect Immun* (2001) 69:2107-2115.
- [19] Trabulsi LR, Keller R, Gomes TAT Typical and atypical enteropathogenic *Escherichia coli*. *Emerg Infect Dis* (2002) 8:508-513.
- [20] Jenkins C, Smith HR, Lawson AJ, Willshaw GA, Cheasty T, Wheeler JG Serotypes, intimin subtypes, and antimicrobial patterns of atypical enteropathogenic *Escherichia coli* isolated in England from 1993 to 1996. (2006) *Eur J Clin Microbiol Infect Dis* 25:19-24.
- [21] Identification of two new intimin types in atypical enteropathogenic *Escherichia coli* Miguel Blanco¹ Jesús E. Blanco¹ Ghizlane Dahbi¹ María P. Alonso¹, Azucena Moral María A. Coira² Cristina Madrid³ Antonio Juárez³ María I. Bernárdez¹ Enrique A. González¹¶ Jorge Blanco¹* *International journal of microbiology* (2006) vol 103-110.