***Original Research Article***

**MODELLING TRENDS OF HEPATITIS E INFECTION**

**Abstract**

**Background**: Hepatitis E is prevalent in many individuals, particularly in developing regions. While the infection often results in a self-limited, acute illness, it has a high likelihood of progressing to a chronic condition. Pregnant women, people with compromised immune systems (older people, people with underlying medical conditions), and people who have had solid organ transplants in particular are susceptible to chronic infections.

**Methods**: A comprehensive literature search on viral hepatitis E in children attending primary, secondary, and higher schools was conducted using various search terms. The focus on schools stems from the assumption that educational institutions should possess greater awareness of viral hepatitis E. A mathematical model was developed to describe the dynamic transmission of the virus, and sensitivity analysis was performed to assess the model's responsiveness to different parameters.

**Results**: The results indicate that oral consumption is the primary mode of transmission, especially in higher education settings. Sensitivity analysis demonstrated positive influences of all parameters associated with infection pathways. By utilizing the mathematical model of dynamic transmission, the projected infection rate can potentially be mitigated.

**Conclusion:** In conclusion, the projected infection rate as indicated by the model can be mitigated through the application of the dynamic transmission mathematical model. Implementing a range of strategies for targeted or widespread vaccination stands as a potent intervention for the improvement of public health.

**Keywords:** Seroprevalence, Dynamic transmission, Modelling, Infection

**Introduction**:

Infection with the hepatitis E virus (HEV), which causes more than half of cases of acute viral hepatitis, is a serious global public health concern. It is especially common in developing nations where access to sanitary facilities and clean water is limited. There have been reports of widespread hepatitis E epidemics in Asia, the Middle East, Northern and Central Africa, and Central America (Wanchuan et al, 2014). Nonetheless, isolated instances of HEV infection have also been reported in industrialized nations, such as the United States and Europe (Wanchuan et al, 2014). Notably, blood transfusions have been linked to sporadic HEV infection in recent research.

HEV may not be an old disease, according to historical reports, as it was first identified as an emergent illness with an unknown cause (Alayande et al, 2021). This experimentally transmitted illness was subsequently given the name "hepatitis E". There was a reported breakout of an uncommon and mysterious hepatitis virus among Soviet soldiers in the Afghanistan War in 1983. Using immunological electron microscopy, subsequent investigations found that the infected troops' faeces contained virus particles. Little quantities of cloned bile from experimentally infected macaques were used to isolate the viral genome.

In its initial form, HEV had at least four genotypes: 1, 2, 3, and 4. While genotypes 3 and 4 are found in several animal species, including deer, wild boars, and pigs, genotypes 1 and 2 are only found in humans. It's interesting to note that although infected animals may not show any symptoms, they can still spread the infection to people.

The colon is the main route by which the virus enters the human body, and infected people excrete it in their faeces. The ingestion of contaminated water and the faecal-oral route are the primary transmission routes—usually, the virus cycles for two to six weeks. A HEV infection typically manifests as fever, anorexia, nausea, vomiting, stomach discomfort, and liver enlargement in addition to jaundice. Severe cases may lead to mortality, severe liver failure, or fulminant hepatitis. It is necessary to find particular IgM and IgG antibodies in the patient's blood to diagnose HEV infection.

Hepatitis E is widespread, particularly in developing regions, and may progress to chronicity, especially in high-risk populations such as pregnant women, immunocompromised individuals (e.g., the elderly and those with underlying illnesses), and solid organ transplant recipients. In some instances, HEV infection can be fatal, as reported in cases of fulminant hepatic failure.

Acute HEV infection affects approximately three million individuals annually, resulting in 70,000 deaths, with the majority of cases occurring in endemic regions. Recent increases in cases have been reported in low-endemic countries, driven by inadequate sanitation, compromised water supplies, and food contamination. High seroprevalence rates of HEV, ranging from 27% to 80%, have been observed in developing nations like India and Southeast Asia. Surprisingly, industrialized countries like the United Kingdom and the USA have reported unexpectedly high seroprevalence rates of HEV (21-25%), potentially attributed to subclinical infections, animal exposures, cross-reactivity with other agents, or false-positive test results. Pregnant women and individuals with compromised immune systems face the highest mortality risks, ranging from 1% to 4%.

In Nigeria, limited investigations into HEV seroprevalence have shown varying results, with IgG and total antibody seroprevalence ranging from 7.0% to 66.75%. Co-infection rates among Nigerian healthcare workers have indicated a 27.3% co-infection rate with hepatitis B virus (HBV) and human Epstein-Barr virus (HEV). Additionally, a seroprevalence of anti-HEV IgM antibodies ranging from 0.4% to 0.9% has been reported. HEV outbreaks have occurred in different regions of Nigeria, including Port Harcourt and Bornu State, with varying genotypes involved. Consequently, further research and awareness efforts are essential to address the risks and provide projections to raise public awareness.

Recent research revealed a case of hepatitis E transmission through blood transfusion, originating from a donor who contracted the virus through zoonotic foodborne exposure. This highlights the growing concern about the safety of blood transfusions regarding hepatitis E. Several countries, including Japan, the United Kingdom, Saudi Arabia, and France, have extensively documented cases of hepatitis E transmission through blood transfusions.

Hepatitis E virus (HEV), a single-stranded positive-sense RNA virus (Putu et al, 2023), primarily spreads through the fecal-oral route. Its genome comprises three open reading frames (ORF) flanked by untranslated regions at the 5' and 3' ends. Typically, HEV infections manifest as acute, self-limiting hepatitis with an illness duration of 4-6 weeks, featuring symptoms like fatigue, appetite loss, nausea, and elevated serum ALT levels. However, in pregnant individuals, HEV infection can lead to fulminant hepatic failure, with a mortality rate as high as 20%. Immunocompromised individuals, including solid organ transplant recipients and HIV/AIDS patients, may develop chronic hepatitis, liver fibrosis, cirrhosis, and extrahepatic symptoms such as neurological and kidney issues.

HEV comprises four main genotypes, with genotypes 1 and 2 exclusively affecting humans, primarily through contaminated water sources in regions with poor sanitation, leading to widespread outbreaks. Consumption of meat from infected animals, especially pigs, occasionally results in HEV genotypes 3 and 4 infections in Western nations and China. Furthermore, novel HEV strains, including HEV-3ra from farmed rabbits in China in 2009 and HEV7 from Bactrian camels in 2016, pose potential risks to human health. Additionally, rat HEV, genetically distinct from HEV1-8, has been found to infect humans, raising concerns about HEV exposure and interspecies transmission.

HEV is a significant concern, with infections causing violent outbreaks, particularly in areas with poor sanitation and contaminated water. Zoonotic transmission plays a key role in clinical cases of hepatitis E in industrialized regions, often associated with consuming contaminated animal products or close contact with infected animals. However, underreporting of HEV incidence is common due to limited diagnostics, variable incubation periods (2-10 weeks), and symptoms resembling other forms of acute viral hepatitis.

Animal models have been essential for studying HEV replication and pathogenesis, especially when efficient cell culture systems are lacking. These models help researchers understand virus-related pathology, host-virus interactions, and evaluate the effectiveness and safety of vaccines and therapeutics.

Expanding the understanding of HEV replication in various tissues may reveal the virus's pathogenic potential and its propensity to cause extrahepatic symptoms. Some HEV genotypes are associated with specific extrahepatic conditions, such as pancreatitis (genotype 1) and renal symptoms (genotype 3).

Four HEV genotypes have been identified, each with distinct characteristics. Genotypes 1 and 2 are responsible for epidemic hepatitis, primarily transmitted through water and the fecal-oral route. Genotypes 3 and 4 are prevalent in domestic and wild pigs. The extent of virus transmission and its clinical implications remain subjects of debate, with insufficient evidence to support universal screening recommendations.

The objective of this study was to model the prevalence of hepatitis E across educational institutions, from elementary schools to higher education, to assess the rate of dynamic transmission and provide projections for public awareness.

**Methodology Approach**

The lack of widely recognized diagnostics and established operating methods for patient testing is a major factor in the exact incidence of HEV infection in many nations, leading to some linkage to formulate the model. Chronological Hepatitis E was adapted from (38).

Chronic Hepatitis E Virus Only a small proportion of individuals infected with HEV develop symptomatic episodes, with the majority experiencing asymptomatic seroconversion. The incubation period typically ranges from 3 to 8 weeks, with an average of 40 days. In otherwise healthy individuals, HEV infections usually result in self-limiting acute viral hepatitis, accompanied by symptoms such as hepatomegaly, jaundice, and dark urine. In rare cases, fulminant hepatitis can occur. Elevated levels of alanine aminotransferase (ALT), aspartate transaminase (AST), bilirubin, alkaline phosphatase, and glutamyl-transferase are commonly observed in liver enzyme panels, with ALT levels usually surpassing AST values. Hepatitis E Virus Persistence The possibility that HEV would develop into a chronic illness is a serious clinical concern, especially in those with weakened immune systems.

 Patients with diseases like leukaemia, HIV/AIDS, solid organ transplants, and immunosuppressive states have all been reported to have chronic HEV infections. A chronic infection is characterized by continuously detecting HEV RNA for over three months. It can result in liver fibrosis, cirrhosis, and eventually liver failure that requires a liver transplant. Fatigue, jaundice, weakness, fever, and stomach pain are possible symptoms. Replication of HEV infections can also occur at extra-hepatic locations in chronic cases. These mutations are of great clinical importance because they can potentially increase virulence and transmissibility. Hepatitis E during gestation: When a pregnant woman contracts HEV in the second or third trimester of her pregnancy, her chances of morbidity and death are enhanced; during outbreaks, death rates can increase by as much as 30%. Although the exact causes of increased mortality during pregnancy are not entirely known, immunological responses, hormonal changes, and genetic factors are thought to be involved. With a high rate of stillbirths, neonatal deaths, and maternal fatalities recorded during HEV epidemics, both the mother and the fetus face significant dangers. Animal-Produced Strains of the HCV Virus Many HEV species have been found in different animal species; some of these species have the potential to become zoonotic, while others are not.

Antibodies to HEV have been found in several animals, indicating the presence of HEV or related agents. However, conclusive evidence of HEV RNA in these animals is lacking. Some animal species, such as dairy cows in China, have been shown to be susceptible to genotypes 3 and 4 HEV infections, complicating the understanding of HEV strains in animals. Differentiating between distinct herpesviruses in these animals may require metagenomic data and virome characterization. HEV's transient viremia and often low viral RNA levels make this task challenging. Metagenomic sequencing of potential hosts from various locations and time periods may be necessary to avoid overlooking this frequently overlooked pathogen.

The most recent medical concern is the diversification of hepatitis E transmission routes, including organ transplants, blood transfusions, and blood, in addition to the traditional fecal-oral and zoonotic modes. Chronic HEV infection, particularly in transplant patients, has become more prevalent, surpassing the wide spectrum of infections, ranging from self-limiting to acute liver failure. Additionally, HEV's extra-hepatic manifestations affecting the kidneys, heart, pancreas, central nervous system (CNS), and other organs raise further concerns. However, advances in cell culture methods and animal models have improved our understanding of HEV's pathogenesis.

**Modelling Formulation**

Dynamic Transmission Hepatitis E epidemiology is rooted in the transmission of the virus through various pathways. The progression of the disease is modeled using the SEIR framework, representing susceptible, exposed, infectious, and recovered or deceased individuals. This model encompasses human-to-human, animal-to-human, and environment-to-human transmission routes, while also accounting for control measures like vaccination and sanitation.

The population is divided into six distinct epidemiological classes: susceptible (S(t)), immunized (V(t)), infectious (IN(t)), animals (A(t)), and recovered (R(t)). Additionally, the model incorporates HEV pathogens in food or water denoted as K(t), V(t), A(t), and P(t).

Based on collected data, it is observed that restroom facilities in most areas are limited, with some people resorting to using open areas such as bushes, leading to contamination of water bodies during rainfall. Many individuals rely on streams for their water supply due to the scarcity of boreholes. The proportion of people with access to sanitation facilities or cisterns is denoted as 'h' to account for this phenomenon. Environmental transmission can also occur through contact with animals, influencing the rate of environmental pollution, which is represented as:

$$\frac{de}{dt}=θ\left( 1-h\right)In-e………………………………………………………………1$$

Where $θ$ is the transmission rate from infected humans *In* to the environment.

Here, θ represents the transmission rate from infected humans to the environment and *In* to the environment. Individuals with access to clean water are protected from waterborne infections unless exposed to infected humans or animals. The susceptible human population (S) is susceptible to HEV with a transmission rate of α and experiences mortality at a rate μ\_p. Those without access to boreholes have a higher risk of HEV transmission from the contaminated environment (e). Additionally, transmission from animals to humans (g) is considered.

The transmission by infected humans to the environment is denoted as 'r,' and virus decay is represented by 'f':

$\frac{dS}{dt}=μ-\left(1-m\right)αgeS$ +r-f…………………………………………………………..2

Upon successful infection, individuals move to the exposed class (E) and experience transmission to the environment (r) and virus decay (f). The incubation period is characterized by$\frac{1}{π}$ days:

$\frac{dE}{dt}=αge-\left(μ+π\right)-f$ ……………………………………………………………….3

 $\frac{dL}{dt}=rs-\left(μ+β\right)L$ …………………………………………………………………4

The class denoted as 'L' represents latent infections, experiencing transmission (rs) and natural death (μ + βL):

Following the incubation period, individuals become infectious (I), displaying various symptoms associated with HEV infection. Recovery occurs at a rate σ:

$\frac{dI}{dt}= πE-\left(μ+σ\right)I$ …………………………………………………………………..5

Some infected individuals may succumb to the infection or recover, transitioning to the immune class (R):

$\frac{dR}{dt}=\left(1-π\right)gI-μR$ ………………………………………………………………….6

Equations 1-5 form a system of equations describing the transmission between humans and their environment. The environment reaches a steady state before humans if environmental dynamics are faster. Equation 1 provides the quasi-stationary-state (QSS) equation:

$e^{\*}=θ\left(1-h\right)In$………………………………………………………..7

Substituting the steady state into the system equations yields the following equations:

$$\frac{dS}{dt}=μ-αg\left(1-h\right)\left(1-m\right)SIn-μS+r-f$$

$\frac{dE}{dt}=αg\left(1-h\right)θ-In-\left(μ+π\right)j-f$ ……………………………………………………..8

 $\frac{dI}{dt}=πβ-\left(μ+σ\right)I$

 $\frac{dR}{dt}=\left(1-ε\right)δI-μR$

According to [55, 56], in this system, the total population is represented as 1 = S + E + I + R + K. Therefore, data will be fitted to the system equations after analysis. The endemic steady state is defined by:

$S^{\*}=\frac{1}{Z\_{0}}$, $E^{\*}=\frac{μ(μ+σ)d}{παθ(1-h)(1-m)}(Z\_{0}-1)$ , $I^{\*}=\frac{μd}{αθ(1-h)(1-m)}(Z\_{0}-1)$

 $Z^{\*}=1-S^{\*}-E^{\*}-I^{\*}$- K\*

Where: $Z\_{0}=\frac{απθ(1-h)(1-m)}{(μ+π)(μ+σ)}$

which represents the HEV reproduction number. The transmission rate of HEV during the infectious period is expressed as

$\frac{απθ(1-h)(1-m)}{(μ+π)(μ+σ)}$ while the survival of the incubation period is denoted as $\frac{π}{μ+π}$.

From table 1, Variable Definition:

S(t) -The number of susceptible humans at time t .

L(t)- The number of latent humans at time t.

 In(t)- The number of contagious people at time t

 R(t) - the total number of persons who have recovered (immune) at t.

 V(t)- the total number of individuals immunized at t.

 P(t) - the total number of HEV pathogens at time t.

Table 2: Parameters and their Definition for the HEV dynamic model.

**Method:**

We conducted an extensive literature search using various combinations of search terms related to viral hepatitis E and children in primary, secondary, and higher schools. Our primary focus was on schools since they are expected to have a better understanding of viral hepatitis E compared to other institutions. We developed a mathematical model to analyze the dynamic transmission of the virus, assuming uniform transmission lineages despite variations in dates. Sensitivity analysis was performed to assess the model's adaptability to different parameters.

Data for this analysis were primarily sourced from three secondary sources, which were documented by:

1. Emmanuel Ekanem et al. - Primary schools
2. (ii) MA Bugaje et al. - Secondary schools
3. (iii) Osanyinlusi et al. - Tertiary institutions
4. (iv) Chioma Ngozichukwu Pauline Mbachu (for data validation)

The laboratory examination procedure employed by most selected authors followed these steps:

Sample Collection: At the University Medical Center, five millilitres of blood were collected into EDTA bottles and then sent, chilled on ice packs, to the Microbiology Laboratory.

Sample processing: According to(Ekanem et al), Sample Collection: At the University Medical Center, five millilitres of blood were collected into EDTA bottles and then sent, chilled on ice packs, to the Microbiology Laboratory.

Within 24 hours of blood sample collection, whole blood samples were analyzed immediately for anti-HEV IgM antibodies using an enzyme-linked immunosorbent assay (ELISA) kit (CTK Biotech, Inc. USA). The kit used for the study had good accuracy, specificity, and sensitivity—98.1%, 99.2%, and 98.9%, respectively. The manufacturer's instructions were followed for performing the assays. It was previously believed that anti-HEV IgM antibodies indicated a recent or active HEV infection.

Data Analysis**:** E-views V10 and SPSS version 22 were used to analyze the data gathered.

Regarding gender (Table 4), a higher prevalence of hepatitis E virus (HEV) infection was observed among males (14.9%) compared to females (9.4%) across all three educational levels. In contrast to previous studies (references 57 & 58) that suggested an increased risk of HEV seropositivity based on the type of drinking water, our study revealed a higher prevalence of HEV infection among blood donors, with rates of 37.1% in primary, 24.4% in secondary, and 7.9% in tertiary education institutions. However, no statistically significant association with HEV positivity was found upon analysis.

It is worth noting that previous research has indicated that the primary route of HEV transmission is through the fecal-oral route (reference 59). In our study, we considered four different variables/observations. Table 10 illustrates the relationship between the type of institution and these variables. The table highlights that the type of drinking water is highly significant compared to other variables, with primary (p=0.081), secondary (p=0.019), and tertiary (p=0.004) institutions showing significant associations with HEV positivity. Notably, 74.77% of participants who tested positive had tap/well water as their source of drinking water (p<0.05), aligning with previous studies that have identified contaminated water as a primary source of HEV infection (reference 56). This may be attributed to inadequate water supply and poor sanitation conditions in the community.

In terms of contact with animals, our analysis did not yield any statistical significance across all three education levels (p>0.05). This finding does not support the hypothesis that zoonotic transmission is a significant route of HEV transmission.

ANALYSIS OF THE DYNAMIC MODEL

Figure 1 illustrates that as vaccination rates increase, the fraction of susceptible individual’s decreases, resulting in fewer people exposed to the disease. Figure 2 demonstrates that higher vaccination rates are associated with a decrease in the number of infected individuals.

Improving sanitation practices can significantly reduce HEV infection rates. Initiatives such as water chlorination, blood transfusion screening, and improved personal hygiene practices can play a vital role in reducing the spread of the disease in the community. Figure 3 show that enhancing sanitation and vaccination rates have a more pronounced impact on reducing the spread of HEV viruses. Eliminating the disease at the primary level could lead to its eventual disappearance by the time it reaches the tertiary level.

Figure 1: Impact of Sanitation Only Figure 2: Impact of Vaccination Only Figure 3: Impact of Both Sanitation and Vaccination

Sensitivity Analysis

Sensitivity analysis evaluates the model's responsiveness to changes in parameter values, which is crucial when dealing with data uncertainties and estimated parameter values. It helps identify which parameters are most influential and require intervention. In our analysis, we utilized Chitnis et al.'s methodology to determine a normalized forward sensitivity index, highlighting the most critical parameters.

A variable's normalized forward sensitivity index $μ$, which depends on a parameter g, is defined as $ψ\_{g}^{μ}= \frac{dμ}{dg} X \frac{μ}{g}$

For example, the sensitivity index of $R\_{k}$ (effective reproduction number) with respect to $s\_{h}$

 $ψ\_{g}^{R\_{k}}=\frac{∂R\_{k}}{∂s\_{h}} X \frac{s\_{h}}{R\_{k}}$

According to table 11, When $φ\_{s\_{h}}^{R\_{k}}>0$, it indicates that the value of c increases with an increase in $s\_{h}$, whereas when $φ\_{s\_{h}}^{R\_{k}}<0$, it signifies that c decreases as $s\_{h}$ decreases. The sensitivity indices' results are presented in the table below. Notably, parameters such as $s\_{h}$, α, π, f, σ, and b exhibit a positive correlation with $R\_{k}$, causing$R\_{k}$ to decrease as these parameters increase. On the other hand, variables like μ and g remain unaffected by changes in the generation of $R\_{k}$. Among all parameters, $s\_{h}$ emerges as the most sensitive, and parameters associated with the infection pathways consistently yield positive sensitivity indices.

**Forecasting of HEV from 2025 to 2040**

$$I\_{s}=F\_{s} x G\_{s}+α\_{s }$$

Where $I\_{s} $is the proportion of individuals infected in levels.

* $G\_{s}$ is the percentage of individuals in level s with access to clean water, sex drive
* $F\_{s}$ is the factor of HEV transmission in level s
* $α\_{s}$ is the transmission parameter in level s

To the best of our knowledge, this current transmission model marks the first attempt to explicitly estimate the risk of developing hepatitis across different levels of schooling. However, it's important to note several limitations that require careful consideration. While the timeframes may vary, the model assumes that the transmission patterns remain consistent. Notably, Table 12 demonstrates a notably higher transmission rate among different genders at the tertiary level.

In summary, the varying levels of HEV seroprevalence uncovered in this study highlight a concerning prevalence of HEV infection among blood donors in south-west Nigeria, underscoring the potential risk of HEV transmission through blood transfusions, which warrants further research. The mathematical non-linear dynamic model of transmission has been developed, incorporating vaccination and sanitation as control measures. The combination of sanitation and vaccination has demonstrated a more effective ability to minimize or eliminate the virus, thus preventing future outbreaks, as evidenced in the model.

The sensitivity analysis was conducted deliberately to assess the factors crucial to transmission. When devising an effective control strategy, parameters such as s\_h (contact rate) and σ (recovery rate) must be carefully considered. This dynamic model can serve as a valuable tool for public health interventions aimed at averting the projected figures from the forecast model, encompassing a range of targeted and general immunization strategies.

**Ethical Approval: All data sources provide ethical approval for their work.**

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Table 1: Variables and Their Definitions for the HEV Dynamic Model, adapted from Korobrinkov, A., 2007

|  |  |
| --- | --- |
| Variable | Definition |
| S(t)L(t)In(t)R(t)V(t)P(t)K(t) | The number of susceptible humans at time t The number of latent humans at a time tThe number of contagious people at time t The total number of persons who have recovered (immune) at t.The total number of individuals immunized at t.The total number of HEV pathogens at time t.The number of non-vaccinated humans at time t |

|  |  |  |  |
| --- | --- | --- | --- |
| Parameter | Definition | Value  | Source |
|  | Contact rate between S and other variables |  |  |
| $$s\_{h}$$ | 100 human/year | Assumed |
| $$α$$ | Transmission rate for Infectious humans | 0.01/year | Assumed |
| $$π$$ | Rate of pathogen release into food or water by infected persons | 0.02/year | Assumed |
| $$f$$ | Mortality rate of HEV pathogens | 1/7% per day | WHO |
| $$σ$$ | The natural pace of recuperation for contagious humans | 0.0238-0.1429$/day$ | WHO |
|   |
| $$g$$ | Transmission rate of animals to human beings | 0<a<1 | Estimated |
| $$b$$ | Rate of vaccination of susceptible people | 0.001/day | Assumed |
| $$μ$$ | Natural human mortality rate | 0.00156/year | WHO |
| $$δ$$ | Per capita rate of recovery from HEV | 1/12 per day | Estimated |
| $$β$$ | The speed at which latent individuals become infectious | 1/10/day | 60 |
|  |  |  |  |

**RESULTS**

**Demographic Analysis of the respondent**

Table 3a: Age of respondents (Primary)

|  |  |  |  |
| --- | --- | --- | --- |
|  S/no | Age | Frequency | Percentage |
| 1 | 1 – 4 | 134 | 39.0 |
| 2 | 5 – 9 | 83 | 24.1 |
| 3 | 10 – 14 | 81 | 23.0 |
| 4 | 15 – 18 | 46 | 13.4 |
| Total | 344 | 100 |

Table 3b: Age of respondents (Secondary)

|  |  |  |  |
| --- | --- | --- | --- |
|  S/no | Age | Frequency | Percentage |
| 1 | 10 – 14 | 124 | 35.8 |
| 2 | 15 – 18 | 153 | 44.2 |
| 3 | 19 – 24 | 69 | 19.9 |
| Total | 346 | 100 |

Table 3c: Age of respondents (Tertiary)

|  |  |  |  |
| --- | --- | --- | --- |
| S/no | Age | Frequency | Percentage |
| 1 | 11 -15 | 103 | 38.6 |
| 2 | 16 – 20 | 152 | 56.9 |
| 3 |  21 – 25 | 12 | 4.5 |
| Total | 267 | 100 |

Table 4: Gender of respondents

|  |  |  |  |
| --- | --- | --- | --- |
|  Gender | Primary | Secondary | Tertiary |
| Male | 169 (49.1) | 243 (70.4) | 106 (39.7) |
| Female | 175 (50.9) | 103 (29.6) | 161 (60.5) |
| Total | 344 (100) | 346 (100) | 267 (100) |

Table 5: Use of Alcohol

|  |  |  |  |
| --- | --- | --- | --- |
|  Alcohol | Primary | Secondary | Tertiary |
| Yes | 1 (0.3) | 22 (6.4) | 31 (11.6) |
| No | 343 (99.7) | 324 (95.6) | 236 (88.4) |
| Total | 344 (100) | 346 (100) | 267 (100) |

Table 6: Blood Transfusion

|  |  |  |  |
| --- | --- | --- | --- |
| Transfusion | Primary | Secondary | Tertiary |
| Yes | - | 6 (1.7) | 9 (3.4) |
| No | 344 (100) | 341 (98.3) | 258 (96.6) |
| Total | 344 (100) | 346 (100) | 267 (100) |

Table 7: Type of Drinking water

|  |  |  |  |
| --- | --- | --- | --- |
|  Type | Primary | Secondary | Tertiary |
| Stream | 108 (31.4 ) | 17 (4.9) | 8 (3.0 ) |
| Borehole/well | 205 ( 59.6) | 217 (62.7) | 73 ( 27.3) |
| Sachet/bottle | 31 ( 9.0) | 112 (32.4) | 186 ( 69.7) |
| Total | 344 (100) | 346 (100) | 267 (100) |

Table 8: Contact with animals/rearing animals

|  |  |  |  |
| --- | --- | --- | --- |
| Contact | Primary | Secondary | Tertiary |
| Yes | 283 (82.3) | 73 (21.1) | 45 (16.9) |
| No | 61 (17.7) | 273 (78.9) | 222 (83.1) |
| Total | 344 (100) | 346 (100) | 267 (100) |

Table 9: Sanitation type

|  |  |  |  |
| --- | --- | --- | --- |
|  Type | Primary | Secondary | Tertiary |
| Bush | 76 (22.1) | 69 (19.9) | 4 (1.5) |
| Pit | 250 (72.7) | 245 (70.8) | 81 (30.3) |
| Cistern | 18 (5.2) | 32 (9.2) | 182 (68.2) |
| Total | 344 (100) | 346 (100) | 267 (100) |

 Table 10: Logistic Regression

|  |  |  |  |
| --- | --- | --- | --- |
| **Variable/Institution** | **Primary** | **Secondary** | **Tertiary** |
| **Coefficients** | **Sig. level** | **Coefficients** | **Sig. level** | **Coefficients** | **Sig. level** |
| Age | 3.203 | 0.182 | 0.331 | 0.295 | 0.080 | 0.628 |
| Sex | 0.632 | 0.270 | 2.443 | 0.323 | 0.512 | 0.145 |
| Water | 0.594 | 0.081 | 0.643 | 0.019 | 0.240 | 0.004 |
| Blood Transfusion | 19.031 | 0.039 | 18.851 | 0.097 | 11.463 | 0.041 |
| Animals | 0.590 | 0.464 | 1.196 | 0.123 | 0.183 | 0.699 |
| Alcohol | 6.002 | 0.766 | 0.605 | 0.587 | 18.804 | 0.998 |
| Sanitation | 1.086 | 0.432 | 0.751 | 0.315 | 0.309 | 0.734 |
| Educational level | 7.042 | 0.048 | 6.878 | 0.031 | 9.317 | 0.011 |

Table 11:  **Sensitivity indices of** $R\_{k}$

|  |  |
| --- | --- |
| Parameters | Sensitivity indices of $R\_{k}$ |
| $$s\_{h}$$ |  0.99866 |
| $$α$$ | 0.00786 |
| $$πc$$ | 0.00542 |
| $$f$$ | 0.00089 |
| $$σ$$ | 0.77623 |
| $$g$$ | -0.00586 |
| $$b$$ | 0.06554 |
| $$μ$$ | -0.09465 |
| $$π$$ | 0.08873 |

Table 12: Projection from the table overtime

|  |  |  |
| --- | --- | --- |
| **Variable** | **Setting** | **Calendar Year** |
| **2025** | **2030** | **2035** | **2040** |
| **Drinking water** | Primary | 3 | 3 | 8 | 11 |
| Secondary | 17 | 19 | 21 | 27 |
| Tertiary | 1 | 2 | 6 | 8 |
| **Blood transfusion** | Primary | 1 | 1 | 1 | 2 |
| Secondary | 18 | 25 | 29 | 36 |
| Tertiary | 29 | 41 | 54 | 62 |
| **Contact with animals** | Primary | 1 | 1 | 1 | 2 |
| Secondary | 3 | 7 | 11 | 16 |
| Tertiary | 1 | 3 | 7 | 13 |