PREVALENCE OF HEPATITIS B AMONG FACULTIES OF MEDICINE, BASIC MEDICAL SCIENCES, DENTISTRY, NURSING AND AGRICULTURE STUDENTS

BY

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A PROJECT SUBMITTED TO THE DEPARTMENT OF MICROBIOLOGY, FACULTY OF LIFE SCIENCES, UNIVERSITY OF BENIN, BENIN CITY IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF DEGREE OF B.Sc. (HONS) IN MICROBIOLOGY

SEPTEMBER 2023

CERTIFICATION

This is to certify that the project work titled "Prevalence of Hepatitis B among University of Benin students" was carried out by **Paul Ajokpaoghene OKORO** with Matriculation Number **LSC1806805** of the Department of Microbiology, Faculty of Life Sciences, University of Benin, Benin City.

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APPROVAL

This is to certify that this project work was accepted in partial fulfillment of the requirement of the award of Bachelor of Sciences (B.Sc.) degree in Microbiology, Faculty of Life Sciences, University of Benin, Benin City.

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DEDICATION

I humbly dedicate this work to the Almighty God, who has given me patience, wisdom, divine health, strength and grace until this point of my life. All glory and honor to Him.

ACKNOWLEDGEMENT

My utmost appreciation goes to God for His divine grace during the course of this research work and throughout the course of this program.

I am sincerely grateful to my supervisor Mr. Afam Dunkwu- Okafor for his patience and understanding. I am very grateful to the Head of Department, Prof. (Mrs.) F.I. Akinnibosun and all other lecturers in the Department of Microbiology, University of Benin. Thank you for sharing your knowledge with me.

My deep gratitude also goes to my Course Adviser, Dr. (Mrs.) B.O. Ologbosere and Dr. (Mrs.) R. Adams for the immense role they played to ensure the completion of this project.

My profound gratitude to my parents, Mr. and Mrs. Okoro for their support and encouragement.

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ABSTRACT

Hepatitis B virus (HBV) infection is a global health disease. Infections can be acute with a propensity to evolve into chronic diseases and their attendant life-threatening complications. This study was aimed at determining the prevalence of HBV among faculty of Medicine, Basic Medical Sciences, Dentistry, Nursing, and Agriculture students in the University of Benin and its associated factors. A total of ninety-three students aged 16 to 59 years from different student using the systematic sampling technique. Ethical approval was obtained from the University of Benin Ethical Review Board, blood samples were collected from students and stored in a 5ml EDTA bottle. HBsAg was tested using the HBsAg rapid test kit. The test was carried out and interpreted according to the manufacturer's instructions. The questionnaire includes both closed and open-ended questions and will be sectionalized into socio-demographic characteristics, risk factors associated with HBV infection and vaccination history. Of the 93 students that were screened, 0 (0%) were positive. There was no statistically significant relationship between the hepatitis B status of the students and the socio-demographic parameters. Study shows a very low prevalence of Hepatitis B among faculty of Medicine, Basic Medical Sciences, Dentistry, Nursing, and Agriculture in the University of Benin Students. Though it is lower then the country's recorded prevalence level, routine vaccination program and public enlightenment should be strengthened for further control of HBV. Age and gender were not significantly associated with HBV infection in this study.

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND OF STUDY

Hepatitis B virus (HBV) is a hepatotropic virus with the potential to cause life-threatening liver damage (Pyrsopoulos *et al*., 2021). It is a disease of global health importance and is endemic in most parts of Sub-Saharan Africa; Nigeria inclusive. The virus has the potential to cause chronic infections and increases the risk of death from childhood hepatic failure, cirrhosis of the liver, and liver cancer. One-third of the World´s population is infected with HBV(Zampino et al., 2015). In the year 2019, the World Health Organisation (WHO) estimated that 296 million people were living with chronic HBV infection globally and also reported 820,000 deaths from it. Africa as a whole is endemic to the virus and within the same country, the endemicity level varies from district to district and in different target groups.

Nigeria is one of the countries in the HBV hyper-endemic zone. A meta-analysis of 46 Nigerian studies puts the average prevalence in the country as 13.6% (Musa *et a*l., 2015). The cause of hepatitis B infection is the hepatitis B virus (HBV), a DNA virus from the family Hepadnaviridae. Fatigue, black urine, fever, and jaundice (yellowing of the eyes and skin) are some signs of hepatitis B. Unprotected sex, sharing a razor, toothbrush, or clippers that have been in contact with blood, as well as from a pregnant woman to her child, are some of the ways that hepatitis B can be spread (Knipe et al 2001)

Hepatitis B virus is very contagious which can be transmitted through percutaneous which means through mucosal exposure to body fluid such as vaginal fluid, semen, sweat and infected blood. However, it is also transmitted intravenously. One of the common intravenous mode of transmission is sharing of sharp object (WHO 2015). One of the main risk factor that is

associated with the 38 development of hepatitis B viral disease is age factor, studies have shown that the immune system of an infant and elderly adult is weak making it a very easy entry for viral infection like hepatitis B (McMahon et al., 2009).

Another risk factor associated with the viral hepatitis B is the vertical transmission from an infected chronic carrier mother to the child especially when the mother contacted the virus at a late stage of the pregnancy (Ahizechukwu et al.,2011). The common means of hepatitis B virus transmission apart from the vertical transmission is by blood transfusion, sexual contact and practice of intravenous drug use. The practice of unsafe injection which involve the repeated use of syringe from one patient to the other without sterilization may enhance the spread HBV infection. This virus can also be transmitted by surgery procedures.

Hepatitis B has a substantial effect on world health. It is a major contributor to liver cancer and also increases the risk of liver cirrhosis and liver failure. It was projected that complications from hepatitis B caused approximately 880,000 deaths in 2015. Hepatitis B is now the tenth most common cause of mortality in the globe.

The prevalence of hepatitis B virus in Nigeria has earlier been reported to be endemic in Nigeria where about 18 million Nigerians are currently infected with hepatitis B virus (Musa et al., 2000). Prevalence is a crucial epidemiological concept that plays a central role in understanding the distribution of diseases and health conditions within a population. It quantifies the extent to which a specific condition exists at a particular point in time or over a specified period within a given population. This study helps to understand the prevalence, its significance, measurement methods, and its applications in public health amongst the Students of the University of Benin (Rothman *et al.*, 2008).

University of Benin is a federal tertiary institution located in Ovia North East LGA in the heart of Edo state, Benin city. This institution house students of different sex(male and female), various ethnic and religious background between the age fifteen and above. University of Benin has two Campus: Ekehnwa campus and Ugbowo campus. The institution has thirteen(13) faculties among which five(5) faculties: Medicine, Basic Medical Sciences, Dentistry, Nursing, and Agriculture were used as target site located in Ugbowo campus making it an ideal setting for study of an endemic Virus such as Hepatitis B.

1.2 AIMS:

The aim of this study is to assess trends in the prevalence of Hepatitis B Virus infection and its associated factors in a tertiary institution (University of Benin) Benin City, Edo State. Nigeria.

The specific objectives of this study include;

1. To assess the knowledge of Students about the mode of transmission of the infection.
2. To estimate the prevalence of risk factors associated with Hepatitis B Virus infection.
3. To estimate number of vaccinated students and their awareness of HBV vaccine
4. To determine the prevalence rate of Hepatitis B

CHAPTER TWO

LITERATURE REVIEW

2.1 HISTORICAL BACKGROUND

Hepatitis B-like symptoms were described in ancient texts dating back to the 5th century BC in China (Hollinger and Liang, 2001). The ancient Greeks and Romans also documented jaundice and liver-related illnesses resembling Hepatitis B (Magner, 2005). Hepatitis B was discovered in 1966. Serendipity led to the identification of the Australia antigen, which we now know as hepatitis B surface antigen (HBsAg). An immunodiffusion precipitin line between the HBsAg present in the serum of an Australian Aborigine and the antibody to HBsAg in a patient with hemophilia who had received multiple transfusions provided the first clue. The subsequent development of acute hepatitis in a laboratory technician provided the essential link to the clinical illness. For these achievements, Dr. Baruch Blumberg received the Nobel Prize in Physiology or Medicine in 1976 (Blumberg et al., 1997).

In rapid succession, discoveries from around the world enhanced our understanding of the complexity of this unusual virus. Although there has not yet been a substantial decrease in the overall prevalence of infection. The history of Hepatitis B spans centuries, from ancient descriptions of similar illnesses to the discovery of the virus and the development of vaccines. Understanding the historical context of Hepatitis B is essential for appreciating the progress made in prevention and treatment and for addressing ongoing challenges in controlling the disease.

2.2 VIROLOGICAL CHARACTERISTICS

HBV belongs to a family of closely related DNA viruses called the hepadnaviruses. Included in this family are the woodchuck hepatitis virus, the duck hepatitis B virus, and several other avian and mammalian variants. All the hepadnaviruses have similar hepatotropism and life cycles in their hosts.**(**Wei et al., 1994) Chronic hepatitis and hepatocellular carcinoma, for example, are commonly observed in woodchucks and less frequently observed in ground squirrels and ducks. The viral genome of HBV is a partially double-stranded circular DNA of approximately 3200 base pairs that encodes four overlapping open reading frames.

The whole virion, or Dane particle, is a 42-nm sphere that contains a core, or nucleocapsid, enclosing the DNA. One peculiar feature of HBV is the great excess of envelope material found in the circulation, consisting of both small spheres and rods with an average width of 22 nm.

The viral envelope encoded by the *S* gene contains three distinct configurations synthesized in all persons, termed the large, middle, and major proteins, which are produced by beginning transcription with, respectively, *preS1, preS2,* or the *S* gene alone.

Hepatitis B core antigen (HBcAg) is the nucleocapsid that encloses the viral DNA. When HBcAg-derived peptides are expressed on the surface of hepatocytes, they induce a cellular immune response that is crucial for killing infected cells.

Hepatitis B e antigen (HBeAg), a circulating peptide derived from the core gene and then modified and exported from liver cells, serves as a marker of active viral replication.**12** HBeAg may act as a tolerogen, since its presence in the circulation has been associated with a diminished immune response because of its close resemblance to HBcAg, the putative target of the immune response. With few exceptions, HBeAg is present only in persons who have circulating serum HBV DNA.

2.3 EPIDEMIOLOGY

The distribution of hepatitis B infection varies greatly throughout the world. In areas where the prevalence is high, such as Southeast Asia, China, and Africa, more than half the population is infected at some time in their lives, and more than 8 percent are chronic carriers of the virus, the result of either neonatal transmission (vertical) or transmission from one child to another (horizontal). Areas with low levels of endemicity include North America, Western Europe, and Australia, where only a minority of people come into contact with the virus, as a result of horizontal transmission among young adults **(**Kane et al., 1995). The World Health Organization estimates that the number of HBV carriers will reach 400 million by the year 2000. The numbers will continue to increase until neonatal vaccination and immunization are universally accepted. Chronic HBV infection afflicts 1.25 million people in the United States. Of the 22,000 infants born each year to HBsAg-positive mothers in the United States, more than 98 percent receive monoprophylaxis (hyperimmune globulin and vaccine) and are protected from infection. A vaccination program aimed at all newborn infants and adolescents is under way in this country (Kim et al.,1995).

Most HBV infections in developed countries result from sexual activity, injection-drug use, or occupational exposure. Other, less frequent causes of infection include household contact, hemodialysis, transmission from a surgeon **(**Harpaz et al., 1996) and receipt of organs or blood products. No clear risk factors are found in 20 to 30 percent of patients, perhaps because of a reluctance to report high-risk behavior or possibly mucosal or other unrecognized routes of infection. Because HBV is present in serum in large quantities (108 to 1010 virions per milliliter), it is not surprising that HBV can also be detected in semen, saliva, cervical secretions, and leukocytes. Respiratory, water-borne, or insect-related infections have not been documented.

2.3.2 EPIDEMIOLOGY OF HEPATITIS B IN NIGERIA

Hepatitis B is endemic in Nigeria, with a high prevalence rate. According to the Nigeria Hepatitis B Consortium, the national prevalence of Hepatitis B surface antigen (HBsAg) among adult's ranges from 9% to 15%, making it one of the highest prevalence rates in the world (Forbi et al., 2015).

The final analyses included 47 studies with 21,702 participants that revealed a pooled prevalence of 9.5%. A prevalence estimate above 8% in a population is classified as high. Sub-group analyses revealed the highest HBV prevalence in rural settings (10.7%). The North West region had the highest prevalence (12.1%) among Nigeria's six geopolitical zones/regions. The estimate of total variation between studies indicated substantial heterogeneity. These variations could be explained by setting and geographical region (Busayo et al 2021).

During the period of 3 months of study among voluntary blood donors in a known tertiary hospital in Edo state, the total number of blood donors was 2893. The gender was not specified in the reports. 29 donors were confirmed positive for HBV, a prevalence of 1.02%. While 49 donors were confirmed positive for HCV and prevalence at 1.64% (Izevbuwa and Ojo 2021)

2.4 IMMUNOPATHOGENESIS

The host's immune system attack against HBV is the cause of the liver injury, mediated by a cellular response to small epitopes of HBV proteins, especially HBcAg, presented on the surface of the hepatocyte. HLA class I–restricted CD8+ cells recognize HBV peptide fragments derived from intracellular processing and presentation on the hepatocyte surface by class I molecules. This process leads to direct cell killing by the CD8+ cytotoxic T lymphocyte (Chisari et al 1995). Only a limited array of peptide residues from the HBV proteins can gain access to the class I binding groove. Recent evidence suggests that the polymorphic nature of the

major-histocompatibility-complex binding sites and differences in the T-cell repertoire among persons leads to highly variable binding affinity for the immunodominant HBV peptides, which in turn determines the outcome after acute HBV infection (Ferrari et al., 1994). HLA class II–restricted CD4+ lymphocytes recognize peptide fragments derived from viral proteins presented in the antigen groove of non-hepatic antigen-presenting cells, principally macrophages. The identification of viral-protein epitopes by the CD4+ cell leads to the stimulation of T-cell proliferation and cytokine synthesis and provides help for B-cell responses.

The different immune responses in patients in whom virus is cleared successfully and those in whom it is not (in the absence of any evidence of immunodeficiency) depend on the match between the HBV peptides presented by host major-histocompatibility-complex molecules and the specific T-cell–receptor repertoire of the host. If sufficient recognition and activation occur, the immune response is carried to completion, all infected cells are destroyed, viral replication is aborted, and antibodies to HBsAg prevent the reinfection of hepatocytes. If the response is inadequate, the infection continues. However, this is an oversimplification. Recent studies suggest that cytotoxic T lymphocytes may directly inhibit viral replication and thus inactivate HBV without killing the infected hepatocyte(Guidotti et al 1996). It is likely that minute quantities of virus remain indefinitely, even when HBsAg and HBV DNA are no longer detectable in serum (Chisari et al., 1997). It has recently been suggested that other genetic differences modifying the host immune response play a part in determining the outcome, including variation in the mannose-binding protein, a viral opsonin (Thomas et al., 1996).

2.4 LIFE CYCLE OF HEPATITIS B VIRUS IN THE HUMAN HOST

Under most circumstances, HBV is not cytopathic (i.e., it does not kill hepatocytes). An intact immune system is vital to cell injury and viral clearance (Jung et al.,1994). For practical purposes, the severity of the hepatocyte injury reflects the vigor of the immune response: the most complete immune response is associated with the greatest likelihood of viral clearance and the most severe liver injury. Ninety-five percent of infected neonates with immature immune systems become asymptomatic chronic HBV carriers, as compared with 30 percent of children infected after the neonatal period but before six years of age. Only 3 to 5 percent of adults remain chronically infected; the remainder have acute infections resulting in viral clearance.

It is useful to consider the life cycle of HBV in four stages, present in some form in all infected patients. The first stage is characterized by immune tolerance. In the healthy adult, this incubation period lasts about two to four weeks. In contrast, with neonatal infection, this period often lasts for decades. In most cases of HBV infection throughout the world, active viral replication continues despite little or no elevation in the aminotransferase levels and no symptoms of illness.

In the second stage, an immunologic response develops or improves, leading to cytokine stimulation and direct cell lysis and the inflammatory process. Secretion of HBeAg still occurs in stage 2, but HBV DNA levels in serum drop as the number of infected cells declines. In patients with acute HBV infection, stage 2 is the period of symptomatic hepatitis and typically lasts three to four weeks. In patients with chronic disease, stage 2 may persist for 10 or more years, leading to cirrhosis and its complications.

When the host is able to mount a response that eliminates infected cells or greatly diminishes their number, active viral replication ends and the third stage begins. In this stage, HBeAg is no longer present, and antibody to HBeAg becomes detectable. A marked decrease in viral DNA is observed,

although many patients remain positive for HBV DNA as detected by PCR. Practically speaking, the infection has cleared by stage 3, and aminotransferase levels become normal. However, patients remain positive for HBsAg, presumably because of the integration of the *S* gene into the host's hepatocyte genome.

Most patients eventually become negative for HBsAg and positive for antibody to HBsAg, marking the fourth, or immune, stage in the HBV life cycle. HBV DNA can no longer be detected by any means, and the patient is unlikely to become reinfected or to have a reactivated infection. Factors affecting the evolution through the four stages, in addition to the genetic predisposition of the host, noted above, include the presence of other viruses, treatment with immunosuppressive agents, sex, and the appearance of HBV mutants.

2.5 ASSOCIATED CLINICAL SYNDROMES

2.5.1HEPATITIS D

The remarkable discovery in 1977 of a passenger virus termed delta, or hepatitis D virus (HDV), added to our understanding of HBV(Rizzetto et al.,1997). HDV is a defective, RNA-containing passenger virus requiring the helper functions provided by HBV, including nucleocapsid assembly and provision of an HBsAg-derived envelope. In virtually all circumstances, HDV cannot replicate in the absence of HBV, because whole virions cannot be formed. HDV resembles certain plant viruses; there are no other known passenger viruses in the animal kingdom. HDV infection occurs either as a simultaneous co-infection with HBV (acute HBV and HDV infections), which is usually self-limited because of the eradication of HBV, or as a superinfection in an HBV carrier, typically an injection-drug user. HDV infection is an important consideration when the condition of a patient with chronic HBV infection worsens or when a test for HBeAg is negative but active liver disease persists(Govindarajan et al.1984).

2.5.2 HEPATITIS C

Many injection-drug users have detectable antibody to HBcAg and hepatitis C virus (HCV), indicating exposure to both parenteral hepatotropic viruses. Although HBV is cleared in most adults, approximately 90 percent have active hepatitis C, and a smaller fraction (approximately 5 percent) have dual infections, with very active liver disease.

2.5.3 HUMAN IMMUNODEFICIENCY VIRUS INFECTION

Patients with both human immunodeficiency virus (HIV) infection and HBV infection do not seem to have altered outcomes as a result of the combined infections. That is, HBV infection does not alter the outcome of HIV infection, nor does HIV infection alter the evolution of HBV infection, despite the recognized effects of HIV on the immune system. Until recently, HBV infection was not treated in HIV-positive persons because of their limited life expectancy; improvements in survival and new agents that are effective against one or both of the viruses have made treatment possible.

2.5.4 FULMINANT HEPATITIS B

Acute liver failure with coagulopathy, encephalopathy, and cerebral edema develops in 1 percent of patients with acute hepatitis B. Fulminant infection occasionally develops after the withdrawal of immunosuppressive agents — for example, in patients receiving chemotherapy for cancer. The cause of the fulminant infection is a heightened immune response to the virus, provided HDV or HCV coinfection or superinfection is ruled out. Patients with acute liver failure have early clearance of HBsAg, which may obscure the diagnosis, but a positive test for IgM antibody to HBcAg should clarify the situation. Rapid clearance of HBV is favorable, since HBV reinfection seldom develops in the allograft, should transplantation be required.

2.6 HEPATITIS B AFTER LIVER TRANSPLANTATION

Most patients with chronic hepatitis B requiring liver transplantation have reinfection of the hepatic graft. In many patients, progressive hepatic failure develops because of a peculiar form of fibrosing cholestatic hepatitis seen almost exclusively in liver grafts but also seen occasionally in patients with bone marrow or renal transplants. High viral titers in serum and liver tissue may directly injure hepatocytes in immunosuppressed patients. Prophylaxis, consisting of high-titer hepatitis B immune globulin, is costly, not uniformly effective, and likely to be replaced by nucleoside analogues.

2.7 INFECTION WITH MUTANT VIRUSES

A small but important group of patients have no detectable HBeAg in serum because of a viral mutation that does not affect replication, underlining the value of testing for both HBeAg and HBV DNA. HBeAg-negative mutants have been associated with fulminant hepatitis B and have caused more severe chronic hepatitis B and more rapid graft loss after transplantation than nonmutant HBV. The most common mutation is a single nucleotide change at position 1896 (G to A), resulting in a stop codon (TGG to TAG) at the end of the precore region, preventing HBeAg synthesis. Since only the precore region is deleted, HBcAg synthesis remains intact. Lack of circulating HBeAg, the immune tolerogen, may contribute to the more aggressive disease frequently observed. Other core-related mutations yielding similar results have been noted. A variety of mutant forms occurring in the same patient (similar to hepatitis C quasispecies) have also been described.

A few patients have been identified who have mutations in the *S* gene sufficient to prevent normal production of HBsAg despite detectable viral DNA levels. These mutations are extremely rare, as are "vaccine escape" mutants, viral subtypes that can cause HBV infection in vaccinated persons.

2.8 EXTRAHEPATIC DISEASES

Hepatitis B is frequently detected in patients with polyarteritis nodosa and less commonly in those with membranous or membranoproliferative glomerulonephritis or leukocytoclastic vasculitis, all immune-complex–mediated diseases. A previously noted association with mixed cryoglobulinemia now seems less likely, since, in retrospect, many of the patients had hepatitis C, and hepatitis B represented an innocent coinfection. Treatment of the underlying HBV infection generally improves the associated disease, particularly in the case of glomerulonephritis. In contrast, immunosuppressive treatment (e.g., for polyarteritis nodosa) results in a remission of the disease but perpetuates the HBV infection.

2.9 HEPATOCELLULAR CARCINOMA

HBV infection is a risk factor for hepatocellular carcinoma, which arises almost exclusively in patients with cirrhosis. No specific HBV-oncogene sequence appears to be responsible for this tendency. As with other forms of liver cancer, tumors associated with hepatitis B result from chronic inflammation and repeated cellular regeneration, typically occurring only after 25 to 30 years of infection. The risk of hepatocellular carcinoma greatly diminishes after the resolution of chronic hepatitis B, as shown by epidemiologic studies. Vaccination to decrease the carrier rate in the population has recently led to a marked decline in the incidence of hepatocellular carcinoma in Taiwanese children.

2.10 TRANSMISSION

In highly endemic areas, hepatitis B is most commonly spread from mother to child at birth (perinatal transmission) or through horizontal transmission (exposure to infected blood), especially from an infected child to an uninfected child during the first 5 years of life. The development of chronic infection is common in infants infected from their mothers or before the age of 5 years.

Hepatitis B is also spread by needlestick injury, tattooing, piercing and exposure to infected blood and body fluids, such as saliva and menstrual, vaginal and seminal fluids. Transmission of the virus may also occur through the reuse of contaminated needles and syringes or sharp objects either in health care settings, in the community or among persons who inject drugs. Sexual transmission is more prevalent in unvaccinated persons with multiple sexual partners.

Hepatitis B infection acquired in adulthood leads to chronic hepatitis in less than 5% of cases, whereas infection in infancy and early childhood leads to chronic hepatitis in about 95% of cases. This is the basis for strengthening and prioritizing infant and childhood vaccination.

The hepatitis B virus can survive outside the body for at least 7 days. During this time, the virus can still cause infection if it enters the body of a person who is not protected by the vaccine. The incubation period of the hepatitis B virus ranges from 30 to 180 days. The virus may be detected within 30 to 60 days after infection and can persist and develop into chronic hepatitis B, especially when transmitted in infancy or childhood.

2.11 SYMPTOMPS

Most people do not experience any symptoms when newly infected. Some people have acute illness with symptoms that last several weeks:

- yellowing of the skin and eyes (jaundice)
- dark urine
- feeling very tired
- nausea
- vomiting
- pain in the abdomen.

When severe, acute hepatitis can lead to liver failure, which can lead to death. Although most people will recover from acute illness, some people with chronic hepatitis B will develop progressive liver disease and complications like cirrhosis and hepatocellular carcinoma (liver cancer). These diseases can be fatal.

2.12 DIAGNOSIS OF HEPATITIS B

It is not possible on clinical grounds to differentiate hepatitis B from hepatitis caused by other viral agents; hence laboratory confirmation of the diagnosis is essential. Several blood tests are available to diagnose and monitor people with hepatitis B. Some laboratory tests can be used to distinguish acute and chronic infections, whilst other can assess and monitor the severity of liver disease. Physical examination, ultrasound, fibroscan can also be performed to assess degree of liver fibrosis and scarring and monitor progression of liver disease. WHO recommends that all blood donations be tested for hepatitis B to ensure blood safety and avoid accidental transmission.

As of 2019, 30.4 million people (10.5% of all people estimated to be living with hepatitis B) were aware of their infection, while 6.6 million (22%) of the people diagnosed were on treatment. According to latest WHO estimates, the proportion of children under five years of age chronically infected with HBV dropped to just under 1% in 2019 down from around 5% in the pre-vaccine era ranging from the 1980s to the early 2000s.

In settings with high Hepatitis B surface antigen seroprevalence in the general population (defined as ≥2% or ≥5% HBsAg seroprevalence), WHO recommends that all adults have access to and be offered HBsAg testing with linkage to prevention and care and treatment services as needed. WHO also recommends blood donor screening, routine testing for hepatitis B all pregnant women to provide the opportunity to institute measures for prevention of MTCT as well as focused or targeted testing of specific high-risk groups (including migrants from endemic regions, partners or family members of infected persons, and health-care workers PWID, people in prisons and other closed settings, MSM and sex workers, HIV-infected persons.

2.13 CONTROL OF HBV INFECTION

There are broadly three strategies for dealing with HBV infection in the developed countries, immunization for at risk population, antiviral drugs (lamivudine, adeforvir and dipivoxil) and immunostimulatory therapy with alpha-interferon for those affected. (Finlayson *et al.*, 1984).

Immunization is the most effective means of controlling and HBV world-wide (Kire et al,. 1993). The vaccine has an outstanding record of safety and efficacy, and it is 95% effective in preventing development of the chronic carrier state. In Africa, vertical transmission accounts for 1-5% of cases, (James et al., 1993) while most children are infected with HBV between ages of 2-1l years through horizontal transmission, hence universal immunization at birth has been

adopted. As cost-effective measure it has been incorporated into WHO expanded programme on immunization (EPI) on global basis according to Yaounde declaration at the International conference on the control of HBV held in 1991.

In addition to the above measures where it is feasible, HBV infection in Nigeria can be prevented or drastically reduced through health education of the general population on the various mode of transmission of HBV and preventive measures (Sirisena *et al.,* 2002). Such measures include careful handling of blood and body fluid since they are potentially infectious. Also discouraging communal sharing of blade/sharp instruments used for shaving, barbing, manicure and body piercing/cutting and high level sexual networking. Prechewing of solid for children by an adult, especially those at risk for HBV infection should be discouraged because saliva is known to transmit HBV.

WHO recommends universal screening of blood and plasma for HBsAg by sensitive method before transfusion (Kire et al,. 1993). Even when all blood donations are screened for HBsAg, donations from volunteered non remunerated donors have been proved to be safest. About 2 out of 1000 units screened plasma donations, negative for HBsAg using a very sensitive test are still infectious because the sensitivity of the third generation test is not 100%. Addition of a low dose hepatitis B immunoglobulin to potentially infectious plasma appears to be reliable measure to eliminate the hepatitis B transmission. This is preferred to other methods for labile plasma derivatives (Brummechuis et al 1983). Where possible only donations from immunized donors with a detectable amount of anti-HBs should be collected either for transfusion or for preparation of plasma derivative. Pasteurization of plasma derivatives like albumin, factors iii and viii at 60°C for at least 10 hours is essential for the elimination of HBV. Because of risks of blood transfusions, it should be given only when it is absolutely necessary as it was said that most

blood transfusions were not necessary (Obiaya et al., 1982).

Babies born to HBsAg positive mother should be given hepatitis B immunoglobulin at birth and active immunization should commence immediately.Post exposure prophylaxis with hepatitis B immunoglobulin should be given promptly in all cases of suspected blood or body fluid inoculation as this could reduce HBV infection.

2.14 TREATMENT

There is no specific treatment for acute hepatitis B. Chronic hepatitis B can be treated with medicines.

Care for acute hepatitis B should focus on making the person comfortable. They should eat a healthy diet and drink plenty of liquids to prevent dehydration from vomiting and diarrhoea.

Chronic hepatitis B infection can be treated with oral medicines, including tenofovir or entecavir.

Treatment can

- slow the advance of cirrhosis
- reduce cases of liver cancer
- improve long term survival.

Most people who start hepatitis B treatment must continue it for life. It is estimated that 12–25% of people with chronic hepatitis B infection will require treatment, depending on setting and eligibility criteria. The ongoing 2023 update of the WHO Hepatitis B treatment guidelines will expand treatment eligibility and increase the proportion of people on treatment.

In low-income settings, most people with liver cancer present late in the course of the disease and die within months of diagnosis. In high-income countries, patients present to hospital earlier in the course of the disease and have access to surgery and chemotherapy, which can prolong life for several months to a few years. Liver transplantation is sometimes used in people with cirrhosis or liver cancer in technologically advanced countries, with varying success.

2.15 PREVENTION

Hepatitis B is preventable with a vaccine. All babies should receive the hepatitis B vaccine as soon as possible after birth (within 24 hours). This is followed by two or three doses of hepatitis B vaccine at least four weeks apart. Booster vaccines are not usually required for people who have completed the three-dose vaccination series.

The vaccine protects against hepatitis B for at least 20 years and probably for life. Hepatitis B can be passed from mother to child. This can be prevented by taking antiviral medicines to prevent transmission, in addition to the vaccine. To reduce the risk of getting or spreading hepatitis B:

- practice safe sex by using condoms and reducing the number of sexual partners
- avoid sharing needles or any equipment used for injecting drugs, piercing, or tattooing
- wash your hands thoroughly with soap and water after coming into contact with blood, body fluids, or contaminated surfaces
- get a hepatitis B vaccine if working in a healthcare setting.

2.16 VACCINATION OF HEPATITIS B

2.16.1 HISTORICAL BACKGROUND:

The development of the Hepatitis B vaccine marked a significant milestone in the field of immunization. The first successful Hepatitis B vaccine was introduced in the early 1980s, following the discovery of the Hepatitis B virus (HBV) and the Australia antigen by Dr. Baruch Blumberg. The vaccine was developed by Dr. Maurice Hilleman and his team, revolutionizing the prevention of this infectious disease (Hilleman 2002).

2.16.2 COMPONENT OF THE VACCINE

1. Recombinant HBsAg: The core component of the Hepatitis B vaccine is a synthetic form of the HBsAg protein. This protein is a part of the outer envelope of the Hepatitis B virus. Importantly, the HBsAg used in the vaccine is produced through genetic engineering techniques and is not derived from actual virus particles. This means that the vaccine does not contain live or inactivated virus and cannot cause Hepatitis B infection.

2. Adjuvants: Adjuvants are substances added to vaccines to enhance the body's immune response to the antigen (in this case, HBsAg). In the case of the Hepatitis B vaccine, aluminum salts (aluminum hydroxide or aluminum phosphate) are commonly used as adjuvants. They help stimulate a stronger and longer-lasting immune response to the HBsAg.

2.16.3 MECHANISM OF ACTION

The Hepatitis B vaccine works by stimulating the immune system to recognize and produce antibodies against the HBsAg, which is a specific protein on the surface of the Hepatitis B virus. Here's how the vaccine's mechanism of action unfolds;

Exposure to HBsAg: When a person is vaccinated with the Hepatitis B vaccine, they are injected with a safe, synthetic form of HBsAg. This antigen mimics the presence of the virus in the body.

Immune Response: The immune system recognizes the HBsAg as foreign and mounts an immune response. Specifically, B cells, a type of white blood cell, are activated.

Antibody Production: B cells produce antibodies, known as anti-HBs antibodies, that are specifically designed to target and neutralize the HBsAg.

Memory Cell Formation: Some B cells become memory B cells, which "remember" the HBsAg. These memory cells persist in the body, ready to respond quickly if the person is ever exposed to the real Hepatitis B virus in the future.

Protection: If the vaccinated individual encounters the actual Hepatitis B virus at any point after vaccination, their immune system recognizes the virus's HBsAg, and the pre-existing antibodies quickly neutralize the virus. This prevents the virus from establishing an infection in the liver.

The result of this process is the development of immunity against the Hepatitis B virus, without the individual ever being exposed to the actual virus or developing the disease. This immunity provides long-lasting protection, often for decades. It's important to note that the Hepatitis B vaccine is typically administered as a series of three or four doses, depending on the vaccine brand and the person's age and health status. Completing the recommended series is crucial to ensure the highest level of protection. Additionally, booster doses may be recommended in

certain situations, such as for healthcare workers or those with weakened immune systems, to maintain long-term immunity.

In summary, the Hepatitis B vaccine uses a synthetic form of the viral surface protein (HBsAg) to stimulate the immune system to produce antibodies. These antibodies provide protection against future Hepatitis B virus exposure, without causing the disease itself. This vaccine has proven to be highly effective in preventing Hepatitis B infection and its associated complications.

CHAPTER THREE

METHODOLOGY

3.1 STUDY SETTING

This study will be conducted in the University of Benin in Benin City, Edo State, Nigeria (6.3998° N, 5.6099° E). Both are male and female students. The university serves students in Edo State as well as neighbouring States. The choice of a university selection is based on pragmatic factors, including the availability of researchers in Benin City.

3.2 Study design

The study will use a cross-sectional design, collecting data on both outcome variables and co-variates at one point in time.

3.3 Sample Size Determination

A representative sample size will be determined using the formula below:

$$n = \frac{3.84\pi\ (1-\pi)}{w^2}$$

Where n=estimated sample size; π=prevalence of HBV among students in Nigeria, estimated at 13.7% (Dan Nwafor et al., 2021); w= precision of estimate.

Therefore, with a prevalence of 13.7 (0.137) and wanting to estimate our sample size to within 3% (w=0.03) with 95% certainty, the estimated sample size for this study is 504. To account for a non-response rate of 15%, the sample size is increased to 630.

3.4 Inclusion and Exclusion Criteria

All students who give oral informed consent and willingness to participate in the study will be eligible to participate in the study. However, students (regardless of age) who are less than two months on school grounds prior to the commencement of study data collection will be excluded from this study.

3.5 Sampling Technique

A simple random sampling technique will be used in this study.

3.6 Ethical Considerations

Ethical approval will be obtained from the University of Benin Ethical Review Board.

3.7 Data and Sample Collection

Five millilitres of blood will be aseptically collected from each student by venipuncture of the cubital vein using sterile disposable vacutainer blood collection needles and bottles. Samples will be placed in plain tubes, and the sera will be separated into sample vials and stored at -20°C until analysis. Serum samples will be analysed for the presence of HBsAg using a rapid lateral flow chromatographic immunoassay kit. The test was carried out and interpreted according to the manufacturer's instructions. A pretested structured interviewer-administered questionnaire adapted from the national Hepatitis survey will be used for data collection. The questionnaire includes both closed and open-ended questions and will be sectionalised into socio-demographic characteristics, risk factors associated with HBV infection and vaccination history. The laboratory test results for participants will be anonymously linked to their questionnaire information through unique identifiers.

3.8 Data Analysis

Multivariate analysis will be performed on factors significantly associated with HBV seropositivity for possible confounding and effect modification—chi-square tests for a significant relationship between the dependent and independent variables. HBV serological marker patterns were interpreted using the WHO HBV markers algorithm. P-values were considered significant at < 0.05, and odds ratios were reported with a 95% confidence interval. A positive HBsAg test will be considered evidence of HBV infection (chronic carrier state or infection) and used to calculate the prevalence.

CHAPTER FOUR

RESULTS

Table 4.1: Shows the characteristics and percentage frequency of students from the different faculties surveyed, the age category of the students, sex, level of study, state of residence (not same as state of origin) and blood group.

Table 4.2: Shows the result of the rapid diagnostic tests outcome for HBV and the percentage occurrence of students positive or negative to HBV. From the results garnered, it is observed that out of the 93 students tested, 93 (100%) of the students tested negative to HBV while only no (0%) students were positive to HBV.

Table 4.3: Sociodemographic characteristics of the study participants in relation to HBV positivity and risk factors

Figure 4.1: A graph showing the density and age distribution of student infected with HBV

Table 4.1: Baseline characteristics of the study participants (N=328)

Characteristics	Frequency (%)
Faculty	
Life Sciences	46 (14.0)
Engineering, Physical Sciences, and Applied Sciences	62 (18.9)
Education, Management Sciences, and Environment Sciences	66 (20.1)
Social Sciences, Arts, and Law	61 (18.6)
Medicine, Basic Medical Sciences, Dentistry, Nursing and Agriculture	93 (28.4)
Mean (SD) age, year	21.6
Age Category, year	
16-20	131 (39.9)
21-25	167 (50.9)
26-59	24 (7.3)
Missing	6 (1.8)
Sex	
Female	175 (53.4)
Male	153 (46.7)
Level of study	
100 level	110 (35.5)

Table 4.1 continued

200 level	60 (18.3)
300 level	60 (18.3)
400 level	78 (23.8)
500-600 level	19 (5.8)
Missing	1 (0.3)
State of residence (*not same as state of origin***)**	
Edo	149 (45.4)
Abia	3 (0.9)
Abuja	6 (1.8)
Akwa-Ibom	1 (0.3)
Anambra	11 (3.4)
Bayelsa	2 (0.6)
Delta	40 (12.2)
Enugu	3 (0.9)
Imo	1 (0.3)
Kaduna	1 (0.3)
Lagos	76 (23.2)
Ogun	9 (2.7)
Ondo	9 (2.7)
Osun	2 (0.6)
Oyo	5 (1.5)
Rivers	4 (1.2)
Missing	6 (1.8)

Table 4.1 continued

Self-reported blood group	
O	196 (59.8
A	51 (15.6)
B	27 (8.2)
AB	25 (7.6)
Unknown	29 (8.8)

Table 4.2: Rapid diagnostic test outcome for HBV test

RDT HBV test	Frequency
Negative	93 (100.0)
Positive	0 (0.0)
Total	93 (100.0)

Table 4.3: Sociodemographic characteristics of the study participants in relation to HBV positivity and risk factors

Characteristics	Frequency
Age category, year	
16-20	45 (48.4)
21-25	39 (41.9)
26-59	9 (9.7)
Sex	
Female	48 (51.6)
Male	45 (48.4)
Level of study	
100 level	41 (44.1)
200 level	11 (11.8)
300 level	9 (9.7)
400 level	20 (21.5)
500-600 level	12 (12.9)
Use of local manicure	
No	92 (98.9)
Yes	1 (1.1)
Ownership of personal clipper	
No	72 (77.4)
Yes	21 (22.6)
Sharing of sharp object	
No	79 (85.0)
Yes	14 (15.0)

Table 4.3 continued

Piercing on the body (other than earrings)	
No	89 (95.7)
Yes	4 (4.3)
Tattoo on the body	
No	92 (98.9)
Yes	1 (1.1)
Alcohol intake	
No	69 (74.2)
Yes	24 (25.8)
Blood transfusion	
No	91 (97.9)
Yes	2 (2.2)
Sexually active	
No	48 (51.6)
Yes	27 (29.0)
Prefer not to say	18 (19.4)
Previous Hepatitis B test	
No	89 (95.7)
Yes	4 (4.3)
Hepatitis B vaccination status	
No	61 (65.6)
Yes	1 (1.1)
Not sure	31 (33.3)

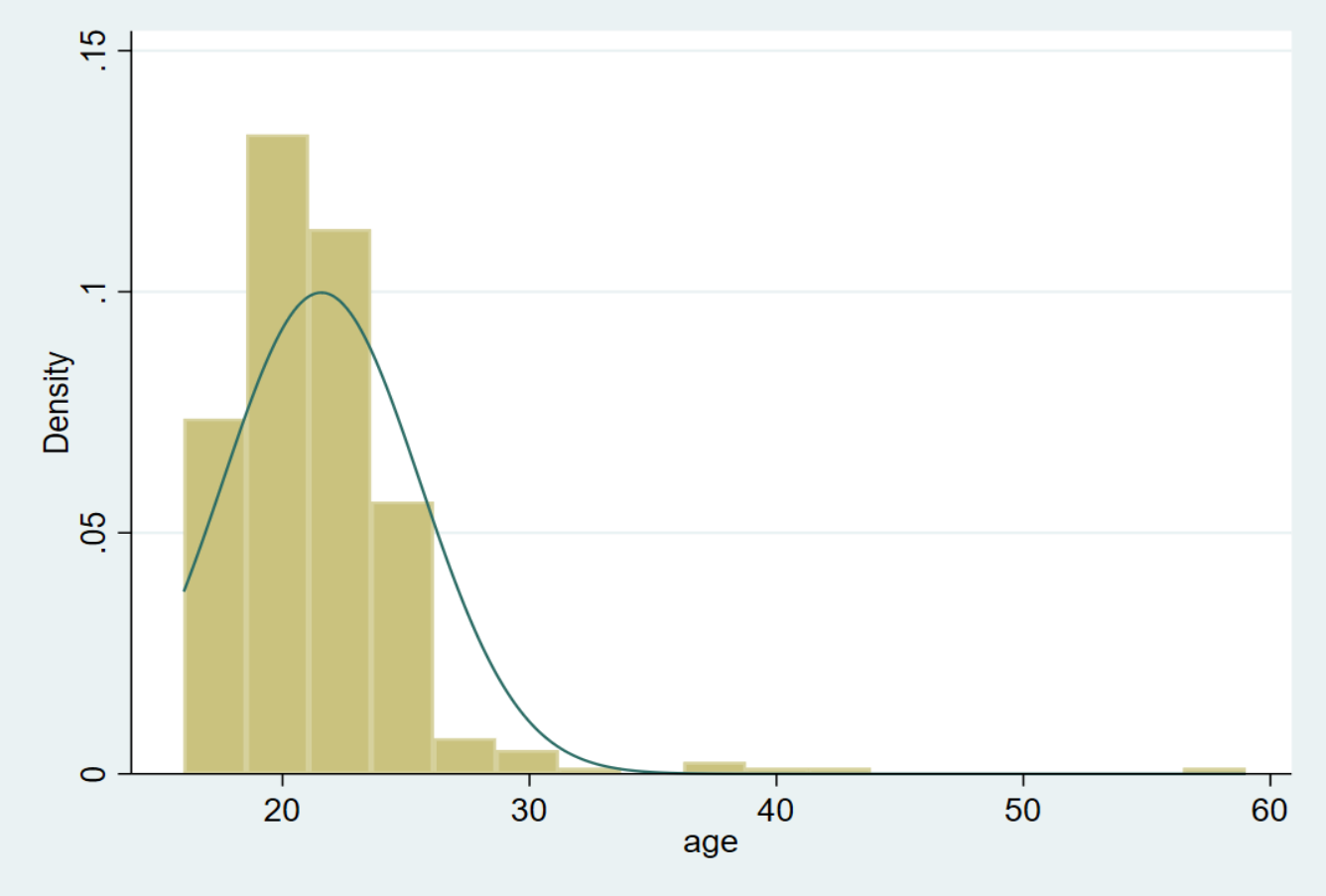

Figure 4.1: A graph showing the density and age distribution of student infected with HBV

CHAPTER FIVE

5.1DISCUSSION AND CONCLUSION

Knowledge about Hepatitis B Virus infection compare to the sex of the participants showed that there is no relationship between the knowledge of HBV infection and gender. Also, the knowledge of hepatitis B virus and age is statistically significant. That is, the older age are more expose to the

knowledge of hepatitis B virus than the younger ones. That is, there is significant difference in the response among the different age groups responding to the knowledge of HBV. The increase in the HBV infection as reported in studies conducted in other faculties could be as result of lack of awareness of HBV infection.

The knowledge about the vaccination as a mean of preventing occurrence of HBV

infection was not influenced by the gender of the participants. Also, it was observed that there was adequate awareness of the vaccination against HBV in this study. Though there was few record of vaccinated students due to lack of available vaccination center around the university. This is further corroborated by the report that suggested a decline in the prevalence of hepatitis B particularly amongst children as a result of inclusion of Hepatitis B vaccine in the routine schedule of immunization.

My result is in variance with the prevalence rate as recorded in the internally displaced persons in Edo state (Micheal *et al*., 2020). This can be due to better awareness of the HBV in campus then there is in the displaced community, also better personal hygiene and possession of personal clippers and sharp objects amongst student in campus. Results from my study is also in varience with the prevalence rate recorded among blood donors in a known tertiary institutional hospital in Edo state (Izevbuwa *et al.*, 2021). This variance can be as a result of the mean age and level of

my study group which is 21 and 100level. This group appears to less sexually active and as a result, reduces the chances of contracting the virus Hepatitis B.

Also, the use of a rapid test kit instead of a more sensitive method of testing such as ELISA (Enzyme-linked Immunoassay) could be a reason for the low prevalence rate recorded in the study.

5.2 CONCLUSION

There is no doubt that the hepatitis virus is endemic in Nigeria and also among students. Though a low prevalence rate was observed in the study, nevertheless, there must be continuous awareness of among student of various faculties in different campuses concerning the modes of transmission and its prevention in reducing the weight of both Hepatitis B. vaccination of Hepatitis B should be incorporated in Health center in both campuses.

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ABBREVIATION

HBV: Hepatitis B Virus

HCC: Hepatocellular Carcinoma

ELISA: Enzyme-linked Immunosorbent Assay

RDT: Rapid Diagnostic Test

PCR: Polymerase Chain Reaction

CI: Confidence Interval

APPENDIX

QUESTIONNAIRE

Prevalence of HBV and HCV amongst undergraduate students

Date_____

14. Do you have tribal marks/ scarification?

Yes No

15. Have you ever had a blood oath?

Yes No

16. Do you patronize local manicure/pedicure vendors?

Yes No

17. Do you use a personal clippers for shaving or hair cut?

Yes No

18. Do you share personal belongings with others e.g., toothbrush, towel?

Yes No

19. Do you share sharp objects e.g., Needles Razor blades clippers?

Yes No

20. Do you have body piercing?

Yes No

21. Do you have Tattoo on your body?

Yes No

22. Do you share needles with partners? Yes

No

23. Are you sexually active? No Yes

If yes to question 23:

- How many sexual partners have you had so far? _____

- Have you had sexual intercourse with someone who is not your regular partner within the last 6 months? Yes No Not sure
- Do you use condom when having sex? Always No Sometimes
- Your sexual partners are? Male only Female only Both
- Have you ever experienced painful urination and discharge? Yes No Don't know
- Have you ever had sex while under the influence of illicit drugs or alcohol Yes No

24. Do you take alcohol?

Yes No

25.Do you smoke weed, Igbo, colos, tramadol and cocaine?

Yes No

26. Have you ever received a blood transfusion?

Yes No

27. Have you ever been tested for Hepatitis B? Yes No

28. If yes what was your result?

Negative Positive

29. Have you ever been tested for Hepatitis C

30. If yes what was your result?

Negative Positive

31. Have you ever received hepatitis B vaccine?

Yes No Not Sure

32. If yes, which year did you receive the vaccine (year)? ……

33. How many doses of the vaccine did you receive?

One dose Two doses Three doses More than 3 doses

Not sure

Result

HBV _____

HCV_____

CONSENT FORM

Informed Consent Form A study on the prevalence of Hepatitis B and its associated factors among university students in Benin City, Nigeria

Hello! My name is_____ ___. I would like to invite you to take part in this study about Helicobacter pylori among university students in Benin City. Hepatitis B virus is the causative agent for Hepatitis B. This is part of our undergraduate final-year project. You are invited to take part in this study but taking part in this study is voluntary. Study Procedures If you take part in this study, we will ask you a few questions concerning yourself, including questions related to your faculty and department of study. In addition, we will take blood, about 2 ml, to do a rapid diagnostic test for the bacterium. You will get the result on the spot. The interview and test will take about 30-40 minutes. Confidentiality The information will be collected on paper. The information is stored securely and can only be accessed by approved study staff. The interview will take place in private. Everything you tell us is strictly confidential. Your identifying information will never be used in any reports. All information reported from this study will not be able to be linked to you. Potential Risks None is expected, other than the time you will spend in participating in the collection of data for the study. Potential Benefits You may or may not benefit by taking part in this study. If you take part in this study, you may help other students in the future as we would have identified the students most at risk of developing Hepatitis B, and will improve our understanding of how to implement interventions targeting the health condition overall. However, as a token of appreciation for your time, we shall provide light refreshments. Costs to Participate It will not cost you anything to take part in this study other than your time. Confidentiality and Access to

your Health Information Access to the information you provide will be limited to persons involved with this study and will be protected in a secure place. Efforts will be made to protect your information and your answers to the interview questions. A unique number will be used instead of your name to identify your personal information and the answers you give. Only study staff can use this number to link your responses to you. Any answers included in the final report will not have your name or personal information on it. Contacting us If you have additional questions or any concerns, please contact the project supervisor: Dr. Kelly Elimian Department of Microbiology, Faculty of Life Sciences Phone: 08056679826 /08155465671 Do you want to ask me anything about the study? 2 Consent Statement I have read this form and/or someone has read it to me. I was encouraged to ask questions and given time to ask questions. Any questions that I had have been answered satisfactorily. I agree to voluntarily take part in the study. I know that after choosing to take part in the study, I may withdraw at any time. I have been offered a copy of this consent form. 1. Do you agree to participate in the study? 'YES' means that you agree to do the interview. 'NO' means that you will NOT do the interview. _____Yes _____No Participant signature _____ Date: ___/___/___ DD/MM/YYYY Printed name of participant _____ Participant ID number _____ Signature of person obtaining consent_____ _____ Date: ___/___/___ DD/MM/YYYY