SEROPREVALENCE OF HEPATITIS B VIRUS INFECTION IN PREGNANT WOMEN IN KADUNA, KADUNA STATE. NIGERIA.

BY

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DEPARTMENT OF MICROBIOLOGY
FACULTY OF SCIENCE
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ZARIA, NIGERIA.

OCTOBER, 2006

DECLARATION

I declare that the work in this thesis entitled "Seroprevalence of Hepatitis B Virus Infection in Pregnant Women in Kaduna, Kaduna State" is original and has been performed by me in the Department of Microbiology, Ahmadu Bello University, Zaria, under the supervision of Professor A.A. Ahmad and Dr. E.D. Jatau, both of the Department of Microbiology.

The information derived from literature has been duly acknowledged in the text and a list of references provided. No part of this thesis was previously presented for another degree or diploma at any university.

EKWUDU O'MEZIE	_____	October 20, 2006
Name of Student	Signature	Date

CERTIFICATION

This thesis, entitled "Seroprevalence of Hepatitis B Virus Infection in Pregnant Women in Kaduna, Kaduna State" By Ekwudu O'mezie, meets the regulations governing the award of the degree of Masters of Science (Microbiology) of Ahmadu Bello University, Zaria, and is approved for its contribution to knowledge and literary presentations.

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DEDICATION

This thesis is dedicated to Ekwunife and Amuche, my biological father and mother. There are no better parents.

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I thank the Shepherd, Lord Jesus Christ, for being everything at every time. I am indebted with my life.

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ABSTRACT

This research work was undertaken to determine the seroprevalence of hepatitis B virus infection in pregnant women in Kaduna, Kaduna state, Nigeria. The study population was randomly drawn from women attending antenatal clinics in the three general hospitals in Kaduna metropolis i.e. Barau Dikko Specialist Hospital, Yusuf Dantsoho Memorial Hospital and Gwamna Awan General Hospital. Blood specimen and personal data were obtained from two hundred and seventy four pregnant women attending the clinics. For this study, three Hepatitis B Virus infection serological markers : anti-HBs, anti- HBc and HBeAg, were employed using ELISA. The three assay kits used were AutoBio Anti-HBs Plus, AutoBio Anti-HBc Plus and AutoBio HBeAg all manufactured by AutoBio Immundiagnostics U.S.A. From the study population of 274 pregnant women screened for the HBV serological markers, 77 (28.1%) were seropositive for anti-HBs, and 57(20.8%) and 6(2.2%) were seropositive for anti-HBc and HBeAg respectively. There was no statistical association between the presence of the hepatitis B virus infection markers and marital status, educational status, age and number of sexual partners. The seroprevalence rate for the HBV infection markers increased with increase in educational status, the tertiary group did not follow this trend. The distribution across age revealed that the frequency of the anti-HBs infection markers was highest (35.3%) in the 20-24 years age group, and for anti-HBc the peak was recorded in the 15-19 years age group, this is likely due to the sexual activity of this age range. Married women accounted for all the 6 (2.4%) that were seropositive to HBeAg. This survey confirms the presence of hepatitis B virus infection markers in Kaduna and also illustrates the percentage that lacks the serological markers which makes them susceptible to HBV infection. This study provided a baseline for further research on the transmission, surveillance and intervention strategies of HBV in Kaduna and Nigeria as a whole. Vaccination of all children against hepatitis B virus irrespective of the serological status of their mothers and routine screening of pregnant women for these HBV serological markers are recommended.

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Chapter 1 INTRODUCTION

1.1 Background Knowledge

Clinical and epidemiologic studies began to differentiate among various types of acute hepatitis in the decades following world war ll. The groundbreaking studies of Krugman and colleagues in 1967 firmly established the existence of at least two types of hepatitis, one of which (then called serum hepatitis, and now called hepatitis B) was parenterally transmitted. Blumberg and colleagues (1965) while searching for serum protein polymorphisms linked to diseases identified an antigen (termed Au) in serum from patients with leukemia, leprosy and hepatitis. Prince and coworkers (1964) independently identified an antigen (termed SH), while systematically studying patients with transfusion-associated hepatitis. Further work established that AU and SH were identical. The antigen represented the hepatitis B surface antigen (HBsAg). These studies made possible the serologic diagnosis of hepatitis B and opened up the field to rigorous epidemiologic and virologic investigations (Prince *et al.,*1964.,Blumberg *et al.,*1965.,Prince, 1968., Krugman *et al.,*1970.,Ganem and Prince, 2004).

Hepatitis B virus (HBV) is transmitted mainly by the percutaneous routes (e.g., needle sharing, acupuncture, ear piercing, tattooing) and through very close personal contact involving the exchange of blood or secretions (e.g., sexual contact, childbirth). It is also transmitted through other body fluids including semen and saliva. The virus starts to replicate within three days of its acquisition, symptoms may not be observed for 45

days or longer, depending on the infectious dose, the route of infection, and the person (Kao and Chen, 2002). The virus replicates in hepatocytes with minimal cytopathic effects, after which infection proceeds for a relatively long time without causing liver damage (i.e., elevation of liver enzyme levels) or symptoms (Blum *et al;*1989., and Murray *et al.,*1998).

The clinical course of hepatitis B virus infection is complex and is influenced by several factors classified into viral factors and host factors. The viral factors include level of hepatitis B virus replication (viral load), hepatitis B virus genotype, and mutations in viral genome. The host factors include age of acquisition of infection, immune status, concurrent infection with other hepatotropic viruses, and alcohol intake (Aggarwal and Ranjan, 2004). Overall, chronic hepatitis progresses to end stage liver disease in 15 to 40 percent of patients (Liaw *et al;* 1988).

For a successful infection, Hepatitis B virus requires an infectious source, a susceptible host, and an established route of transmission (Kao and Chen, 2002). The predominant routes of transmission in various locations vary according to the endemicity of HBV infection. The virus is 100 times more infectious than human immunodeficiency virus (HIV) and unlike HIV, it can live outside the body in dried blood for longer than a week (Ott, 1999; Kirchner and Lin, 2004).

The most efficient way to control hepatitis B is to prevent individuals from contracting it rather than treat the infections. Two main approaches lead to achieving this goal:

interrupting the virus at the various routes of transmission and immunizing susceptible host. Although immunization is more effective, public health measures should include both approaches. Prevention strategies must include Hepatitis B vaccination of high risk groups and all new born infants, screening of blood and blood products before transfusion, using universal precautions in healthcare settings like avoid sharing needle among injecting drug users and promote safe sexual practices (Chen, 2005).

In the early 1980s, vaccination against hepatitis B became available, first with plasma-derived vaccines and then with recombinant DNA vaccines. All these vaccines are safe and have a protective efficacy of approximately 95%. Despite this success, the duration of protection afforded by hepatitis B vaccination is unknown (Chen, 2005). Detecting persistent antibody is the easiest but not necessarily the most accurate way to measure persistent protection (West *et al;* 1996.,Chen, 2005).

The implications of hepatitis B in pregnancy cannot be over emphasized. Hepatitis B virus can be transmitted from mother to child *in utero*. The sequelae of infection in children can be phenomenal (Owusu-Boateng, 2002). The exact mechanism of perinatal transmission is not clear. It is unusual for infants to be infected *in utero*, cord blood is usually negative for hepatitis B markers; however occasional intrauterine infection does occur (Ko *et al;*1994).

The serological markers that define the state of hepatitis B infection include Hepatitis B surface antigen (HBsAg), antibody to hepatitis B surface antigen (anti-HBs), hepatitis B core antigen (HBcAg), antibody to hepatitis B core antigen (anti-HBc),

hepatitis B e antigen (HBeAg), antibody to hepatitis B e antigen (anti-HBe). The presence of anti-HBs indicates past infection and immunity to HBV, it also means presence of passive antibody from hepatitis B immunoglobulin (HBIG), immune response from HBV vaccine. Antibody to hepatitis B core antigen (anti-HBc) indicates infection with HBV at some undefined time in the past, they could be asymptomatic carriers. Hepatitis B e antigen is associated with the nucleocapsid; it circulates as soluble antigen and indicates ongoing viral replication (Brooks *et al;*2004*)*. These markers when assayed for in pregnant women will assess the possibility of transmission to neonates. Women who are HBsAg negative may be vaccinated safely during pregnancy (Lin *et al;*2004) . No current evidence supports the use of cesarean delivery to reduce vertical transmission of HBV (Lin *et al;* 2004). Vaccination of hepatitis B carrier mothers, which is standard practice in many endemic areas of the world has prevented more than 90% of this transmission (Brooks *et al;*2004*,* Lin *et al;* 2004).

1.2 Statement of Research Problem

Hepatitis B virus infection is a global public health problem with approximately 400 million people chronically infected (Lin *et al*; 2004). An estimated one third of the world's population has serologic evidence of past infection, and the virus causes more than I million deaths annually (Zuckerman and Zuckerman, 1999).

In Sub-Saharan Africa HBV, infection usually is acquired through maternal-fetal transmission or in early childhood leading to a high prevalence of chronic infection

(Aggarwal, 2004).A proportion of people infected with hepatitis B Virus (5%-10%) among adults progress to chronicity, defined as persistence of infection for more than six months (Aggarwal and Ranjan, 2004). The rate of chronicity is much higher among neonates born to hepatitis B infected mothers and children because they have an immature immune system. Ninety percent of infants infected perinatally progress to chronic infection. Progression to chronic HBV infection occurs in 25 to 30 percent of persons infected before five years of age, and in 3 to 5 percent of those infected later in childhood or as adults (Lok, 2001). Once the infection becomes chronic, HBV or part of the viral genome usually persists a lifetime in the liver of the carrier. The carriers are not only reservoirs of the virus but also victims of chronic liver diseases themselves (Kao, 2002; Chen, 2003). Thus, control of HBV infection, especially in terms of preventing chronic carrier status of the virus, is extremely important (Ganem and Prince, 2004).

Moreover, the higher prevalence of chronic infection due to maternal-fetal transmission also translates into higher rates of cirrhosis and cancer in these areas. Treatments are not curative because they rarely produce permanent remission of the disease (Lin *et al*; 2004).

1.3 Justification

Transmission of HBV from mother to child, (vertical transmission), is associated with a greater probability of generating carrier status and consequently of maintaining the chronic infection in the population. Some authors observed that the proportion of

carriers among children born to mothers with a history of HBsAg seropositivity can range between 70% and 90% (Saenz-Gonzalez, 2001; Vazquez-Martinez *et al*; 2003).

In 1981, the prevalence rate of HBsAg carrier state in Nigerian pregnant women was determined to be 11.2% (Ayoola *et al*; 1981). Recent studies that describe the magnititude of the problem in Nigeria are limited. Seroepidemiologic studies for pregnant women carried out revealed a wide range of prevalences for HBV infection markers ranging from 15.8% to 22.0% in Maiduguri (Harry *et al;1994* and Baba *et al;*1999), 2.19% for Benin city (Onakewhor *et al*; 2001), 9.3% for Anambra State (Agbonlahor *et al;* 2004), 10.4% for Gombe (Mustapha and Jibrin, 2004). Screening for hepatitis B virus infection serological markers in all pregnant women bases prevention of perinatal transmission on seroprevalence studies which provide direct outcome measure of program effectiveness (AJPH, 1999).

Seroprevalence studies help to identify those at greatest risk of infection and therefore assist in effectively targeting prevention strategies (AJPH, 1999).

Kaduna, the commercial and industrial centre of Northern Nigeria with a population of approximately 1,580,000 has no prevalence of HBV infection markers.

1.4 Aims and Objectives

The objective of this study is to:

1. To determine the seroprevalence of hepatitis B infection in pregnant women in Kaduna, as well as the risk factors associated with its occurrence.

1.5 Research Question

This work was therefore designed to answer this question:

1. What is the baseline hepatitis B seroprevalence rate among pregnant women in Kaduna, Kaduna state?

Chapter 2

LITERATURE REVIEW

2.1 Hepadnaviridae

Hepadnaviridae comprises of a family of partially double stranded DNA viruses of 42nm diameter consisting of a lipid bilayer envelope surrounding an icosahedral nucleocapsid (Brooks *et al;2004*).

Members of the family Hepadnaviridae are known as hepadnaviruses. They are associated with liver infections. They are acid-sensitive. They replicate through an RNA intermediate (which they transcribe back into DNA using reverse transcriptase). Most hepadnaviruses will only replicate in specific hosts and this makes experiment using *in vitro* methods very difficult (Brooks *et al;* 2004). The virus binds to specific receptors on cells and the core particles enter the host cell's cytoplasm. This is then translocated to the nucleus where the partial double stranded DNA is "repaired" to form a completed circular DNA . This then undergoes transcription by the host cell RNA polymerase and the transcript is translated by host cell's ribosomes. New viral particles are formed which may bud from the pre-Golgi membranes acquiring HBsAg-containing envelopes, and may exit the cells as virions or may be reimported into the nucleus and initiate another round of replication in the same cell (Wiesland and Chisari, 2005).

There are two genera in this family:

Genus Orthohepadnavirus and genus Avihepadnavirus, Hepatitis B virus is a type species of the Orthohepadnavirus (Ganem, 1991; Ganem and Schneider, 2001 **)**

Hepatitis B virus (HBV) is the prototype noncytopathic member of the hepadnaviridae (hepatotropic DNA virus) family. Hepadnaviruses have a strong preference for liver cells, but small amounts of hepadnaviral DNA can be found in the kidney, pancreas, and mononuclear cells. However, infection at these sites are not linked to extrahepatic diseases (Korba *et al*; 1988; Marion, 1988).

2.2 Virologic Features

2.2.1 Hepatitis B Virus Structure

HBV virions are double–shelled particles, 40 to 42nm in diameter, with an outer lipoprotein envelope that contains three related envelope glycoproteins (or surface antigens). Within the envelope is the viral nucleocapsid, or core. The core contains the viral genome, a relaxed circular, partially duplex DNA of 3.2kb, and a polymerase that is responsible for the synthesis of viral DNA in infected cells (Summers *et al;1975)*. DNA sequencing of many isolates of HBV has confirmed the existence of multiple viral genotypes, each with a characteristic geographic distribution (Kao, 2002). In addition to virions, HBV-infected cells produce two distinct subviral lipoprotein particles: 20-nm spheres and filamentous forms of similar diameter. These HBsAg particles contain only envelope glycoproteins and host derived lipids and typically outnumber virions by 1000:1 to 10,000:1 (Ganem and Schneider, 2001).

2.2.2 Viral Genes And Proteins

The HBV genome has only four long open reading frames. The pre S-S (presurface-surface) region of the genome encodes the three viral surface antigens by differential initiation of translation at each of three in-frame initiation codons (Ganem and Schneider, 2001; Ganem, 2001). The most abundant protein is the 24- kD S protein (which is known as HBsAg). Initiation at the most upstream start codon generates the M (or preS2) protein, the function of which is unknown. It also yields the L (or preS1) protein, which is thought to play key roles in the binding of the virus to host cell receptors (Klingmuller and Schaller, 1993) and in the assembly of the virion and its release from the cell (Bruss and Ganem,1991).The pre C-C (precore-core) region encodes hepatitis B core antigen (HBcAg) and hepatitis B e antigen (HBeAg). These two proteins are also derived by alternative initiation of translation at two in-frame start(AUG) codons. The internal AUG directs the synthesis of the 21-kD core protein, the structural polypeptide of the viral capsid, whereas the upstream AUG directs production of the 24- kD pre-c protein. The pre-c region encodes a signal sequence, which directs the polypeptide chain into the secretory pathway. As the chains traverse the Golgi complex, cleavage by cellular proteases generates HBeAg, a 16-kD fragment that is secreted into the blood. HBeAg plays no role in viral assembly, and its function is not clear. It is not required for viral replication; mutants bearing chain-terminating lesions within the pre-c region replicate well in culture and, in fact, arise frequently during natural infection (Okamoto *et al;* 1990).

The viral polymerase (P) coding region is specific for the viral polymerase, a multifunctional enzyme involved in DNA synthesis and RNA encapsidation. The X

open reading frame encodes the viral X protein (HBx), which modulates host cell signal transduction and directly and indirectly affect host and viral gene expression (Ganem, 2001). X – Protein activity is absolutely required for the *in vivo* replication and spread of the virus (Zoulim *et al;* 1994).

2.3 Viral Replication Cycle

The cardinal feature of the HBV replication cycle is the replication of the DNA genome by reverse transcription virions bind susceptible cell-surface receptors, IgA receptors on liver cells. Following of an RNA intermediate (Summers and Mason, 1982; Wang and Seeger, 1993). Incoming HB infection, the 3.2kb partially double-stranded DNA HBV genome is delivered to the nucleus and converted into a covalently closed circular double-stranded HBV DNA (ccc DNA) molecule that serves as a transcriptional template for the host RNA polymerase II machinery, which produces four capped and polyadenylated mRNAs that encode the viral core and envelope structural proteins and the precore, polymerase, and X non structural viral proteins. One of the major HBV transcripts is a 3.5-kb greater than genome length RNA that is translated to produce the viral core and polymerase proteins and also serves as a pregenomic RNA that is encapsidated with the polymerase by the core protein in the cytoplasm of the hepatocyte. Viral replication occurs entirely within these capsids by reverse transcription of the pregenomic RNA to produce a single-strand DNA copy, which serves as the template for second- strand DNA synthesis, producing a circular double stranded DNA. Viral capsids containing double stranded DNA traffic either to the nucleus where they amplify the viral ccc DNA genome to

maintain stable intranuclear pool of transcriptional templates or to the endoplasmic reticulum, where they acquire the viral envelope proteins, bud into the lumen, and exit the cell as virions that can infect other cells (Seeger and Mason, 2000; Ganem and Prince, 2004).

2.4 Pathogenesis of Hepatitis B

The HBV replication cycle is not directly cytotoxic to cells (Chisari and Ferrari, 1995). This fact accords well with the observation that many HBV carriers are asymptomatic and have minimal liver injury, despite extensive and ongoing intrahepatic replication of the virus (de Franchis *et al;* 1993). It is now thought that host immune reponses to viral antigens displayed on infected hepatocytes are the principal determinants of hepatocellular injury. This notion is consistent with the clinical observation that patients with immune defects who are infected with HBV often have mild acute liver injury but high rates of chronic carrier status (Ganem and Prince, 2004). The immune responses to HBV and their role in the pathogenesis of hepatitis B are incompletely understood. Correlative clinical studies show that in acute, self-limited hepatitis B, strong T-cell responses to many HBV antigens are readily demonstrable in the peripheral blood (Chisari and Ferrari, 1995).

These responses involve both major-histocompatibility complex (MHC) class II-restricted, CD4+ helper T cells and MHC class I – restricted, CD8+cytotoxic T lymphocytes. The antiviral cytotoxic T-lymphocyte response is directed against multiple epitopes within the HBV core, polymerase, and envelope proteins; strong

helper T-cell responses to C and P proteins have also been demonstrated in acute infection. By contrast in chronic carriers of HBV, such virus-specific T-cell responses are greatly attenuated, at least as assayed in cells from the peripheral blood. However, antibody responses are vigorous and sustained in both situations (although free antibodies against HBsAg [anti-HBs antibodies] are not detectable in carriers because of the enormous circulating HBsAg). This pattern strongly suggest that T-cell responses, especially the responses of cytotoxic T lymphocytes, play a central role in viral clearance (Baron *et al;* 2002). The mechanism by which cytotoxic T lymphocytes kill liver cells and cause viral clearance have been incisively investigated in transgeneic mice that express viral antigens or contain replication-competent viral genomes in the liver (Chisari, 1996).These mice harbor HBV genes in their germ-line DNA, they are therefore largely tolerant to HBV proteins, and accordingly, clinically significant liver injury does not develop. However, if antiviral cytotoxic T lymphocytes to syngeneic animals are transferred into such mice, acute liver injury with many features of clinical hepatitis B develops (Guidotti *et al;* 1996). The number of hepatocytes killed by direct engagement between cytotoxic T lymphocytes and their targets in the mice models is very small and clearly insufficient to account for most of the liver damages .This suggests that much of the injury is due to secondary antigen-non specific inflammatory responses that are set in motion by the response of the cytotoxic T lymphocytes. Presumably, much of the damage occurring is due to cytotoxic by-products of the inflammatory response, such as tumor necrosis factor (TNF), free radicals, and proteases. Other immune-cell populations, notably natural killer T cells, probably also contribute to liver injury (Baron *et al;* 2002). Recent

experiments suggest that some of the inflammatory by-products, such as interferon IFN- γ and TNF-α can have antiviral effects that do not involve killing the target cells. When cytotoxic T-lymphocytes are transferred to mice that bear replicating HBV, viral DNA and RNA throughout the liver rapidly disappear throughout the liver, even from viable, uninjured hepatocytes an effect that can be blocked by the administration of antibodies to TNF-α and IFN-γ (Guidotti, *et al;* 1996).

Such non-cytocidal antiviral effects may be important for viral clearance in natural infection. Infact, cytokine-release triggered by unrelated hepatic infections in HBV – transgenic mice can have the same effect (Cavanaugh, *et al;* 1998). This phenomenon may explain the suppression and occasional clearance of chronic HBV infection in patients with super imposed acute hepatitis caused by unrelated viruses.

2.5 Natural History

Primary HBV infection in susceptible (non immune) hosts can be either symptomatic or asymptomatic. The latter is more common than the former, especially in young children. Most primary infections in adults, whether symptomatic or not, are self-limited, with clearance of virus from blood and liver and the development of lasting immunity to reinfection (Wright and Lau, 1993). However, some primary infections in healthy adults (generally less than 5 percent) do not resolve but develop into persistent infections. In such cases, viral replication continues in the liver and there is continual viremia, although the titers of virus in the liver and blood are variable. Persistent HBV infection may be symptomatic or asymptomatic. People with subclinical persistent infection, normal serum aminotransferase levels, and normal or near normal findings

on liver biopsy are termed asymptomatic chronic HB carriers; those with abnormal liver function and histologic features are classified as having chronic hepatitis B (Ganem et al;2001). Cirrhosis, a condition in which regenerative nodules and fibrosis co-exist with severe liver injury, develops in about 20 percent of people with chronic hepatitis B. The resulting hepatic insufficiency and portal hypertension make this process one of the most feared consequences of chronic HBV infection(Ganem,2004). HBV is worldwide in transmission. Transmission modes and response to infection vary, depending on age at time of infection (Brooks *et al;2004*)

2.6 Primary Infection

In primary HBV infection, HBsAg becomes detectable in the blood after an incubation period of 4 to 10 weeks, followed shortly by antibodies against the HBV core antigen (anti-HBC antibodies), which early in infection are mainly of the IgM isotype. Viremia is well established by the time HBsAg is detected. Titres of virus in acute infection are very high, frequently 10^9 to 10^{10} virions per milliliter (Ribeiro *et al;* 2002) when circulating HBeAg becomes detectable in most cases. Studies of chimpanzees and other animals with primary hepadnaviral infection show that 75 to 100 percent of hepatocytes are infected when this antigen is evident (Kajino, 1994). Thus, it is not surprising that epidemiologic studies consistently show high rates of both vertical and horizontal transmissibility during acute HBV infection (Koff *et al*; 1977). Figure 2.1 illustrates the typical serologic course in acute hepatitis B virus infection with recovery (Chen, 2005). When liver injury occurs in primary infection, alanine aminotransferase levels do not increase until after viral infection is well

established, reflecting the time required to generate the T-cell mediated immune response that triggers liver injury. Once this response is under way, titers of virus in blood and liver begin to drop. The fact that infection can be cleared from virtually all

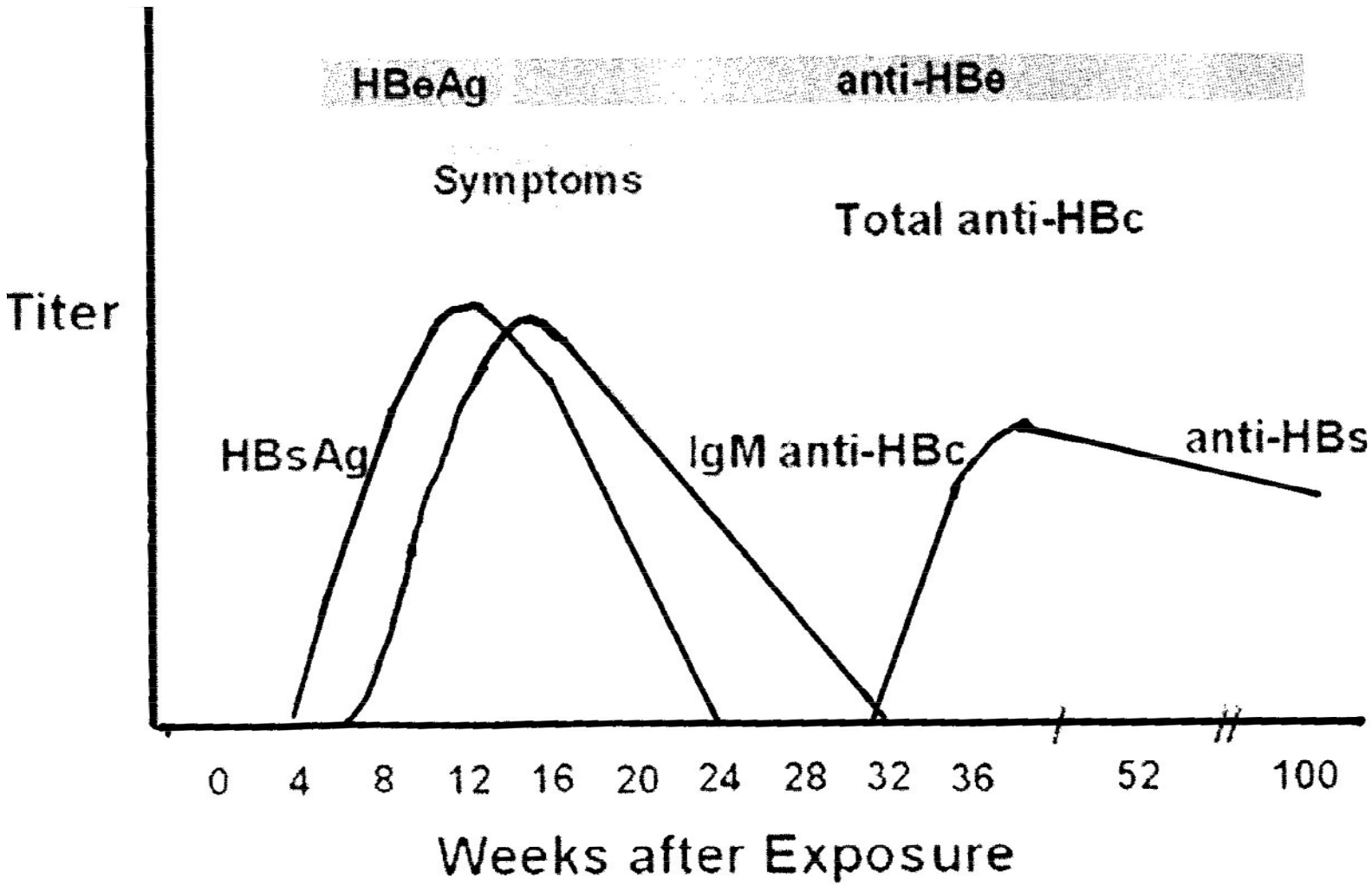


Figure 2.1: Typical Serological Course of Acute HBV Infection with Recovery (Chen, 2005).

hepatocytes without massive hepatic destruction (in most cases) is a testament to the extraordinary power of the noncytolytic clearance mechanisms. With clearance of the infection, the viral antigens HBsAg and HBeAg disappear from the circulation, and free anti-HBs antibodies become detectable (Marcellin *et al;* 2003).

Surprisingly, in self-limited infection, as defined by the disappearance of the viral antigens and the appearance of anti-HBs antibodies, low levels of HBV DNA in the blood may persist for many years, if not for life (Prince *et al*; 2001). It is not known whether this DNA contains the entire HBV genome, or even whether it is contained in virions. However inoculation of serum from three subjects with persistent HBV DNA into chimpanzees has not led to documented infectivity (Prince *et al*; 2001)

2.7 Persistent Infection

In persistent HBV infection, the early events are similar to self-limited infection, but HBsAg remains in the blood and virus production continues, often for life. However, levels of viremia in chronic infection are generally substantially lower than during primary infection, although they can vary considerably from person to person. High titers of HBV in the blood are often indicated by continued presence of HBsAg. Typically, there are 10^7 to 10^9 virions per millimeter in the blood in such cases, which are highly infectious, those with anti-HBe antibodies, have somewhat lower levels of viremia (Weinberger, *et al*; 2002). Figure 2.2, illustrates the typical serologic course of progression to chronic hepatitis B virus infection (Chen, 2005).

In most cases, HBsAg remains detectable for life, while titer of viral DNA tend to decline over time. With the passage of time, there is also a tendency for HBeAg to

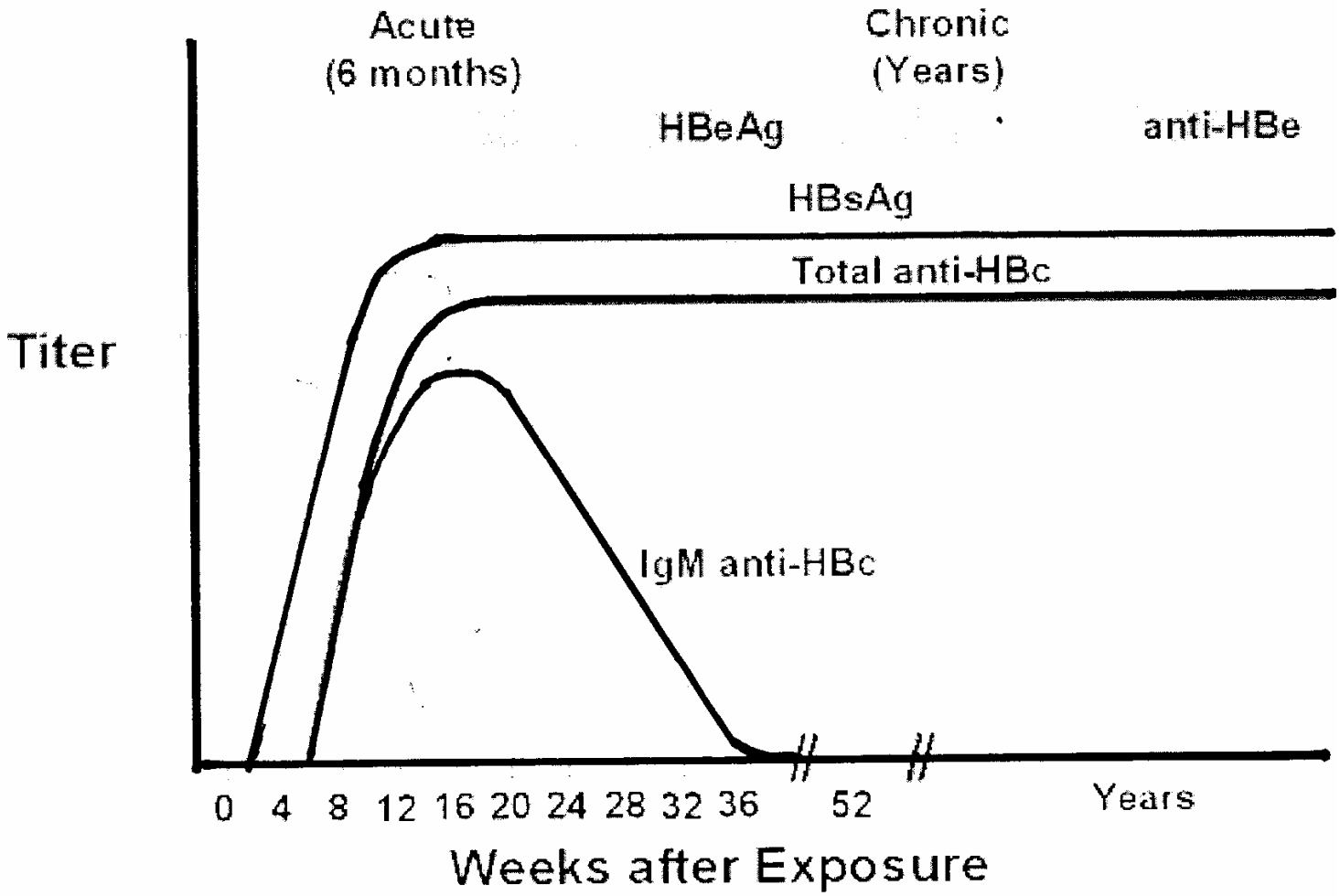


Figure 2.2: Typical Serological Course of progression to Chronic HBV Infection (Chen, 2005).

disappear from blood, along with seroconversion to positivity for anti-HBe antibodies – a progression that occurs at a rate of 5 to 10 percent per year in persistently infected people (Ribeiro, *et al;* 2002). Often the disappearance of HBeAg is preceded or accompanied by a transient rise in alanine aminotransferase levels, known as a flare, which suggests that the process reflects immune mediated destruction of infected hepatocytes. Reductions in the level of viremia as great as five orders of magnitude may accompany seroconversion to anti-HBe antibodies (Tedder *et al;* 2002).

Thus, the natural history of HBV persistence suggests that there is an ongoing immune attack on infected cells in the liver – an attack that is usually inadequate to eradicate infection altogether, but that does reduce the number of infected cells and thereby lowers the circulating viral load (Ribeiro *et al;* 2002) .The widely held view that circulating viral DNA disappears when anti-HBe antibodies appear is incorrect; this idea reflects the fact that, for many years, HBV DNA was measured by relatively insensitive hybridization methods with a detection limit of 10^5 to 10^6 virions per milliliter. The advent of polymerase-chain-reaction (PCR) method, has established that at least 70 to 85 percent of people with anti-HBe antibodies have detectable viral DNA in the circulation, typically in the range of 10^3 to 10^5 molecules per milliliter, and sometimes higher (Tedder *et al;* 2002). Although these levels of HBV DNA are relatively low, they are hardly negligible. HBeAg-negative carriers are a heterogenous group. Most of such carriers have low levels of viral DNA, relatively normal levels of alanine aminotransferase, and a good prognosis (de Franchis *et al;* 1993; Sung *et al;* 2002). The virus in many such carriers harbors mutations in the pre C region that prevents the production of HBeAg (Brunetto *et al;* 1991). It has been suggested that

persistently abnormal levels of alanine aminotransferase and elevated levels of viral DNA may denote a subgroup of HBeAg-negative carriers who should receive active antiviral therapy (Lok and McMahon, 2001).

2.8 Hepatocellular Carcinoma

Primary hepatocellular carcinoma is a feature of the natural history of HBV infections. Chronically infected subjects have a risk of hepatocellular carcinoma that is 100 times as high as that for noncarriers; within the HBsAg-positive group, those positive for HBeAg have the highest risk of hepatocellular carcinoma, but even HB carriers with anti-HBe antibodies have a substantial risk of cancer (Yang *et al*; 2002).

Although the role of HBV in provoking hepatocellular carcinoma is undisputed, its cellular and molecular mechanisms remain incompletely understood (Popper *et al;*1987; Fourel *et al;* 1990; Matsubara, 1990; Kim, *et al;* 1991; Laver *et al;* 1992).
An investigation carried out on the prevalence of hepatitis B surface antigen among patients suspected of liver diseases in University of Maiduguri Teaching Hospital, a Nigerian hospital showed that a total of 197 (38%) of 517 patients tested positive for HBsAg, 81(49%) out of 144 with symptoms suggestive of hepatitis, 75(50%) of 149 HCC and 10 (56%) of 18 liver cirrhosis were HBsAg positive (Baba *et al;* 2000) Twice–a-year screening of chronically infected patients with measurements of serum alpha fetoprotein or hepatic ultrasonography, or both, is warranted (Lok, 2001).

2.9 Transmission of Hepatitis B Virus

Hepatitis B virus is spread by contact with blood and blood products or body fluids. Common routes of infection are perinatal (or vertical), intrafamilial transmission among young children in endemic communities, parenteral exposure either through intravenous drug use (IVDU) or occupational exposure. Transmission by blood transfusion is now rare, whereas before screening of donor blood, it was not uncommon. The exact mechanism of perinatal transmission is not clear. It is unusual for infants to be infected in utero. Cord blood is usually negative for hepatitis B serological markers; however, occasionally intrauterine infection does occur. Fetal blood sampling may increase this risk, but amniocentesis does not appear to enhance the risk of intrauterine transmission (Summers *et al;* 1987; Ko *et al;* 1994). Infection presumably occurs at or soon after birth. Perhaps birth trauma is important. Transmission perhaps occurs through breast (feeding) milk, as it is known to carry hepatitis B. Transmission could occur if there was an inapparent breach in the mucosa of the mouth, or during teething (Beasley *et al;* 1997).

Perinatal transmission, in the era before vaccination, was a major mode of neonatal infection in endemic areas. Mothers carrying the hepatitis B e antigen, (i.e. who were carrying actively replicating virus) could transmit the disease agent to their offspring in more than 90% of cases. Mothers carrying antibodies to the HBe antigen (anti-HBe) transmitted disease in about 25% of cases. Vaccination of hepatitis B carrier mothers, which is standard practice in many endemic areas of the world, has prevented more than 90% of this transmission (Summers *et al;* 1987).

Historically, hepatitis B was common in institutions for the care of mentally retarded children. More recently, horizontal transmission has been documented in endemic areas. In Africa, the prevalence of hepatitis B markers increases with age, from age one to five years (Abdool-Karim, 1999; Botha *et al;* 1999). In Africa, transmission is thought to be related to impetigo or scabies in young children who play together. Other possible modes of transmission include an infected parent kissing the cut finger or scraped knee of a young child, and sharing of items of personal hygiene such as toothbrushes between parents and children, (e.g. safety pins which have been in the parent's mouth inadvertently pricking the infant, etc). The extent to which horizontal transmission occurs in Nigeria is unknown. It is likely that as socioeconomic standards rise, transmission of HBV to young children would decrease. Among young black adults transmission is most frequently via sexual contacts or intra venous drug use (IVDU) (Abdool-Karim, 1999).

2.10 Prevention of hepatitis B virus transmission

Maternal-fetal transmission – All pregnant women should be screened for HBV infection markers, so that the mode of delivery does not put the newborn at the risk of acquirimg HBV, if mother is HBsAg positive. Among infants born to HBsAg positive mothers, the risk of vertical transmission is particularly high if the mother is positive for hepatitis B e antigen (HBeAg), has a high viral load, or is infected with HIV. Such infants should receive both vaccine and HBIgG (0.5 ml) within 12 hours of birth. The infants should be tested for HBsAg. Anti-HBs, and Anti-HBc at 12 months of age. Presence of anti-HBs indicates vaccine induced immunity and detection of both Anti-HBs and Anti-HBc indicates infection modified by immunoprophylaxis, whereas presence of HBsAg indicates failure of prophylaxis (Summers *et al;* 1987; Aggarwal and Ranjan, 2004).

Accidental exposure of hepatitis B virus-people who have not been immunized and are exposed to hepatitis B (through needlestick injury, splashing, or sexual exposure to

partners infected with hepatitis B virus) should receive HBIG (0.04-0.07 ml/kg) as soon after exposure as possible. Vaccination for the newborn should be started simultaneously with the first dose given at a site different from that for HBIG; an accelerated four dose immunization schedule (0,1,2, and 12 months) is preferred in this maternal-fetal transmission setting (Aggarwal and Ranjan, 2004).

2.11 Hepatitis B Laboratory Diagnostic Tests

Serologic markers for HBV infection are about six, and the diagnosis and assessment of HBV infections are complex. In addition to the traditional markers of e antigen (HBeAg) and surface antigen (HBsAg), the clinical utility of new serologic markers is being explored (e.g. antibodies to the HBsAg epitopes pre-SI and pre-S2 proteins). Table 2.1 summarizes the interpretations of Hepatitis B diagnostic tests (Younossi, 2003); the serologic diagnosis of HBV infection is established by detecting either antibodies and/or their respective antigens (i.e. the HBsAg and Anti-HBs, HBcAg and Anti-HBc, or the HBeAg and Anti-HBe).

2.11.1 HBsAg and Anti-HBs.

The (HBsAg and Anti-HBs) tests target the mature viral envelope protein of HBV present in serum either as excess protein or complete viral particles. Highly sensitive, specific, and rapid test systems for the HBsAg include RIA, enzyme-linked imunosorbent assay (ELISA), as well as reverse passive hemagglutination methods. These techniques have been developed to meet the needs of extensive screening programs such as those for blood supply. Enzyme immunoassay is the most commonly used test for HBsAg, whereas the most commonly used tests for Anti-HBs employ both EIA and RIA methods.

2.11.2 HBcAg and Anti-HBc.

Tests for HBcAg are generally limited for use in testing liver biopsy samples. Hepatitis B core antigen is detected by immuno-staining in liver. The presence of HBcAg in liver tissue indicates ongoing viral replication and is often interpreted as ability to infect susceptibles (infectivity). Measurement of anti-HBc 1gM (principally by RIA or ELISA) can discriminate between acute HBV infection (positive) and chronic HBV cases (negative), as increased Anti-HBc is indicative of decreased viral replication. Positive anti-HBc IgM indicates acute hepatitis B infection.

2.11.3 HBeAg and Anti-HBe.

Hepatitis B e antigen is a viral protein secreted by HBV-infected cells. Its presence indicates high levels of virus in the blood, and it is an indicator of the infectiousness of HB carrier. If this test is negative, but a person is known to the HBsAg positive, then it indicates low levels of virus in the blood or an "integrated phase" of HBV in which the virus is integrated into the host's DNA. This test is often used to monitor the effectiveness of some HBV therapies, whose goal is to convert an actively replicating state to "e-antigen negative" state.

2.11.4 HBV DNA.

The presence of HBV DNA (the viral genome) indicates active viral replication. Hepatitis B virus DNA is assayed by polymerase chain reaction (PCR) methods, branched-chain DNA (bDNA) methods, and by nucleic acid hybridization methods,

PCR-based assays tend to be more sensitive, but bDNA and hybridization-based assay may provide more specificity, although the latter point is controversial. All of these assay methods detect HBV DNA in serum and are important in monitoring the effect of antiviral therapy on hepatitis B virus replication (Khakoo *et al.,* 1996).

Monitoring of HBV infection and response to treatment can be performed using combinations of these tests. Typical test panels for specific situations include (I) diagnosis of acute viral hepatitis B: HBsAg and anti-HBc; (2) for diagnosis of remote HBV infection: HBsAg, total Anti-HBc, and Anti-HBs; and (3) for monitoring ongoing HBV infection: HBsAg, total Anti-HBc, Anti-HBs, HBeAg, Anti-HBe, and HBV DNA (Younossi, 2000, Younossi, 2003).

Table 2.1: Interpretation of Hepatitis B Diagnostic Tests

Test	Acute Hepatitis B	Past Exposure (Immunity)	Previous Immunization	Chronic Hepatitis B (CHB)	Chronic Precore	Healthy Carrier	Acute Delta	Chronic Delta

HbsAg	+	-	-	+	+	+	+	+
Anti-HBs	-	+	+	-	-	-	-	-
HBeAg	+	-	-	+/-	-	-	+/-	-
Anti-HBe	-	+/-	-	+/-	+	+	-	+
Anti-HBc	+	+	-	+	+	+	+	+
IgM anti-HBc	+	-	-	-	-	-	+/-	-
HBV DNA	+	-	-	+/-	+	-	+/-	Low or –
ALT	Elevated	Normal	Normal	Elevated	Elevated	Normal	Elevated	Elevated

(Younossi, 2003)

Serologic findings also vary over time with the stage of infection; this is illustrated in Table 2.2. In the first stage of viral replication, patients generally are asymptomatic and have normal ALT levels. In the second phase of replication, an immunologic response develops or increases and ALT levels tend to become elevated as HBV DNA levels decline. Replication ends as the virus is cleared, but this is followed by a phase of integration of HBV into the infected hepatocyte's genome. This is a quiescent phase with low HBV DNA levels and normal or slightly abnormal ALT levels. The final phase is clearance of HBsAg and HBV DNA, which can occur naturally or through therapeutic intervention. HBV is not considered a curable disease, because even when viral replication is stopped, the virus persists in an inactive form (latent infections). Persons who are HBsAg negative may still have tiny amounts of HBV DNA in their liver tissue. However, though "not cured," most patients with previous exposure who are not chronically infected and lack HBsAg have minimal long-term risk from HBV (Younossi, 2003).

Table 2.2: The 4 Classic Stages of HBV Infection

	I	II	III	IV
	Replicative		Intergrative	
HBsAg	+	+	+	-
Anti-HBs	-	-	-	+
Anti-HBc	+	+	+	+
HbeAg	+	+	-	-
Anti-HBe	-	-	+	+
HBV DNA	+++	++	+PCR	-
ALT	Normal	+++	Normal	Normal

(Younossi, 2003)

2.12 Management of hepatitis B virus Infection

Treatment of HBV infections are not curative because they rarely produce permanent remission of the disease (Kin *et al; 2004)*

Chronic hepatitis B virus infection is a heterogeneous condition and can be divided into many subsets based on clinical status and status of viral replication.

Liver biopsy is the gold standard for determining disease stage (necro-inflammation) and stage of fibrosis (Ishak *et al*; 1995) but it is often contraindicated in patients with decompensate liver disease. Even in patients with compensated liver disease, though biopsy may provide information for therapeutic and prognostic decision-making, it is frequently not done (Lok and McMahon, 2001).

Three drugs- interferon alpha, lamivudine, and adefovir are approved in several countries for use in chronic hepatitis B (Aggarwal and Ranjan, 2004).

The goals of treatment of chronic hepatitis B are to reduce HB viremia and ameliorate hepatic dysfunction to suppress necro-inflammatory activities thus preventing progression to cirrhosis and hepatocellular carcinoma. The unpredictable clinical course of hepatitis B virus infection, and poor response to treatment and doubts about its cost effectiveness, render therapeutic decisions difficult (Perrillo *et al*; 2002).

Most clinical studies have focused on chronically infected patients with elevated aminotransferase levels and circulating HBeAg, in whom viral loads can be readily measured even with first generation DNA tests. There are clear indications for therapy in HBeAg-positive patients. They have an increased risk of early progression to chronic active hepatitis and cirrhosis, and they have a risk of developing

hepatocellular carcinoma, the risk is substantially higher than that of other carriers (Yang *et al*; 2002).

The usual markers of successful therapy are the elimination of HBeAg from HB patient's serum, seroconversion to Anti-HBe antibodies, and reduction of the circulating viral load. These are useful indicators, since patients with stable seroconversion to Anti-HBe-positive status typically have improved histologic findings in the liver, and this improvement tends to be maintained over long period of time (Niederau *et al*; 1996). True cure of infection (loss of HBsAg and complete disappearance of viremia, as measured by stringent PCR assays is achieved only infrequently (in 1 to 5 percent of patients) with current regimens, although the increasing numbers of active antiviral drugs might lead to an upward revision of this figure in the future. In the case of patients with HBeAg-negative chronic hepatitis, there is no information about which markers best measure the response to therapy.

2.12.1 Drugs/ Agents used in treating chronic HBV infection

2.12.1.1 Interferon-alpha

Interferon alpha, a human cytokine produced in response to any viral invasion, has immunomodulatory, antiviral, and anti-fibrotic properties. It was first used in the 1980s and was the first drug to be found useful in the treatment of of chronic hepatitis B. The dose is 5 million units a day or 10 MU thrice weekly (30-35MU/week), given subcutaneously, usually for 16 weeks.

Interferon-alpha can have several adverse effects on users. An influenza-like illness (fever, chills, headache, malaise, myalgia) occurs in 25-30% of patients but rarely requires discontinuation of treatment. However, more serious adverse effects (myelosuppression with leucocyte count <1000cells/ul and platelets <60 000cells/ul), emotional lability and depression, development of autoantibodies, and thyroid dysfunction may lead to discontinuation of interferon alpha treatment; thus pretreatment screening for psychiatric patients: low leucocyte and platelets counts, autoantibodies, and thyroid function is mandatory.

Interferon alpha treatment of chronic HBV infections in patients with cirrhosis has been reported to reduce the risk of hepatocellular carcinoma (Ikeda *et al;*1998).

Pegylated interferon, a longer acting interferon preparation may be better than conventional interferon, but presently, data on its use are limited, thereby precluding its recommendation for routine use (Cooksley *et al;* 2003.)

2.12.2 *Lamivudine*

Lamivudine, a synthetic nucleoside (cytosine) analogue available since 1998, undergoes intracellular phosphorylation to its active metabolite to lamuvidine triphosphate and inhibits HB viral reverse transcriptase, causing premature chain termination during viral DNA synthesis. It emerged from the search for inhibitors of the HIV reverse transcriptase and was consequently introduced into clinical practice for the management of HIV infection (Puoiti *et al;* 2002)

The drug is usually well tolerated, a factor that has led to rapid displacement of interferon alpha from the roster of first-line therapies for HBV infection. Lamivudine is not immunomodulatory and can be used in patients with decompensate cirrhosis.

Even polyarteritis nodosa associated with HBV has been shown to respond dramatically to treatment with lamivudine plus plasma exchange (Trepo *et al*; 2001; Chen *et al*; 1999). The principal limitation of lamivudine mono therapy is the development of drug resistance by point mutations at the tyrosine-methionine-aspartate-aspartate motif in the catalytic center of the HB viral reverse transcriptase, this hence confers lamivudine resistance (Liaw et al; 2000; Marcellin *et al;* 2003). Newer anti-HBV drugs are now available, additional options exist for patients with resistant strains of HBV(Yang *et al;* 2002).

2.12.3 Adefovir

Adefovir dipivoxil, a nucleotide analogue of deoxy-adenosine monophosphate, inhibits viral transcriptase activity in both wild and mutant hepatitis B virus (Peters *et al;* 2004). It is the drug of choice for patients treated with lamivudine who have developed resistance due to mutation in HB viral genome. It has also been used in patients with HbeAg positive chronic hepatitis B and HBeAg negative chronic hepatitis B, with efficacy rates at one year similar to those with lamivudine, albeit with no drug-induced resistance mutations (Hadjiyannis *et al;* 2003).

2.13 Hepatitis B Vaccine

Hepatitis B vaccines are of two types, plasma derived and DNA recombinant. Recombinant DNA vaccines are produced by cloning the gene encoding HBsAg into yeast cells and are increasingly replacing plasma derived vaccines.

Vaccines are given in three doses of 10-30 µg (usually 20 µg for adults and 10 µg for children). The vaccines are extremely safe and induce antibodies that will neutralize HBsAg (Anti-HBs) in most (>95%) recipients: antibody levels in excess of 10 mlU/ml are considered protective. Certain groups of people such as those aged over 40 years, obese people, those with chronic renal failure, haemodialysis patients, immunosuppressed individuals, organ transplant recipients, have poorer response rates (Aggarwal and Ranjan,2004). The protection lasts for at least 15 years, and because of strong immunological memory it continues after Anti-HBs has become undetectable. Immunity is thus believed to be life long, and boosters are not routinely recommended; however, these may have a role in immunosuppressed individuals and those at a particularly high risk of exposure. Universal neonatal vaccination is effective and has been shown to favourably alter the clinical course of hepatitis B virus infection in regions where the disease is endemic (Aggarwal and Ghoshal, 2003; Aggarwal and Ranjan, 2004).

2.14 Liver Transplantation

Liver transplantation is not contraindicated in hepatitis B virus related liver disease. Treatment with lamivudine or adefovir before the transplantation reduces the risk of recurrence through reducing the viral DNA load; though the best results have been obtained with post-transplantation prophylaxis consisting of both hepatitis B immune globulin and lamivudine. Hepatitis B immune globulin is very expensive and contributes substantially to the cost of transplantation (Anselmo *et al;* 2002).

Chapter 3 MATERIALS AND METHODS

3.1 Area of Study

This study was conducted using blood specimens collected randomly from pregnant women attending antenatal clinics in General Hospitals Kaduna metropolis. These include Barau Dikko Specialist Hospital (BDSH), Yusuf Dantsoho Memorial Hospital (YDMH) Tudun wada , and Gwamna Awan General Hospital,(GAGH) Kakuri .

Kaduna was founded by the British in 1913 and became the capital of the former Northern region of Nigeria between 1917 and 1967. It is presently the capital of Kaduna State, north central Nigeria. It is centrally located and share boundaries with the following states: Niger, Plateau, Abuja, Nasarawa, Katsina, Kano. Zamfara and Bauchi. It is made up of 23 Local Government Areas: Birnin Gwari, Chikun, Giwa, Igabi, Ikara , Jaba, Jama'a, Kachia, Kaduna North, Kaduna South, Kagarko, Kajuro, Kaura, Kauru, Kubau, Kudan, Makarfi, Lere, Sabon Gari, Sanga, Soba, Zangon Kataf, and Zaria The main ethnic group are Bajju, Kataf, Kagoro,Moro'a Jaba, Gbagyi, Kaninkon, Ninzam, Hausa Fulani, Chawai, Kurama.

Kaduna State has a population of 3.96 million (1991 Census), while Kaduna city has a population of 1,580,000 (1998 estimate). Kaduna metropolis comprises of two local government areas: Kaduna north local government and Kaduna South Local government areas. These two local governments are made up of mostly civil servants, industrial workers, traders, farmers and students. Kaduna is the commercial and industrial centre of Northern Nigeria. Hausa is the major language apart from English

which is the nation's lingua franca.(www.onlineNigeria.com/links/Kaduna, www.kaduna-state.com).

3.2 Data Collection

3.2.1 Sample Size

The sample size was obtained by using the equation

$$n = \frac{Z^2Pq}{L^2}$$ (Araoye, 2003).

Where

n = the desired sample size (range statistically analyzable)

z = the standard normal deviate, which corresponds to the 95% confidence interval = 1.96

p = known prevalence of the condition = 25.9% based on (Harry *et al.,* 1994)

q = 1.0 – p

L = allowable error = 0.05

$$n = \frac{1.96^2 \times 0.259 \times 0.741}{(0.05)^2} = 294$$

Three hundred and twelve samples were randomly collected in all by selecting the first two out of every three patients at each hospital. 112 specimen were collected from Barau Dikko Specialist Hospital (with an average ante-natal population of about 182 per month). 100 specimens were collected from Yusuf Dantsoho Memorial Hospital (with an average ante-natal population of about 170 per month), and 100 specimens from Gwamna Awan General Hospital, Kakuri. (with average ante-natal population of

about 180 per month). Out of the 312 specimen 274 valid specimens (92 from Yusuf Dantsoho Memorial Hospital, 90 from Barau Dikko Specialist Hospital and 92 from Gwamna Awan General Hospital) were randomly picked by balloting with replacement. The controls were provided with the kits (both positive and negative controls). The matrons recruited women in the study, those that after entering the hospital's antenatal care gave their consent after the weekly antenatal enlightenment talks.

3.2.2 Study Design

This work is a descriptive cross-sectional survey.

3.3 Questionnaire Administration

Using a questionnaire, the following data were collected from each participant: personal identification data, age, gyneacologic and obstetric variables, transmission mechanisms, potential risk factors, and spouses' data. A sample of the questionnaire used is shown in Appendix C.

3.4 Collection of Samples

Blood samples (3ml) were collected from each participant by venepunture using 5cc syringes fixed with 21 guage needles. The samples were collected into blood bottles without anticoagulant and were allowed to clot. The sera were separated into sterile blood bottles by centrifugation at 1000 rpm for 30 minutes and the sera samples were

immediately stored at (-20^{0}C) until screened. Each serum sample bottle was appropriately labeled for identification.

3.5 Reagents

The presence of HBeAg, antibodies to HBsAg (Anti-HBs) and HBcAg (Anti-HBc) were determined using AutoBio Anti-HBs plus, AutoBio HBeAg, AutoBio Anti-HBc plus , commercial assay kits made by AutoBio ImmunoDiagnostics, 115 chess Drive, Foster City, CA 94044, U.S.A.

3.6 Equipment and Materials

1. Precision Micropipettes and disposable pipette tips (50μL)
2. Incubator (37^{o}C)
3. Microtiter plate reader for the measurement of absorbance at 450 nm (Sigma Diagnostics EIA multi-well Reader II)
4. Adsorbent pad
5. Distilled water
6. Physiological saline.

3.7 Anti-HBs Kit Components

1. Coated microtitreplates: One microtiter plate each with 96 wells, coated with natural inactivated HBsAg (8 removable strips with 12 wells each).
2. Enzyme Conjugate: One vial (7.5ml) containing a solution of HRP-conjugated-HBsAg.

3. Hepatitis B surface antibody (HBsAb) Negative Control: One vial (1 ml) containing human serum positive for HbsAb.
4. HBsAb Positive Control: One vial (1 ml) containing human serum positive for HBsAb.
5. Chromogen Substrate Solution A: One vial (7.5ml) containing Urea Hydrogen Peroxide
6. Chromogen Substrate Solution B: One vial (7.5 ml) containing stabilized 3,3'-5,5' Tetramethylbenzidine (TMB).
7. Stop Solution: One vial (7.5 ml) containing 1.0 M H_2S0_4, Ready to use.
8. Washing solution (20X Conc.): One vial (30 ml) containing Phosphate Buffered Saline.

3.7.1 Principle of the Assay (Anti-HBs Detection)

The HBsAb EIA is a solid-phase simultaneous immunoassay to detect antibody against HBsAg. Micro wells were already coated with HBsAg. A serum specimen is added to the micro wells together with Horseradish Peroxidase (HRP) conjugated HBsAg. After incubation, the complex of antigen-antibody-antigen (HRP-conjugated HBsAg, Anti-HBS and HBsAg on the wells) will be formed. Thus, the amount of HRP-HBsAg bound to the well is proportional to the concentration of Anti-HBs in the specimen. The unbound enzyme conjugates is washed away and then the chromogen substrate solution containing hydrogen peroxide is added to the wells. A blue color develops intensity of which is in proportion to the amount of Anti-HBs in the serum specimen. The enzyme-substrate reaction is stopped by the addition of sulphuric acid.

The absorbance of controls and specimens is determined by using EIA reader with wavelength set at 450 nm.

3.7.2 Test Procedure (Anti-HBs Detection)

The procedure according to the manufacturer was adhered to as follows:-

1. The serum samples and all reagents were brought to room temperature.
2. The entire contents of the concentrated wash solution was poured into a graduated beaker of 800ml capacity. The vial was rinsed with distilled water and more distilled water poured into the beaker up to a total volume of 600ml. The content of the beaker was then transferred into a 1000ml bottle for storage until used to wash the microtiter plates.
3. The relative numbers of specimens and wells were written down on a data sheet taking into cognizance the blank, the negative and positive control wells.
4. The first well (1A) was left blank.
5. 50 µl of the negative control was pipetted into the next two wells 2A and 3A .
6. 50 µl of the positive control was pipetted into the following two wells 4A and 5A.
7. 50 µl of the specimen was pipetted into the remaining wells.
8. 50 µl of the HBsAg-peroxidase-conjugate working solution was pipetted into each well, except the blank (1A)
9. The plates were then incubated at $+37^0$C in a water-bath for 30 minutes.
10. At the end of the incubation, the plates were washed 6 times with the working wash solution. Each of the 6 times, the plates were filled with the wash buffer,

inverted vigorously to get water out and blocking the rim of well on adsorbent paper for 30 seconds.

11. 50 µl of chromogen substrate A and B was dispensed into each well including the blank and mixed horizontally by swirling.
12. The microtiter plates were incubated for 20 minutes in a water bath at $+37^0$ C (exposure to direct sunlight was avoided).
13. 50 µl of blocking reagent (stop solution) was dispensed ;into each well including the blank in the same order adopted for the addition of the chromogen substrate solution.
14. The plates were read immediately according to the manufacturer's instruction at wavelength of 450/630nm in a dual-filter enzyme immunoassay reader (Sigma diagnostics EIA multi-well Reader II).
15. Each test result was recorded to match the serum sample.

3.8. Anti-HBc KIT Components

1. Coated microtitreplates: One microtiter plate each with 96 wells, coated with recombinant HBcAg (8 removable strips with 12 wells each).
2. Enzyme Conjugate: One vial (7.5) ml) containing a solution of anti HBc-HRP conjugate.
3. Anti HBc Negative Control: One vial (1 ml) containing human serum based solution, not reactive for anti-core (HBc) anti-bodies .
4. Anti HBc Positive Control: One vial (1 ml) containing anti-HBc anti-bodies in serum protein solution containing preservatives.

5. Chromogen Substrate Solution A: One vial (7.5 ml) containing Urea Hydrogen Peroxide
6. Chromogen Substrate Solution B: One vial (7.5 ml) containing stabilized 3,3'-5,5' Tetramethylbenzidine (TMB).
7. Stop Solution: One vial (7.5 ml) containing 1.0 M H_2S0_4, Ready to use.
8. Washing solution (20X Conc.): One vial (30 ml) containing Phosphate Buffered Saline.

3.8.1 Principle of the Assay (Anti-HBc Detection)

Anti-HBc in patient sample and Anti-HBc peroxidase conjugate (Anti-HBc-HRP , Enzyme tracer) compete for the limited binding sites of the recombinant Hepatitis B core antigen, coated onto the wells. After incubation, the unbound reagents are removed by washing and tetramethylbenzidine (TMB) solution containing hydrogen peroxide is added to the well. During the incubation, a blue color develops intensity of which is in proportion to the amount of HRP-Anti-HBc bound to the well. If a patient sample does not contain Anti-HBc, the quantity of Anti-HBc-HRP conjugate remaining bound to the solid phase immunocomplex is maximum and, therefore, the color developed during the enzymatic reaction is quite intense on the contrary, if the sample contains Anti-HBc, the amount of the Anti-HBc-HRP remaining bound is reduced and the color is weaker. Sample positivity or negativity is given by reference to a cut-off-control serum as read by spectrophotometer or multi-well EIA-reader II.

3.8.2 Test Procedure (Anti-HBc Detection)

The procedure according to the manufacturer was adhered to as follows:-

1. The serum samples and all reagents were brought to room temperature.
2. The entire contents of the concentrated wash solution was poured into a graduated beaker of 800ml capacity. The vial was rinsed with distilled water and more distilled water poured into the beaker up to a total volume of 600ml. The content of the beaker was then transferred into a 1000ml bottle for storage until used to wash the micro titer plates.
3. The relative numbers of specimens and wells were written down on a data sheet, taking into cognizance the blank, the negative and positive control wells.
4. The first well (1A) was left blank.
5. 50 µl of the negative control was pipetted into the next two wells 2A and 3A .
6. 50 µl of the positive control was pipetted into the following two wells 4A and 5A.
7. 50 µl of the specimen was pitted into the remaining wells.
8. 50 µl of the anti-HBs peroxidase –conjugate was pipetted into each well, except the blank (1A)
9. The plates were then incubated at $+37^0$C in a water-bath for 30 minutes.
10. At the end of the incubation, the plates were washed 6 times with the working wash solution. Each of the 6 times, the plates were filled with the wash buffer, inverted vigorously to get water out and blocking the rim of well on adsorbent paper for 30 seconds.

11. 50 µl of chromogen substrate A and B was dispensed into each well including the blank and mixed horizontally by swirling.
12. The microtiter plates were incubated for 20 minutes in a water bath at $+37^0$ C (exposure to direct sunlight was avoided).
13. 50 µl of blocking reagent (stop solution) was dispensed ;into each well including the blank in the same order adopted for the addition of the chromogen substrate solution.
14. The plates were read immediately according to the manufacturer's instruction at a wavelength of 450/630nm in a dual-filter enzyme immunoassay reader (Sigma diagnostics EIA multi-well Reader II).
15. Each test result was appropriately recorded against its sample label.

3.9 HBeAg Kit Components

1. Coated microtitreplates: One microtiter plate each with 96 wells, coated with monoclonal antibodies against HBeAg (8 removable strips with 12 wells each).
2. Enzyme Conjugate: One vial (7.5ml) containing a solution of IgG Anti-HbeAg conjugated to peroxidase.
3. HBeAg Negative Control: One vial (1 ml) containing human serum free of HBeAg markers it contains protein stabilizers and preservatives.
4. HBeAg Positive Control: One vial (1 ml) containing human serum positive for HBeAg with protein stabilizers and preservatives.

5. Chromogen Substrate Solution A: One vial (7.5 ml) containing urea hydrogen peroxide
6. Chromogen Substrate Solution B: One vial (7.5 ml) containing stabilized 3,3'-5,5' Tetramethylbenzidine (TMB).
7. Stop Solution: One vial (7.5 ml) containing 1.0 M H_2S0_4, Ready to use.
8. Washing solution (20X Conc.): One vial (30 ml) containing phosphate buffered saline.

3.9.1 Principle of the Assay, (HBeAg Detection)

The AutoBio HBeAg on microstrip is an enzyme immunoassay for the qualitative detection of Hepatitis B 'e' antigen based on the sandwich method. The wells of the microstrips area coated with monoclonal antibody specific for HBeAg which forms stable complex with HBeAg present in the serum or plasma, samples pipetted into the wells. The enzymatic tracer (IgG anti-HBeAg, labeled with horseradish peroxidase) is added to the wells and where the antigen/antibody complex is present, the peroxidase conjugate binds to the antigen forming the complex. Antibody-Antigen-Antibody-HRP. The activity of the HRP is then revealed by the addition of the chromogen/substrate which becomes blue and then turns yellow after stopping the reaction with sulphuric acid.

3.9.2 Test Procedure (HBeAg Detection)

The procedure according to the manufacturer was adhered to as follows:-

1. The serum samples and all reagents were brought to room temperature.

2. The entire contents of the concentrated wash solution was poured into a graduated beaker of 800ml capacity. The vial was rinsed with distilled water and more distilled water poured into the beaker up to a total volume of 600ml. The content of the beaker was then transferred into a 1000ml bottle for storage until used to wash the microtiter plates.
3. The relative numbers of specimens and wells were written down on a data sheet, taking into cognizance the blank, the negative and positive control wells.
4. The first well (1A) was left blank.
5. 50 µl of the negative control was pipetted into the next two wells 2A and 3A .
6. 50 µl of the positive control was pipetted into the following two wells 4A and 5A.
7. 50 µl of the specimen was pipetted into the remaining wells.
8. 50 µl of the anti-HBe peroxidase –conjugate was pipetted into each well, except the blank (1A)
9. The plates were then incubated at 37^0C in a water-bath for 30 minutes.
10. At the end of the incubation, the plates were washed 6 times with the working wash solution. Each of the 6 times, the plates were filled with the wash buffer, inverted vigorously to get water out and blocking the rim of well on adsorbent paper for 30 seconds.
11. 50 µl of chromogen substrate A and B was dispensed into each well including the blank and mixed by swirling horizontally.

12. The microtiter plates were incubated for 20 minutes in a water bath at 37^0 C (exposure to direct daylight was avoided).
13. 50 µl of blocking reagent (stop solution) was dispensed ;into each well including the blank in the same order adopted for the addition of the chromogen substrate solution.
14. The plates were read immediately according to the manufacturer's instruction at a wavelength of 450/630nm in a dual-filter enzyme immunoassay reader (Sigma diagnostics EIA multi-well Reader II).

3.10 Precautions

The following precautions were observed while performing the assay:

1. Polyvinylchloride(PVC) gloves were worn during the whole process of handling reagents and specimens, hands were thoroughly washed afterward.
2. Separate disposable pipettes were used for each specimen.
3. Non-acid containing spills were wiped using 5% hypochlorite solution.
4. Activity of horse-radish peroxidase conjugate is easily inhibited by metal and other substances, it was therefore handled with great care.
5. 3-3'-5,5' Tetramethlylbenzidine (TMB) was not exposed to direct daylight, metal or oxidants.
6. Components of the different kits were never interchanged.
7. The positive control of HBeAg was treated as "highly infectious material."
8. Applicable standard laboratory practices were observed.

3.11 Data Analysis

Data collected from the HBV assay and questionnaires were outlined in tables, this is shown in Appendix D.

Data analysis included central frequency, measures of dispersion for continuous variables and percentages for categorical variables. Chi –square cross tabulation, Pearson correlation and T-test were used to determine statistical significance between the HBV infection markers and the parameters considered.

In the interpretation of analysis, a P value of less than or equal to 0.05 implies significance for the two variables considered. All statistical analysis were done using SPSS II and EPI INFO statistical software, this is shown in appendix H.

3.12 Determination of Cut off Values and Interpretation of Tests' Results

The cut off values were determined and the results appropriately interpreted using the AutoBio KC4 Program, this is shown in Appendices E,F and G.

3.13 Ethical Consideration

Informed consent was obtained from the Ministry of Health, Kaduna State through the office of the honorable Commissioner for Health, the Medical Directors of all the hospitals and study subjects participated on their own volition. A copy of request to collect samples and the written consents appear in appendix A and B.

Chapter 4 RESULTS

4.1 Results of the HB Serological Markers Assays

The results of the assay carried out on the serum samples with AutoBio plus showed that out of the 274 pregnant women tested, 77 (28.1%) were positive for antibodies to hepatitis B surface antigen (Anti-HBs), 57 (20.8%) were positive for Anti-HBc and 6 (2.2%) were positive for HBeAg.

Table 4.1 summarizes the seroprevalence of HBV infection markers among pregnant women attending ante natal clinics in Kaduna metropolis.

Table 4.1: Prevalence by hospitals of Hepatitis B Virus Infection Markers Among Pregnant Women Attending Antenatal Clinics in Kaduna Kaduna State.

Hospital	Number Tested	HBV INFECTION MARKERS [NUMBER POSITIVE (PERCENTAGE POSITIVE)]		
		ANTI-HBs	ANTI-HBc	HBeAg
Barau Dikko	90	29 (32.2%)	16 (17.8%)	4 (4.4%)
Yusuf Dantsoho	92	29 (31.5%)	23 (25%)	1 (1.1%)
Gwamna Awan	92	19 (20.7%)	18 (19.6%)	1 (1.1%)
Total	274	77 (28.1%)	57 (20.8%)	6 (2.2%)

Figures in parentheses represent percentages per hospital.

Table 4.2 Summarizes the educational status distribution of HBV infection markers among pregnant women attending Antenatal clinics in Kaduna. Participants were divided into groups: Illiterate, read and write only joined with primary school, secondary school and tertiary education.

Table 4.2: Distribution by Educational Status of HBV Infection Markers Among Pregnant Women Attending Antenatal Clinics in Kaduna, Kaduna State.

Educational Status	Number Tested	HBV INFECTION MARKERS NUMBER POSITIVE (PERCENTAGE POSITIVE)]		
		ANTI-HBs	ANTI-HBc	HBeAg
Illiterate	34	7 (20.6%)	10 (29.4%)	1 (2.9%)
Primary	50	14 (28.0%)	12 (24.0%)	0 (0.0%)
Secondary	142	45 (31.7%)	23 (16.2%)	4 (2.8%)
Tertiary	48	11 (22.9%)	12 (25.0%)	1 (2.1%)
Total	274	77 (28.1%)	57 (20.8%)	6 (2.2%)

Figures in parentheses represent percentage per status.

Married women accounted for 254 (92.7%) of the study population and interestingly had the highest prevalence rates for the HBV infection markers screened for. Table 4.3 summarizes the marital status distribution of HBV infection markers among pregnant women attending antenatal clinics in Kaduna.

Table 4.3 Marital Status Distribution of HBV Infection Markers Among Pregnant Women attending Antenatal Clinics Kaduna, Kaduna State

Marital Status	Number Tested	HBV INFECTION MARKERS NUMBER POSITIVE (PERCENTAGE POSITIVE)]		
		ANTI-HBs	ANTI-HBc	HBeAg
Single	2 0	7 (35.0%)	4 (20.0%)	0 (0.0%)
Married	254	70 (27.6%)	53 (20.9%)	6 (2.4%)
Total	274	77 (28.1%)	57 (20.8%)	6 (2.2%)

Figures in parentheses represent percentage per status.

Table 4.4. summarises the age distribution of HBV infection markers among pregnant women attending antenatal clinics in Kaduna. The number of women that were positive for the infection markers were prominent among 20-24 and 25-29 age groups.

Table 4.4 Age Distribution of HBV Infection Markers among Pregnant Women attending Antenatal Clinics Kaduna.

Age (Years)	Number Tested	HBV INFECTION MARKERS NUMBER POSITIVE (PERCENTAGE POSITIVE)]		
		ANTI-HBs	ANTI-HBc	HBeAg
15-19	2 4	4 (16.7%)	8(33.3%)	0 (0.0%)
20-24	68	24 (35.3%)	13 (19.1%)	2 (2.9%)
25-29	95	27 (28.4%)	21 (22.1%)	2 (2.1%)
30-34	66	16 (24.2%)	12 (18.2%)	2 (3.0%)
35-40	14	4 (28.6%)	1 (7.1%)	0(0.0%)
40 +	7	2 (28.6%)	2 (28.6%)	0(0.0%)
Total	274	77 (28.1%)	57 (20.8%)	6 (2.2%)

Figures in parentheses represent percentages per age bracket.

86 (31.4%) of the 274 pregnant women reported more than one sexual partner in the previous year. The prevalence rate was highest among those that claimed to have one sexual partner. Table 4.5 summarises the distribution of HBV infection markers with regards to the number of sexual partners.

Table 4.5: Number of Sexual Partners Distribution of HBV Infection Markers Among Pregnant Women Attending Antenatal Clinics in Kaduna .

Number of Sexual Partners	Number Tested	HBV INFECTION MARKERS NUMBER POSITIVE (PERCENTAGE POSITIVE)]		
		ANTI-HBs	ANTI-HBc	HBeAg
One	188	51 (27.1%)	42 (22.3%)	4 (2.1%)
Two	55	12 (21.8%)	9 (16.4%)	1 (1.8%)
Three	11	5 (45.5%)	2 (18.2%)	1 (9.1%)
Three plus	20	9 (3.3%)	4 (1.5%)	0 (0.0%)
Total	274	77 (28.1%)	57 (20.8%)	6 (2.2%)

Figures in parentheses represent percentages per group.

It is evident as shown in Tables 4.6, 4.7 and 4.8 that there are no statistical associations between HBV infection markers and the parameters considered. This is because the Chi Square P values were not less than or equal to 0.05.

Table 4.6 : Statistical Association between Anti-HBs and Some Subjects' Parameters.

Parameter	Odds Ratio Value	95% Confidence Interval Upper	Lower	Chi Square P Value
Marital Status	0.82	0.6812	1.1113	1.9211
Age	1.20	0.9012	1.4170	2.2180
Educational Status	0.56	0.4702	0.9886	5.2321
No. of Sexual Partners	0.86	0.7112	1.3333	4.5011

There is statistical association when $P \leq 0.05$.

Table 4.7 : Statistical Association between Anti-HBc and Some Subjects' Parameters

Parameter	Odds Ratio Value	95% Confidence Interval Upper	Lower	Chi Square P Value
Marital Status	0.68	0.5999	1.222	2.4142
Age	0.98	0.6210	1.4952	1.0026
Educational Status	0.59	0.2010	1.1112	3.4221
No. of Sexual Partners	0.81	0.5213	1.3721	2.5634

There is statistical association when $P \leq 0.05$.

Table 4.8 : Statistical Association between Anti-HBc and Some Subjects' Parameters

Parameter	Odds Ratio Value	95% Confidence Interval Upper	Lower	Chi Square P Value
Marital Status	0.87	0.4351	1.6771	1.3313
Age	0.99	0.3223	1.5344	0.1591
Educational Status	0.56	0.2154	1.0811	4.3002
No. of Sexual Partners	0.70	0.3758	1.4002	2.0013

There is statistical association when $P \leq 0.05$.

Chapter 5 DISCUSSION

The seroprevalence of hepatitis B virus infection in Kaduna has not been studied. According to the results of this study, the seroprevalence rates of Anti-HBs, Anti-HBc and HBeAg in pregnant women in Kaduna metropolis are 28.1%, 20.8% and 2.2% respectively. Overall exposure to hepatitis B virus is an important indicator of epidemiologic pattern of disease. In this study, all cases that tested positive for any of the three HBV infection markers: anti-HBs, anti-HBc, and HBeAg, were regarded as having been exposed to HBV. Two hundred and seventy-four serum samples from pregnant women were analyzed. The trend of the seropositivity to the three serological markers in relation to age was observed. For anti-HBs, a marker that suggests past infection with conferred immunity (Brooks *et al;*2004), the highest prevalence (35.3%) was observed in the 20-24 years age group. The 15-19 years age group recorded a lower prevalence of 16.7%. For anti-HBc, which is considered the most representative marker for HBV exposure and which indicates infection with HBV at some time in the past (Castro *et al;2003, Brooks et al;2004*), the highest prevalence (33.3%) was observed in the (15-19) years age group. The 20-24 years and 25-29 years age groups had 19.1% and 22.1% respectively. For HBeAg, a marker which indicates viral replication (Vasquez-Martiez *et al;*2003, Brooks *et al;* 2004), it is observed that only the (20-24) years , (25-29)years and (30-34) years age groups had this serological marker, with the highest prevalence (3.0%) being in the (30-34) years age group. The most likely explanation for this observation is the high sexual activity of this age range. It is

obvious from this survey that 51(31.3%) of the 163 that were tested within the (20-29) years age group have been exposed to HBV in the past. One of the distinctive features of HBV infection is that risk of chronicity varies greatly with age at which the infection is acquired. For neonates born to HBeAg positive mothers and children younger than 12 months of age who acquire the infection, the risk of infection becoming chronic is 90% (Lai *et al;*2003). The seroprevalence rates of anti-HBs increased with an increase in educational status, however the tertiary group did not follow this trend. For anti-HBc, the seroprevalence rate decreased with increase in educational status, this again is without the tertiary group. The HBeAg marker was found in all the educational status groups except the 'primary school' group, the highest prevalence being in the illiterate group (2.9%) followed by the tertiary group (2.1%). It is observed that educational status had effect on the seropositivity of the HBV infection markers, although the effect was not statistically meaningful. This could be due to the fact that in multivariate analysis, viral infections were independently associated not only with behaviours such as intravenous drug usage and commercial sex but also educational levels (Stover, *et al;* 2003). In urban areas like Kaduna, preventive measures are more available than in rural areas, but the use of these health facilities increases with educational level. People with a higher level of education tend to use health services like health education, vaccination, and high quality dentistry services, etc (Mehmet *et al;*2005). With regards to marital status, married women accounted for 254 (92.7%) of the 274 antenatal clinic attendees that participated in this study of which 70 (27.6%) are seropositive to anti-HBs while 53(20.9%) and 6(2.4%) were

seropositive to anti-HBc and HBeAg respectively. These may reflect the fact that some of them have had past exposure to HBV and while some might have been conferred with immunity, or are asympyomatic carriers, others which incude the 6(2.4%) that were seropositive to HBeAg are at risk of transmitting HBV to their babies. It is also interesting to note that despite the fact that multi partnership is unusual with married women yet the prevalence rates of the serological markers: anti-HBs and HBeAg under study were highest (27.6% and 2.4%) respectively for married women. This could be due to unfaithfulness or the polygamous nature of some families. The rate of seroprevalence observed in relation to the number of sexual partners show that there is a progressive increase in the seropositivity rate of anti-HBs as the number of sexual partners increase with the exception of those that claimed two sexual partners. There is no uniform trend as regards the number of sexual partners in relation to seropositivity to anti-HBc and HBeAg, while 42(22.3%) of 188 who claimed one sexual partner are positive to anti-HBc, 4(2.1%) of the same group (one sexual partner) are positive to HBeAg. This is suprising, as it is thought that significant number of HBV infections are sexually transmitted, because in resource-poor countries, where most HBV infections occur, sexual transmission is still relevant to adults (Donovan, 2004). In this study, the distribution recorded with regards to the number of sexual partners raised a considerable interest due to the fact that women that claimed one sexual partner in the previous year were the majority that tested positive for infection markers tested. This is in contrast to the findings of Vazquez-Martinez *et al* (2003) who found a significantly higher HBsAg and Anti-HBs seroprevalence in pregnant

women with multiple sexual partners. The reason for this disparity is not clear. The seroprevalence rates of similar HBV infection markers from pregnant women attending ante-natal Clinic in Maiduguri (North East Nigeria) in 1994 gave 25.9%, 64.3% and 12.0% for Anti-HBs, Anti-HBC and HBeAg respectively (Harry et al, 1994). These figures are relatively higher than what was obtained in this study. It is possible that the different prevalences mentioned are due to geographic variations. Time difference might also play a role because within these twelve years, there is a high probability that awareness has increased leading to more immunizations and less harmful practices/behaviours that can predispose to HBV infections. A striking finding is that 71.9% and 79.2% of the sampled population are anti-HBs negative and anti-HBc negative respectively, this is worrisome and is an indication that they have not had past exposure by any means. This shows that a large percentage of the study population are still vulnerable to hepatitis B infection. Some potential problems with the prevalences detected in this study could be associated with deceptive responses in filling of the questionnaire.

Chapter 6 SUMMARY, CONCLUSION AND RECOMMENDATIONS

This study has shown that HBV is present in Kaduna, Kaduna state and the seroprevalance rates of anti-HBs, anti- HBc and HBeAg in Kaduna pregnant women is 28.12, 20.8% and 2.2% respectively.

Within the limits of this study, there was no statistically proven association between the HBV infection markers considered and the chosen parameters; educational status, number of sexual partners, age and marital status.

It is my recommendation that the possibility of implementing preventive measures should be assessed, it is necessary to screen all blood donors not only for HBsAg but also for HBeAg and discard those found positive. Pregnant women should be routinely screened for this markers and babies of positive ones administered with hepatitis B immunoglobulin (HBIG) in 24 hours and vaccinated preferably. It is also recommended that though it has been included on expanded programme on immunization (EPI), a more extensive hepatitis B vaccination program be enforced. This will ensure vaccination of all children against hepatitis B irrespective of the serological status of their mothers. Also mass immunization is recommended for the population .

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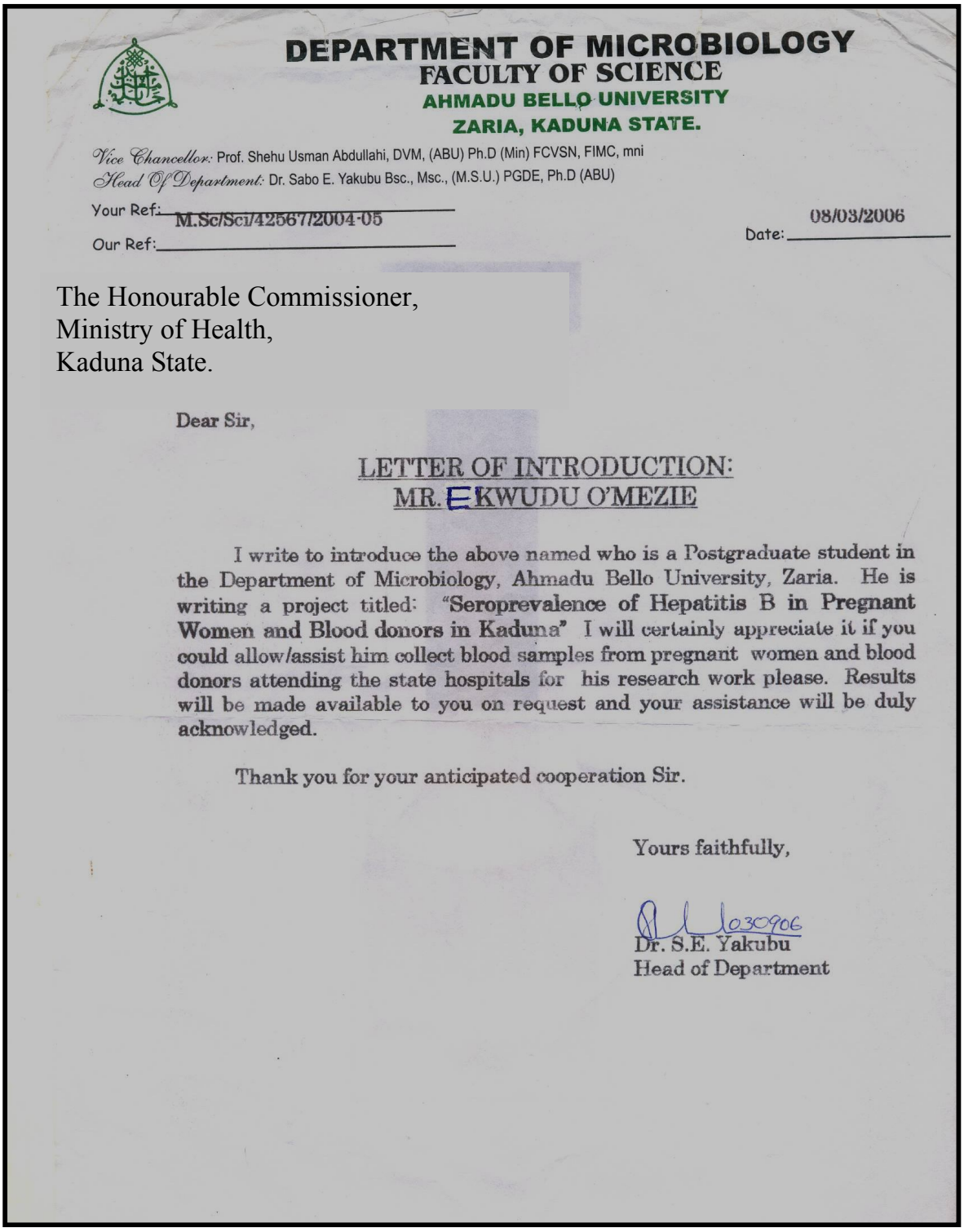
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APPENDIX A

LETTER OF INTRODUCTION REQUESTING PERMISSION TO COLLECT BLOOD SAMPLES AND DATA FROM PREGNANT WOMEN ATTENDING ANTE-NATAL CLINICS IN STATE GENERAL HOSPITALS



DEPARTMENT OF MICROBIOLOGY
FACULTY OF SCIENCE
AHMADU BELLO UNIVERSITY
ZARIA, KADUNA STATE.

Vice Chancellor: Prof. Shehu Usman Abdullahi, DVM, (ABU) Ph.D (Min) FCVSN, FIMC, mni
Head Of Department: Dr. Sabo E. Yakubu Bsc., Msc., (M.S.U.) PGDE, Ph.D (ABU)

Your Ref: M.Sc/Sci/42567/2004-05
Our Ref:

Date: 08/03/2006

The Honourable Commissioner,
Ministry of Health,
Kaduna State.

Dear Sir,

LETTER OF INTRODUCTION:
MR. EKWUDU O'MEZIE

I write to introduce the above named who is a Postgraduate student in the Department of Microbiology, Ahmadu Bello University, Zaria. He is writing a project titled: "**Seroprevalence of Hepatitis B in Pregnant Women and Blood donors in Kaduna**" I will certainly appreciate it if you could allow/assist him collect blood samples from pregnant women and blood donors attending the state hospitals for his research work please. Results will be made available to you on request and your assistance will be duly acknowledged.

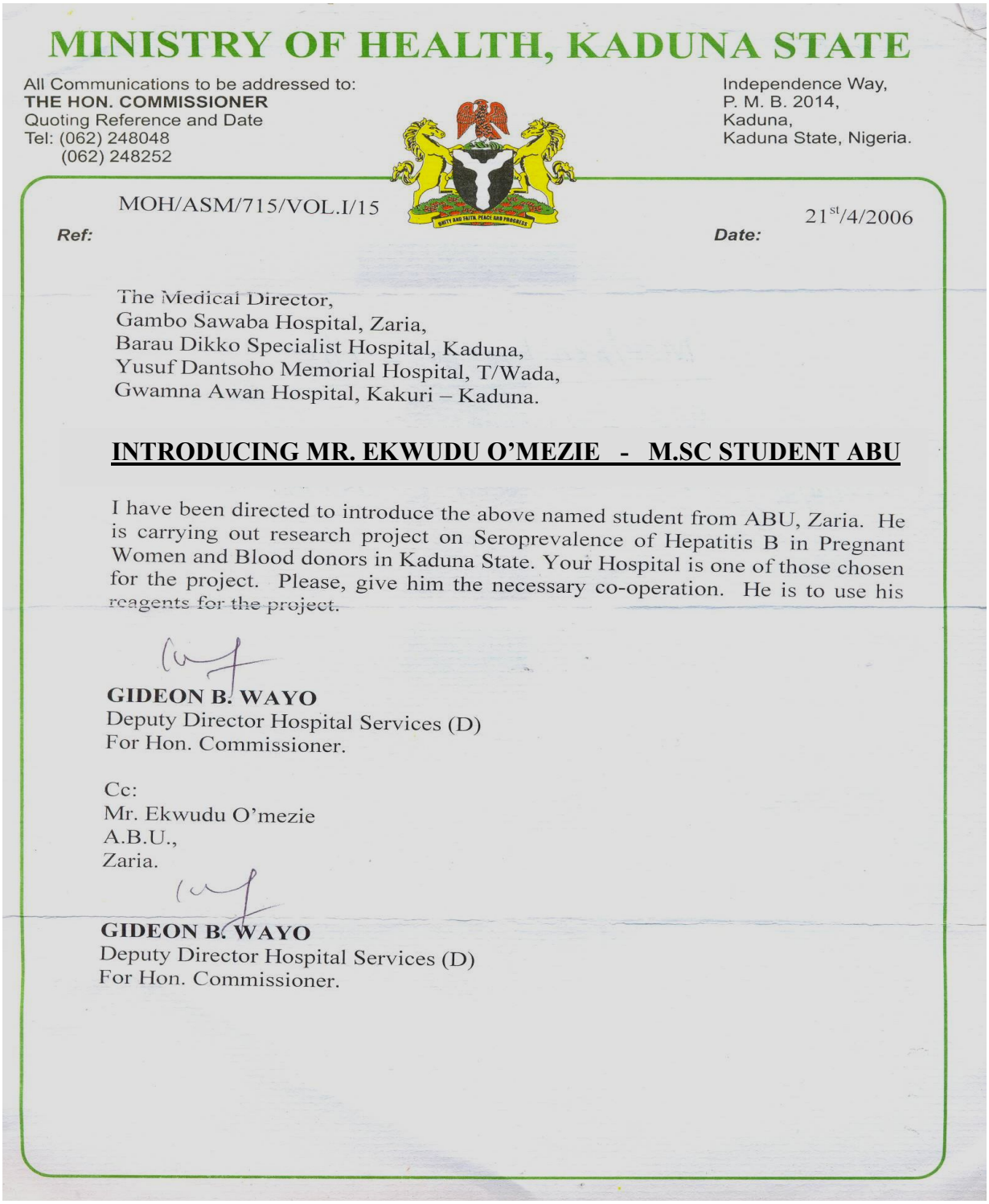
Thank you for your anticipated cooperation Sir.

Yours faithfully,

030906
Dr. S.E. Yakubu
Head of Department

APPENDIX B

CONSENT TO COLLECTION OF BLOOD SAMPLES AND DATA FROM PREGNANT WOMEN ATTENDING ANTE-NATAL CLINICS IN STATE GENERAL HOSPITALS.



MINISTRY OF HEALTH, KADUNA STATE

All Communications to be addressed to:
THE HON. COMMISSIONER
Quoting Reference and Date
Tel: (062) 248048
(062) 248252

Independence Way,
P. M. B. 2014,
Kaduna,
Kaduna State, Nigeria.

UNITY AND FAITH, PEACE AND PROGRESS

Ref: MOH/ASM/715/VOL.I/15

Date: 21st/4/2006

The Medical Director,
Gambo Sawaba Hospital, Zaria,
Barau Dikko Specialist Hospital, Kaduna,
Yusuf Dantsoho Memorial Hospital, T/Wada,
Gwamna Awan Hospital, Kakuri – Kaduna.

INTRODUCING MR. EKWUDU O'MEZIE - M.SC STUDENT ABU

I have been directed to introduce the above named student from ABU, Zaria. He is carrying out research project on Seroprevalence of Hepatitis B in Pregnant Women and Blood donors in Kaduna State. Your Hospital is one of those chosen for the project. Please, give him the necessary co-operation. He is to use his reagents for the project.

GIDEON B. WAYO
Deputy Director Hospital Services (D)
For Hon. Commissioner.

Cc:
Mr. Ekwudu O'mezie
A.B.U.,
Zaria.

GIDEON B. WAYO
Deputy Director Hospital Services (D)
For Hon. Commissioner.

APPENDIX C

Sample of the questionnaires used for Data collection .

QUESTIONNAIRE

This question is prepared in order to collect data on the prevalence of Hepatitis B virus among pregnant women in Kaduna State. The information obtained will be handled properly and confidentially for contribution to knowledge. Patients identity shall be kept with confidentiality.

Site:_____

Name: _____ Address: _____

Lab. NO: _____Date: _____

1. Age: _____

2. Sex: Male ☐ Female ☐

3. Highest education level:
   - Primary ☐
   - Secondary ☐
   - Tertiary ☐
   - Illiterate ☐

4. Occupation:
   - Civil Servant ☐
   - Business ☐
   - Unskilled Worker ☐
   - Unemployed ☐

5. Marital Status: Single ☐
   - Married ☐
   - ☐

Divorced

Separated ☐

Widowed ☐

6. Family Type Polygamy Monogamy ☐

7. No of Sexual Partners: 1 ☐ 2 ☐ 3 ☐ 4 or more

8. Use of Condom: Yes ☐ No ☐

9. Previous blood transfusion: Yes ☐ No ☐

If yes, how many times Once ☐ Twice or more ☐

If yes, for which condition? _____

10. Previous blood donations: Yes ☐ No ☐

If yes, how many times: Once ☐ Twice or more ☐

11. Previous surgery/ transplantation: Yes ☐ No ☐

12. History of dialysis: Yes ☐ No ☐

13. Do you smoke? Yes ☐ No ☐

If yes, how often: Once per week ☐ Twice ☐

15. History of Tribal mark/ tattooing: Yes ☐ No ☐

16. History of Vaccinations:

_____

_____

_____

_____

17. Tribe/ Ethnic group: _____

18. Have you ever had?

a) Glandular Swellings? Yes _____ No _____

b) Yellow eyes: Yes _____ No _____

19. Has your spouse ever had?

a) Glandular Swellings? Yes _____No _____

b) Yellow eyes: Yes _____ No _____

20. Do you

a) Patronize traditional barbers? Yes _____ No _____

b) Patronize pedicure/manicure salons? yes _____ No _____

c) Share clippers? yes _____ No _____

d) Share shaving sticks? Yes_____ No _____

e) Share toothbrushes? Yes _____ No _____

APPENDIX D

Data obtained from HBV analysis and questionnaire

SN	V1	V2	V3	V4	V5	V6	V7	V8	V9	V10	V11	V12	V13	V14	V15	V16	V17	V18
1	1	3	1	2	1	2	1	0	0	2	1	1	2	2	2	2	1	2
2	1	2	1	1	1	2	4	1	0	1	2	2	1	1	2	1	2	2
3	1	5	1	1	1	3	2	0	0	2	2	2	2	3	2	2	2	2
4	1	2	1	2	1	2	4	0	0	2	2	2	2	3	2	2	1	2
5	1	3	1	2	1	1	4	0	0	2	2	1	2	2	2	1	2	2
6	1	1	1	2	1	1	4	0	0	2	2	2	2	2	2	2	2	2
7	1	5	1	2	1	0	4	0	0	2	1	2	2	2	1	1	2	2
8	1	4	1	1	1	2	4	0	0	2	2	1	1	3	2	1	2	2
9	1	4	1	2	2	0	4	0	0	2	2	2	2	2	2	1	2	2
10	1	3	1	2	1	3	2	0	1	2	1	1	2	3	2	2	1	2
11	1	5	1	1	1	1	4	0	0	2	2	2	2	2	2	2	1	2
12	1	3	1	2	1	0	4	0	0	2	2	1	2	2	2	2	1	2
13	1	4	1	1	1	2	4	0	0	2	1	1	2	2	2	1	2	2
14	1	4	1	1	1	2	2	0	0	2	2	1	1	2	2	2	1	2
15	1	3	1	2	1	2	2	0	0	2	2	2	2	2	2	2	2	2
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17	1	5	1	2	2	1	2	0	0	2	2	1	2	2	2	1	2	2
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22	1	3	1	2	1	2	4	0	0	2	1	2	1	2	1	2	2	2
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24	1	1	1	1	1	1	2	0	0	2	1	2	2	2	2	1	2	2
25	1	4	1	2	2	2	2	0	0	2	2	2	2	2	2	2	2	2
26	1	4	1	2	1	2	4	0	0	2	2	2	2	4	2	2	2	2
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28	1	2	1	2	1	0	4	0	0	2	2	2	2	2	2	2	2	2
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35	1	3	1	1	1	2	3	0	0	2	2	2	2	4	2	2	2	2
36	1	3	1	1	1	3	2	0	0	2	2	1	2	3	2	2	2	2

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44	1	4	1	1	1	2	2	0	0	2	2	2	2	4	2	2	2	2
45	1	3	1	1	1	1	4	0	0	2	2	2	2	2	2	2	2	2
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51	1	4	1	1	1	0	2	0	0	2	2	2	2	3	2	1	2	2
52	1	2	1	1	1	0	4	0	0	2	2	2	2	2	2	2	2	2
53	1	4	1	2	1	2	2	0	0	2	1	2	2	4	2	2	2	2
54	1	2	1	1	1	2	3	0	0	2	2	2	2	2	2	1	2	2
55	1	3	1	1	1	1	2	0	0	2	2	2	2	2	2	2	2	2
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74	3	3	1	1	1	3	1	0	0	2	1	1	2	3	2	1	2	2
75	3	3	1	1	1	2	3	0	0	2	1	2	2	3	2	1	2	2
76	3	3	1	1	1	2 -		0	0	2	2	1	2	4	2	2	2	2
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Keys

V_1 – Hospital: 1= Yusuf Dantsoho Memorial Hospital

2= Barau Dikko Specialist Hospital

3 = Gwamna Awan General Hospital

V_2 – Ages: 1- 15-19yrs

2 – 20-24yrs

3 – 25-29yrs

4 – 30- 34yrs

5 – 35-40yrs

6 - 41yrs& above

V_3 Marital Status: 1 =Married

2 = Divorced

3 = Single

V_4 Family Type: 1 = Monogamy

2 = Polygamy

V_5 = No of Sexual Partners: 1 = One Sexual Partners

2=Two Sexual partner

3=Three Sexual partner

4= More than Three sexual partners

V_6 Educational Status: 0 = Illiterate

1 = Primary School

2 = Secondary School

3=Tertiary and Above

V_7 Occupation: 1 = Business

2 = Civil/ Public Servant

3 = Unskilled Workers

4 = Unemployed /Student

V_8 Previous Blood Transfusion: 0= No

1=yes

V_9 Alcohol Consumption: 0 =No

1=Yes

V_{10} Use of Condom:

1-Yes

2 – No

V_{11} Patronize Traditional barbers:

1- Yes

2-No

V_{12} Sharing of Clipper/shaving sticks and tooths brush:

1 – Yes

2 – No

V_{13} Visit manicure and pedicure bars:

1-Yes

2-No

(V_{14}) Tribe / Ethnic Group: 1-Igbo

2- Hausa / Fulani

3-Yoruba

4-Others in the North

5-Others in the South

(V_{15}) Have had yellow eyes before: 1-Yes

2-No.

(V_{16}) Anti HBs: 1-Postive

2-Negative

(V_{17}) Anti HBc: 1-Postive

2-Negative

(V_{18}) HBAg 1-Positive

2- Negative

The cut-off-values were determined using the AutoBio KC4 program, according to the following formular: Cut-off -value = 2.1 x Mean Negative Control

Anti-HBs MICROPLATE 1

$$\text{Mean Negative Control} = \frac{0.018 + 0.016}{2}$$

$$= \frac{0.034}{2}$$

$$= 0.017$$

$$\text{Cut-off-value} = 2.1 \times 0.017$$

$$= 0.0357$$

For Anti-HBs microtitreplate 1, any sample ;in which the absorbance was equal to or higher than 0.0357, was considered positive for Anti-HBs. Any sample in which the absorbance was lower than 0.0357, was considered negative for Anti-HBs.

Anti-HBs MICROPLATE 2.

$$\text{Mean Negative Control} = \frac{0.059 + 0.057}{2}$$

$$= \frac{0.116}{2}$$

$$= 0.058$$

$$\text{Cut-off-value} = 2.1 \times 0.058$$

$$= 0.1218$$

For Anti-HBs Microtitreplate 2, any sample in which the absorbance was equal to or higher than 0.1218, was considered positive for Anti HBs. Any sample in which the absorbance was lower than 0.1218, was considered negative for Anti-HBs.

Anti-HBs MICROPLATE 3.

Mean Negative Control $= \frac{0.051 + 0.080}{2}$

$= \frac{0.131}{2}$

$= 0.0655$

Cut-off-value $= 2.1 \times 0.0655$

$= 0.13755$

For Anti – HBs Microtitreplate 3, any sample in which the absorbance was equal to or higher than 0.13755, was considered positive for Anti-HBs. Any sample in which the absorbance was lower than 0.13755, was, considered negative for Anti-HBs.

APPENDIX F

DETERMINATION OF CUT-OFF VALUE AND INTERPRETATION OF TESTS' RESULTS FOR ANTI-HBc DETECTION ASSAY

The cut-off values were determined using the AutoBio KC4 program, according to the following formular:

$$\text{Cut-off-value} = \left(\frac{\text{Mean Negative Control} + \text{Mean Positive Control}}{2}\right.$$

Anti-HBc MICROPLATE 1

$$\text{Mean Negative Control} = \frac{2.576 + 2.619}{2}$$

$$= \frac{5.195}{2}$$

$$= 2.5975$$

$$\text{Mean Positive Control} = \frac{0.047 + 0.031}{2}$$

$$= \frac{0.078}{2}$$

$$= 0.039$$

$$\text{Cut-off-value} = \frac{2.5975 + 0.039}{2}$$

$$= \frac{2.6365}{2}$$

$$= 1.31825.$$

For Anti –HBc Microtitreplate 1, any sample in which the absorbance was equal to or higher than 1.31825, was considered positive for ;Anti-HBc. Any sample in which the absorbance was slower than 1.31825, was considered negative for Anti-HBc.

Anti-HBc MICROPLATE 2.

Mean Negative Control $= \frac{1.906 + 1.811}{2}$

$= \frac{3.717}{2}$

$= 1.8585$

Mean Positive Control $= \frac{0.024 + 0.023}{2}$

$= \frac{0.047}{2}$

$= 0.0235$

Cut-off-value $= \frac{0.0235 + 1.8585}{2}$

$= \frac{1.882}{2}$

$= 0.941$

For Anti -HBc microtitreplate 2, any sample in which the absorbance was equal to or higher than 0.941, was considered positive for Anti-HBc. Any sample in which the absorbance was lower than 0.941, was considered negative for Anti-HBc.

Anti-HBc MICROPLATE 3.

Mean Negative Control $= \frac{1.209 + 1.266}{2}$

$$= \frac{2.475}{2}$$

$$= 1.2375$$

Mean Positive Control $$= \frac{0.005 + 0.028}{2}$$

$$= \frac{0.033}{2}$$

$$= 0.0165$$

Cut-Off-Value $$= \frac{1.2375 + 0.0165}{2}$$

$$= \frac{1.254}{2}$$

$$= 0.627$$

For Anti-HBc Microtitreplate 3, any sample in which the absorbance was equal to or higher than 0.627, was considered positive for Anthi-HBc. Any sample in which the absorbance was lower than 0.627 was considered negative for Anti-HBc.

APPENDIX G

DETERMINATION OF CUT-OFF-VALUES AND INTERPRETATION OF TESTS' RESULTS FOR HBeAg DETECTION ASSAY.

The cut-Off-Values were determined using the AutoBio KC4 program, according to the following formular:

Cut-Off-Value = 2.1 x Mean Negative Control

HBeAg MICROPLATE 1

Mean Negative Control $= \frac{0.025 + 0.048}{2}$

$= \frac{0.073}{2}$

$= 0.0365$

Cut-Off-Value = 2.1 x 0.0365

= 0.07665

For HBeAg Microtitreplate 1, any sample in which the absorbance was equal to or higher than 0.07665 was considered positive for HBeAg. Any sample in which the absorbance was lower than 0.07665 was considered negative for HBeAg.

HBeAg MICROPLATE 2.

Mean Negative Control $= \frac{0.030 + 0.029}{2}$

$= \frac{0.059}{2}$

$= 0.0295$

Cut-Off-Value $= 2.1 \times 0.0295$

$= 0.06195$

For HBeAg Microtitreplate 2, any sample in which the absorbance was equal to or higher than 0.06195 was considered positive for HbeAg. Any sample in which the absorbance was lower than 0.06195, was considered negative for HbeAg.

HBeAg MICROPLATE 3.

Mean Negative Control $= \frac{0.023 + 0.017}{2}$

$= \frac{0.04}{2}$

$= 0.02$

Cut-Off-Value $= 0.02 \times 2.1$

$= 0.042$

For HBeAg Microtitreplate 3, any sample in which the absorbance was equal to or higher than 0.042 was considered positive for HBeAg. Any sample in which the absorbance was lower than 0.042, was considered negative for HBeAg.

APPENDIX H

Statistical Data Analysis

V1 HOSPITAL

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
YD	1	92	33.6	33.6	33.6
BD	2	90	32.8	32.8	66.4
GA	3	92	33.6	33.6	100.0
	TOTAL	274	100.0	100.0	

Mean 2.000 Std Dev .821 Minimum 1.000
Maximum 3.000

Valid Cases 274 Missing Cases 0

V2 AGE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
15-19YRS	1	24	8.8	8.8	8.8
20-24YRS	2	68	24.8	24.8	33.6
25-29YRS	3	95	34.7	34.7	68.2
30-34YRS	4	66	24.1	24.1	92.3
35-40YRS	5	14	5.1	5.1	97.4
40YRS ABOVE	6	7	2.6	2.6	100.0
	TOTAL	274	100.0	100.0	

Mean 2.996 Std Dev 1.131 Minimum 1.000
Maximum 6.000

Valid Cases 274 Missing Cases 0

V3 MARITAL STATUS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
MARRIED	1	254	92.7	92.7	92.7
SINGLE	2	13	4.7	4.7	97.4
DIVORCED	4	7	2.6	2.6	100.0
	TOTAL	274	100.0	100.0	

Mean 1.124 Std Dev .513 Minimum 1.000
Maximum 4.000

Valid Cases 274 Missing Cases 0

V7 OCCUPATION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	11	4.0	4.0	4.0
CIVIL SERVANT	1	30	10.9	10.9	15.0
BUSINESS	2	84	30.7	30.7	45.6
UNSKILLED	3	40	14.6	14.6	60.2
UNEMPLLOYED	4	109	39.8	39.8	100.0
	TOTAL	274	100.0	100.0	

Mean 2.752 Std Dev 1.203 Minimum .000
Maximum 4.000

Valid Cases 274 Missing Cases 0

- -

V8 PREVIOUS BLOOD TRANSFUSION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	245	89.4	89.4	89.4
YES	1	29	10.6	10.6	100.0
	TOTAL	274	100.0	100.0	

Mean .106 Std Dev .308 Minimum .000
Maximum 1.000

Valid Cases 274 Missing Cases 0

- -

V9 ALCOHOLIC/SMOKING

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	257	93.8	93.8	93.8
YES	1	17	6.2	6.2	100.0
	TOTAL	274	100.0	100.0	

Mean .062 Std Dev .242 Minimum .000
Maximum 1.000

Valid Cases 274 Missing Cases 0

Valid Cases 274 Missing Cases 0

V4 FAMILY TYPE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	1	.4	.4	.4
MONOGAMY	1	226	82.5	82.5	82.8
POLYGAMY	2	47	17.2	17.2	100.0
	TOTAL	274	100.0	100.0	

Mean 1.168 Std Dev .384 Minimum .000
Maximum 2.000

Valid Cases 274 Missing Cases 0

- -

V5 NO OF SEXUAL PARTNERS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	5	1.8	1.8	1.8
ONE	1	183	66.8	66.8	68.6
TWO	2	55	20.1	20.1	88.7
THREE	3	11	4.0	4.0	92.7
MORE THAN 3	4	20	7.3	7.3	100.0
	TOTAL	274	100.0	100.0	

Mean 1.482 Std Dev .899 Minimum .000
Maximum 4.000

Valid Cases 274 Missing Cases 0

- -

V6 EDUCATIONAL STATUS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
ILLITERATE	0	34	12.4	12.4	12.4
PRIMARY	1	50	18.2	18.2	30.7
SECONDARY	2	142	51.8	51.8	82.5
TERTIARY	3	48	17.5	17.5	100.0
	TOTAL	274	100.0	100.0	

Mean 1.745 Std Dev .890 Minimum .000
Maximum 3.000

Valid Cases 274 Missing Cases 0

V7 OCCUPATION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	11	4.0	4.0	4.0
CIVIL SERVANT	1	30	10.9	10.9	15.0
BUSINESS	2	84	30.7	30.7	45.6
UNSKILLED	3	40	14.6	14.6	60.2
UNEMPLLOYED	4	109	39.8	39.8	100.0
	TOTAL	274	100.0	100.0	

Mean 2.752 Std Dev 1.203 Minimum .000
Maximum 4.000

Valid Cases 274 Missing Cases 0

- -

V8 PREVIOUS BLOOD TRANSFUSION

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	245	89.4	89.4	89.4
YES	1	29	10.6	10.6	100.0
	TOTAL	274	100.0	100.0	

Mean .106 Std Dev .308 Minimum .000
Maximum 1.000

Valid Cases 274 Missing Cases 0

- -

V9 ALCOHOLIC/SMOKING

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO	0	257	93.8	93.8	93.8
YES	1	17	6.2	6.2	100.0
	TOTAL	274	100.0	100.0	

Mean .062 Std Dev .242 Minimum .000
Maximum 1.000

Valid Cases 274 Missing Cases 0

V10 USE OF CONDOME

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	5	1.8	1.8	1.8
YES	1	29	10.6	10.6	12.4
NO	2	240	87.6	87.6	100.0
	TOTAL	274	100.0	100.0	

Mean 1.858 Std Dev .399 Minimum .000
Maximum 2.000

Valid Cases 274 Missing Cases 0

- -

V11 USE TRADITIONAL BARBERS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	1	.4	.4	.4
YES	1	32	11.7	11.7	12.0
NO	2	241	88.0	88.0	100.0
	TOTAL	274	100.0	100.0	

Mean 1.876 Std Dev .341 Minimum .000
Maximum 2.000

Valid Cases 274 Missing Cases 0

- -

V12 SHARE CLIPPERS

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	6	2.2	2.2	2.2
YES	1	49	17.9	17.9	20.1
NO	2	219	79.9	79.9	100.0
	TOTAL	274	100.0	100.0	

Mean 1.777 Std Dev .467 Minimum .000
Maximum 2.000

Valid Cases 274 Missing Cases 0

V13 MANICURE/PENDICURE

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	1	.4	.4	.4
YES	1	17	6.2	6.2	6.6
NO	2	256	93.4	93.4	100.0
	TOTAL	274	100.0	100.0	

Mean 1.931 Std Dev .269 Minimum .000
Maximum 2.000

Valid Cases 274 Missing Cases 0

- -

V14 ETHNIC GROUP

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	3	1.1	1.1	1.1
IGBO	1	16	5.8	5.8	6.9
HAUSA/FULANI	2	103	37.6	37.6	44.5
YORUBA	3	34	12.4	12.4	56.9
OTHER NORTH	4	108	39.4	39.4	96.4
OTHER SOUTHS	5	10	3.6	3.6	100.0
	TOTAL	274	100.0	100.0	

Mean 2.942 Std Dev 1.118 Minimum .000
Maximum 5.000

Valid Cases 274 Missing Cases 0

- -

V15 HAVE YELLOW EYES

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NO RESPONSE	0	1	.4	.4	.4
YES	1	27	9.9	9.9	10.2
NO	2	246	89.8	89.8	100.0
	TOTAL	274	100.0	100.0	

Mean 1.894 Std Dev .320 Minimum .000
Maximum 2.000

Valid Cases 274 Missing Cases 0

V16 MATURE VIRAL

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NEGATIVE	0	197	71.9	71.9	71.9
POSITIVE	1	77	28.1	28.1	100.0
	TOTAL	274	100.0	100.0	

Mean	.281	Std Dev	.450	Minimum	.000
Maximum	1.000				

Valid Cases 274 Missing Cases 0

- -

V17 ANTI-HBC

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NEGATIVE	0	217	79.2	79.2	79.2
POSITIVE	1	57	20.8	20.8	100.0
	TOTAL	274	100.0	100.0	

Mean	.208	Std Dev	.407	Minimum	.000
Maximum	1.000				

Valid Cases 274 Missing Cases 0

- -

V18 HIGH LEVEL

Value Label	Value	Frequency	Percent	Valid Percent	Cum Percent
NEGATIVE	0	268	97.8	97.8	97.8
POSITIVE	1	6	2.2	2.2	100.0
	TOTAL	274	100.0	100.0	

Mean	.022	Std Dev	.147	Minimum	.000
Maximum	1.000				

Valid Cases 274 Missing Cases 0

Crosstabulation: V16 MATURE VIRAL
By V1 HOSPITAL

V16	V1->	1	2	3	Row Total
0 NEGATIVE	Count	63	61	73	197
	Row Pct	32.0	31.0	37.1	71.9
	Col Pct	68.5	67.8	79.3	
	Tot Pct	23.0	22.3	26.6	
1 POSITIVE	Count	29	29	19	77
	Row Pct	37.7	37.7	24.7	28.1
	Col Pct	31.5	32.2	20.7	
	Tot Pct	10.6	10.6	6.9	
Column Total		92	90	92	274
		33.6	32.8	33.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.81579	2	.1484	25.292	None

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V2 AGE

V16	V2->	1	2	3	4	5	Row Total
0 NEGATIVE	Count	20	44	68	50	10	197
	Row Pct	10.2	22.3	34.5	25.4	5.1	71.9
	Col Pct	83.3	64.7	71.6	75.8	71.4	
	Tot Pct	7.3	16.1	24.8	18.2	3.6	
1 POSITIVE	Count	4	24	27	16	4	77
	Row Pct	5.2	31.2	35.1	20.8	5.2	28.1
	Col Pct	16.7	35.3	28.4	24.2	28.6	
	Tot Pct	1.5	8.8	9.9	5.8	1.5	
Column Total		24	68	95	66	14	274
(Continued)		8.8	24.8	34.7	24.1	5.1	100.0

Crosstabulation: V16 MATURE VIRAL
By V2 AGE

V16 \ V2->	40YRS AB OVE (6)	Row Total
0 NEGATIVE	5	197
	2.5	71.9
	71.4	
	1.8	
1 POSITIVE	2	77
	2.6	28.1
	28.6	
	.7	
Column Total	7	274
	2.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.78783	5	.5803	1.967	2 OF 12 (16.7%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V3 MARITAL STATUS

V16 \ V3->	MARRIED (1)	SINGLE (2)	DIVORCED (4)	Row Total
0 NEGATIVE	184	8	5	197
	93.4	4.1	2.5	71.9
	72.4	61.5	71.4	
	67.2	2.9	1.8	
1 POSITIVE	70	5	2	77
	90.9	6.5	2.6	28.1
	27.6	38.5	28.6	
	25.5	1.8	.7	
Column Total	254	13	7	274
	92.7	4.7	2.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.72833	2	.6948	1.967	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V4 FAMILY TYPE

V16	V4->	NO RESPO NSE 0	MONOGAMY 1	POLYGAMY 2	Row Total
0 NEGATIVE	Count	1	164	32	197
	Row Pct	.5	83.2	16.2	71.9
	Col Pct	100.0	72.6	68.1	
	Tot Pct	.4	59.9	11.7	
1 POSITIVE	Count		62	15	77
	Row Pct		80.5	19.5	28.1
	Col Pct		27.4	31.9	
	Tot Pct		22.6	5.5	
Column Total		1 .4	226 82.5	47 17.2	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.77901	2	.6774	.281	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V5 NO OF SEXUAL PARTNERS

V16	V5->	NO RESPO NSE 0	ONE 1	TWO 2	THREE 3	MORE THA N 3 4	Row Total
0 NEGATIVE	Count	5	132	43	6	11	197
	Row Pct	2.5	67.0	21.8	3.0	5.6	71.9
	Col Pct	100.0	72.1	78.2	54.5	55.0	
	Tot Pct	1.8	48.2	15.7	2.2	4.0	
1 POSITIVE	Count		51	12	5	9	77
	Row Pct		66.2	15.6	6.5	11.7	28.1
	Col Pct		27.9	21.8	45.5	45.0	
	Tot Pct		18.6	4.4	1.8	3.3	
Column Total		5 1.8	183 66.8	55 20.1	11 4.0	20 7.3	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
7.49987	4	.1117	1.405	3 OF 10 (30.0%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V6 EDUCATIONAL STATUS

V6->	Count / Row Pct / Col Pct / Tot Pct	ILLITERATE 0	PRIMARY 1	SECONDARY 2	TERTIARY 3	Row Total
V16						
0 NEGATIVE		27	36	97	37	197
		13.7	18.3	49.2	18.8	71.9
		79.4	72.0	68.3	77.1	
		9.9	13.1	35.4	13.5	
1 POSITIVE		7	14	45	11	77
		9.1	18.2	58.4	14.3	28.1
		20.6	28.0	31.7	22.9	
		2.6	5.1	16.4	4.0	
Column Total		34	50	142	48	274
		12.4	18.2	51.8	17.5	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
2.49389	3	.4764	9.555	None

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V7 OCCUPATION

V7->	Count / Row Pct / Col Pct / Tot Pct	NO RESPONSE 0	CIVIL SERVANT 1	BUSINESS 2	UNSKILLED 3	UNEMPLLOYED 4	Row Total
V16							
0 NEGATIVE		9	24	63	26	75	197
		4.6	12.2	32.0	13.2	38.1	71.9
		81.8	80.0	75.0	65.0	68.8	
		3.3	8.8	23.0	9.5	27.4	
1 POSITIVE		2	6	21	14	34	77
		2.6	7.8	27.3	18.2	44.2	28.1
		18.2	20.0	25.0	35.0	31.2	
		.7	2.2	7.7	5.1	12.4	
Column Total		11	30	84	40	109	274
		4.0	10.9	30.7	14.6	39.8	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.36778	4	.4983	3.091	1 OF 10 (10.0%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V8 PREVIOUS BLOOD TRANSFUSION

V16	V8->	NO (0)	YES (1)	Row Total
NEGATIVE (0)	Count	177	20	197
	Row Pct	89.8	10.2	71.9
	Col Pct	72.2	69.0	
	Tot Pct	64.6	7.3	
POSITIVE (1)	Count	68	9	77
	Row Pct	88.3	11.7	28.1
	Col Pct	27.8	31.0	
	Tot Pct	24.8	3.3	
Column Total		245	29	274
		89.4	10.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.02343	1	.8783	8.150	None
.13802	1	.7103	(Before Yates Correction)	

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V9 ALCOHOLIC/SMOKING

V16	V9->	NO (0)	YES (1)	Row Total
NEGATIVE (0)	Count	182	15	197
	Row Pct	92.4	7.6	71.9
	Col Pct	70.8	88.2	
	Tot Pct	66.4	5.5	
POSITIVE (1)	Count	75	2	77
	Row Pct	97.4	2.6	28.1
	Col Pct	29.2	11.8	
	Tot Pct	27.4	.7	
Column Total		257	17	274
		93.8	6.2	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.60983	1	.2045	4.777	1 of 4 (25.0%)

2.39431	1	.1218	(Before Yates Correction)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V10 USE OF CONDOME

V16	V10->	NO RESPO/NSE 0	YES 1	NO 2	Row Total
NEGATIVE	0	3	22	172	197
		1.5	11.2	87.3	71.9
		60.0	75.9	71.7	
		1.1	8.0	62.8	
POSITIVE	1	2	7	68	77
		2.6	9.1	88.3	28.1
		40.0	24.1	28.3	
		.7	2.6	24.8	
Column Total		5	29	240	274
		1.8	10.6	87.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.58221	2	.7474	1.405	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V11 USE TRADITIONAL BARBERS

V16	V11->	NO RESPO/NSE 0	YES 1	NO 2	Row Total
NEGATIVE	0	1	19	177	197
		.5	9.6	89.8	71.9
		100.0	59.4	73.4	
		.4	6.9	64.6	
POSITIVE	1		13	64	77
			16.9	83.1	28.1
			40.6	26.6	
			4.7	23.4	
Column Total		1	32	241	274
		.4	11.7	88.0	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.15971	2	.2060	.281	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V12 SHARE CLIPPERS

V16	V12->	NO RESPO NSE 0	YES 1	NO 2	Row Total
NEGATIVE	Count	4	30	163	197
	Row Pct	2.0	15.2	82.7	71.9
	Col Pct	66.7	61.2	74.4	
	Tot Pct	1.5	10.9	59.5	
POSITIVE	Count	2	19	56	77
	Row Pct	2.6	24.7	72.7	28.1
	Col Pct	33.3	38.8	25.6	
	Tot Pct	.7	6.9	20.4	
Column Total		6 2.2	49 17.9	219 79.9	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.53857	2	.1705	1.686	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V13 MANICURE/PENDICURE

V16	V13->	NO RESPO NSE 0	YES 1	NO 2	Row Total
NEGATIVE	Count	1	12	184	197
	Row Pct	.5	6.1	93.4	71.9
	Col Pct	100.0	70.6	71.9	
	Tot Pct	.4	4.4	67.2	
POSITIVE	Count		5	72	77
	Row Pct		6.5	93.5	28.1
	Col Pct		29.4	28.1	
	Tot Pct		1.8	26.3	
Column Total		1 .4	17 6.2	256 93.4	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.40536	2	.8165	.281	3 OF 6 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V14 ETHNIC GROUP

V14-> / V16	Count / Row Pct / Col Pct / Tot Pct	NO RESPONSE 0	IGBO 1	HAUSA/FULANI 2	YORUBA 3	OTHER NORTH 4	Row Total
NEGATIVE	0	2	9	78	23	76	197
		1.0	4.6	39.6	11.7	38.6	71.9
		66.7	56.3	75.7	67.6	70.4	
		.7	3.3	28.5	8.4	27.7	
POSITIVE	1	1	7	25	11	32	77
		1.3	9.1	32.5	14.3	41.6	28.1
		33.3	43.8	24.3	32.4	29.6	
		.4	2.6	9.1	4.0	11.7	
Column Total		3	16	103	34	108	274
		1.1	5.8	37.6	12.4	39.4	100.0

(Continued)

Crosstabulation: V16 MATURE VIRAL
By V14 ETHNIC GROUP

V14-> / V16	Count / Row Pct / Col Pct / Tot Pct	OTHER SOUTHS 5	Row Total
NEGATIVE	0	9	197
		4.6	71.9
		90.0	
		3.3	
POSITIVE	1	1	77
		1.3	28.1
		10.0	
		.4	
Column Total		10	274
		3.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
4.77813	5	.4436	.843	4 OF 12 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V16 MATURE VIRAL
By V15 HAVE YELLOW EYES

V16	V15->	NO RESPONSE 0	YES 1	NO 2	Row Total
NEGATIVE	0		16	181	197
			8.1	91.9	71.9
			59.3	73.6	
			5.8	66.1	
POSITIVE	1	1	11	65	77
		1.3	14.3	84.4	28.1
		100.0	40.7	26.4	
		.4	4.0	23.7	
	Column Total	1 .4	27 9.9	246 89.8	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
5.03637	2	.0806	.281	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V1 HOSPITAL

V17	V1->	YD 1	BD 2	GA 3	Row Total
NEGATIVE	0	69	74	74	217
		31.8	34.1	34.1	79.2
		75.0	82.2	80.4	
		25.2	27.0	27.0	
POSITIVE	1	23	16	18	57
		40.4	28.1	31.6	20.8
		25.0	17.8	19.6	
		8.4	5.8	6.6	
	Column Total	92 33.6	90 32.8	92 33.6	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.56913	2	.4563	18.723	None

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V2 AGE

V17	V2->	15-19YRS (1)	20-24YRS (2)	25-29YRS (3)	30-34YRS (4)	35-40YRS (5)	Row Total
0 NEGATIVE	Count	16	55	74	54	13	217
	Row Pct	7.4	25.3	34.1	24.9	6.0	79.2
	Col Pct	66.7	80.9	77.9	81.8	92.9	
	Tot Pct	5.8	20.1	27.0	19.7	4.7	
1 POSITIVE	Count	8	13	21	12	1	57
	Row Pct	14.0	22.8	36.8	21.1	1.8	20.8
	Col Pct	33.3	19.1	22.1	18.2	7.1	
	Tot Pct	2.9	4.7	7.7	4.4	.4	
Column Total		24	68	95	66	14	274
		8.8	24.8	34.7	24.1	5.1	100.0

(Continued)

Crosstabulation: V17 ANTI-HBC
By V2 AGE

V17	V2->	40YRS AB OVE (6)	Row Total
0 NEGATIVE	Count	5	217
	Row Pct	2.3	79.2
	Col Pct	71.4	
	Tot Pct	1.8	
1 POSITIVE	Count	2	57
	Row Pct	3.5	20.8
	Col Pct	28.6	
	Tot Pct	.7	
Column Total		7	274
		2.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
4.61950	5	.4641	1.456	3 OF 12 (25.0%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V3 MARITAL STATUS

V17 / V3->	MARRIED 1	SINGLE 2	DIVORCED 4	Row Total
0 NEGATIVE	201	10	6	217
	92.6	4.6	2.8	79.2
	79.1	76.9	85.7	
	73.4	3.6	2.2	
1 POSITIVE	53	3	1	57
	93.0	5.3	1.8	20.8
	20.9	23.1	14.3	
	19.3	1.1	.4	
Column Total	254	13	7	274
	92.7	4.7	2.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.22188	2	.8950	1.456	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V4 FAMILY TYPE

V17 / V4->	NO RESPO NSE 0	MONOGAMY 1	POLYGAMY 2	Row Total
0 NEGATIVE	1	180	36	217
	.5	82.9	16.6	79.2
	100.0	79.6	76.6	
	.4	65.7	13.1	
1 POSITIVE		46	11	57
		80.7	19.3	20.8
		20.4	23.4	
		16.8	4.0	
Column Total	1	226	47	274
	.4	82.5	17.2	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.48336	2	.7853	.208	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V5 NO OF SEXUAL PARTNERS

V17	V5->	NO RESPONSE 0	ONE 1	TWO 2	THREE 3	MORE THAN 3 4	Row Total
NEGATIVE	0	5	141	46	9	16	217
		2.3	65.0	21.2	4.1	7.4	79.2
		100.0	77.0	83.6	81.8	80.0	
		1.8	51.5	16.8	3.3	5.8	
POSITIVE	1		42	9	2	4	57
			73.7	15.8	3.5	7.0	20.8
			23.0	16.4	18.2	20.0	
			15.3	3.3	.7	1.5	
Column Total		5 1.8	183 66.8	55 20.1	11 4.0	20 7.3	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
2.53740	4	.6380	1.040	4 OF 10 (40.0%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V6 EDUCATIONAL STATUS

V17	V6->	ILLITERATE 0	PRIMARY 1	SECONDARY 2	TERTIARY 3	Row Total
NEGATIVE	0	24	38	119	36	217
		11.1	17.5	54.8	16.6	79.2
		70.6	76.0	83.8	75.0	
		8.8	13.9	43.4	13.1	
POSITIVE	1	10	12	23	12	57
		17.5	21.1	40.4	21.1	20.8
		29.4	24.0	16.2	25.0	
		3.6	4.4	8.4	4.4	
Column Total		34 12.4	50 18.2	142 51.8	48 17.5	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
4.18119	3	.2426	7.073	None

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V7 OCCUPATION

V17	V7->	NO RESPONSE 0	CIVIL SERVANT 1	BUSINESS 2	UNSKILLED 3	UNEMPLLOYED 4	Row Total
NEGATIVE	0	8	24	68	34	83	217
		3.7	11.1	31.3	15.7	38.2	79.2
		72.7	80.0	81.0	85.0	76.1	
		2.9	8.8	24.8	12.4	30.3	
POSITIVE	1	3	6	16	6	26	57
		5.3	10.5	28.1	10.5	45.6	20.8
		27.3	20.0	19.0	15.0	23.9	
		1.1	2.2	5.8	2.2	9.5	
	Column Total	11 4.0	30 10.9	84 30.7	40 14.6	109 39.8	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.88143	4	.7576	2.288	1 OF 10 (10.0%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V8 PREVIOUS BLOOD TRANSFUSION

V17	V8->	NO 0	YES 1	Row Total
NEGATIVE	0	191	26	217
		88.0	12.0	79.2
		78.0	89.7	
		69.7	9.5	
POSITIVE	1	54	3	57
		94.7	5.3	20.8
		22.0	10.3	
		19.7	1.1	
	Column Total	245 89.4	29 10.6	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.50166	1	.2204	6.033	None
2.15305	1	.1423	(Before Yates Correction)	

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V9 ALCOHOLIC/SMOKING

V17	V9->	NO (0)	YES (1)	Row Total
NEGATIVE	0	207	10	217
		95.4	4.6	79.2
		80.5	58.8	
		75.5	3.6	
POSITIVE	1	50	7	57
		87.7	12.3	20.8
		19.5	41.2	
		18.2	2.6	
Column Total		257	17	274
		93.8	6.2	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.34307	1	.0675	3.536	1 of 4 (25.0%)
4.56632	1	.0326	(Before Yates Correction)	

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V10 USE OF CONDOME

V17	V10->	NO RESPONSE (0)	YES (1)	NO (2)	Row Total
NEGATIVE	0	4	23	190	217
		1.8	10.6	87.6	79.2
		80.0	79.3	79.2	
		1.5	8.4	69.3	
POSITIVE	1	1	6	50	57
		1.8	10.5	87.7	20.8
		20.0	20.7	20.8	
		.4	2.2	18.2	
Column Total		5	29	240	274
		1.8	10.6	87.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.00232	2	.9988	1.040	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V11 USE TRADITIONAL BARBERS

V11->	Count / Row Pct / Col Pct / Tot Pct	NO RESPO NSE 0	YES 1	NO 2	Row Total
V17					
0 NEGATIVE		1	24	192	217
		.5	11.1	88.5	79.2
		100.0	75.0	79.7	
		.4	8.8	70.1	
1 POSITIVE			8	49	57
			14.0	86.0	20.8
			25.0	20.3	
			2.9	17.9	
Column Total		1 .4	32 11.7	241 88.0	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.63727	2	.7271	.208	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V12 SHARE CLIPPERS

V12->	Count / Row Pct / Col Pct / Tot Pct	NO RESPO NSE 0	YES 1	NO 2	Row Total
V17					
0 NEGATIVE		5	37	175	217
		2.3	17.1	80.6	79.2
		83.3	75.5	79.9	
		1.8	13.5	63.9	
1 POSITIVE		1	12	44	57
		1.8	21.1	77.2	20.8
		16.7	24.5	20.1	
		.4	4.4	16.1	
Column Total		6 2.2	49 17.9	219 79.9	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.53389	2	.7657	1.248	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V14 ETHNIC GROUP

V17	V14-> OTHER SOUTHS (5)	Row Total
0 NEGATIVE	Count 7 / Row Pct 3.2 / Col Pct 70.0 / Tot Pct 2.6	217 / 79.2
1 POSITIVE	Count 3 / Row Pct 5.3 / Col Pct 30.0 / Tot Pct 1.1	57 / 20.8
Column Total	10 / 3.6	274 / 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.81184	5	.5768	.624	4 OF 12 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V17 ANTI-HBC
By V15 HAVE YELLOW EYES

V17	V15-> NO RESPONSE (0)	YES (1)	NO (2)	Row Total
0 NEGATIVE	Count 1 / Row Pct .5 / Col Pct 100.0 / Tot Pct .4	21 / 9.7 / 77.8 / 7.7	195 / 89.9 / 79.3 / 71.2	217 / 79.2
1 POSITIVE	Count 0 / Row Pct 0 / Col Pct 0 / Tot Pct 0	6 / 10.5 / 22.2 / 2.2	51 / 89.5 / 20.7 / 18.6	57 / 20.8
Column Total	1 / .4	27 / 9.9	246 / 89.8	274 / 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.29644	2	.8622	.208	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V1 HOSPITAL

V18	Count / Row Pct / Col Pct / Tot Pct	V1-> 1 YD	2 BD	3 GA	Row Total
NEGATIVE	0	91 34.0 98.9 33.2	86 32.1 95.6 31.4	91 34.0 98.9 33.2	268 97.8
POSITIVE	1	1 16.7 1.1 .4	4 66.7 4.4 1.5	1 16.7 1.1 .4	6 2.2
	Column Total	92 33.6	90 32.8	92 33.6	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
3.18093	2	.2038	1.971	3 OF 6 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V2 AGE

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V18	Count / Row Pct / Col Pct / Tot Pct	V2-> 1 15-19YRS	2 20-24YRS	3 25-29YRS	4 30-34YRS	5 35-40YRS	Row Total
NEGATIVE	0	24 9.0 100.0 8.8	66 24.6 97.1 24.1	93 34.7 97.9 33.9	64 23.9 97.0 23.4	14 5.2 100.0 5.1	268 97.8
POSITIVE	1		2 33.3 2.9 .7	2 33.3 2.1 .7	2 33.3 3.0 .7		6 2.2
(Continued)	Column Total	24 8.8	68 24.8	95 34.7	66 24.1	14 5.1	274 100.0

Crosstabulation: V18 HIGH LEVEL
By V2 AGE

V18 / V2->	40YRS AB OVE (6)	Row Total
0 NEGATIVE	7 2.6 100.0 2.6	268 97.8
1 POSITIVE		6 2.2
Column Total	7 2.6	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.40758	5	.9235	.153	6 OF 12 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V3 MARITAL STATUS

V18 / V3->	MARRIED (1)	SINGLE (2)	DIVORCED (4)	Row Total
0 NEGATIVE	248 92.5 97.6 90.5	13 4.9 100.0 4.7	7 2.6 100.0 2.6	268 97.8
1 POSITIVE	6 100.0 2.4 2.2			6 2.2
Column Total	254 92.7	13 4.7	7 2.6	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.48302	2	.7854	.153	2 OF 6 (33.3%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V4 FAMILY TYPE

V18 \ V4->	NO RESPONSE 0	MONOGAMY 1	POLYGAMY 2	Row Total
0 NEGATIVE		221 82.5 97.8 80.7	47 17.5 100.0 17.2	268 97.8
1 POSITIVE	1 16.7 100.0 .4	5 83.3 2.2 1.8		6 2.2
Column Total	1 .4	226 82.5	47 17.2	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
45.71945	2	.0000	.022	4 OF 6 (66.7%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V5 NO OF SEXUAL PARTNERS

V18 \ V5->	NO RESPONSE 0	ONE 1	TWO 2	THREE 3	MORE THAN 3 4	Row Total
0 NEGATIVE	3 1.1 60.0 1.1	181 67.5 98.9 66.1	54 20.1 98.2 19.7	10 3.7 90.9 3.6	20 7.5 100.0 7.3	268 97.8
1 POSITIVE	2 33.3 40.0 .7	2 33.3 1.1 .7	1 16.7 1.8 .4	1 16.7 9.1 .4		6 2.2
Column Total	5 1.8	183 66.8	55 20.1	11 4.0	20 7.3	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
37.33079	4	.0000	.109	6 OF 10 (60.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V4 FAMILY TYPE

V18	V4-> NO RESPONSE 0	MONOGAMY 1	POLYGAMY 2	Row Total
0 NEGATIVE		221	47	268
		82.5	17.5	97.8
		97.8	100.0	
		80.7	17.2	
1 POSITIVE	1	5		6
	16.7	83.3		2.2
	100.0	2.2		
	.4	1.8		
Column Total	1	226	47	274
	.4	82.5	17.2	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
45.71945	2	.0000	.022	4 OF 6 (66.7%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V5 NO OF SEXUAL PARTNERS

V18	V5-> NO RESPONSE 0	ONE 1	TWO 2	THREE 3	MORE THAN 3 4	Row Total
0 NEGATIVE	3	181	54	10	20	268
	1.1	67.5	20.1	3.7	7.5	97.8
	60.0	98.9	98.2	90.9	100.0	
	1.1	66.1	19.7	3.6	7.3	
1 POSITIVE	2	2	1	1		6
	33.3	33.3	16.7	16.7		2.2
	40.0	1.1	1.8	9.1		
	.7	.7	.4	.4		
Column Total	5	183	55	11	20	274
	1.8	66.8	20.1	4.0	7.3	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
37.33079	4	.0000	.109	6 OF 10 (60.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V6 EDUCATIONAL STATUS

V18	V6->	ILLITERATE 0	PRIMARY 1	SECONDARY 2	TERTIARY 3	Row Total
0 NEGATIVE	Count	33	50	138	47	268
	Row Pct	12.3	18.7	51.5	17.5	97.8
	Col Pct	97.1	100.0	97.2	97.9	
	Tot Pct	12.0	18.2	50.4	17.2	
1 POSITIVE	Count	1		4	1	6
	Row Pct	16.7		66.7	16.7	2.2
	Col Pct	2.9		2.8	2.1	
	Tot Pct	.4		1.5	.4	
Column Total		34	50	142	48	274
		12.4	18.2	51.8	17.5	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.47231	3	.6887	.745	4 OF 8 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V7 OCCUPATION

V18	V7->	NO RESPONSE 0	CIVIL SERVANT 1	BUSINESS 2	UNSKILLED 3	UNEMPLLOYED 4	Row Total
0 NEGATIVE	Count	11	30	83	38	106	268
	Row Pct	4.1	11.2	31.0	14.2	39.6	97.8
	Col Pct	100.0	100.0	98.8	95.0	97.2	
	Tot Pct	4.0	10.9	30.3	13.9	38.7	
1 POSITIVE	Count			1	2	3	6
	Row Pct			16.7	33.3	50.0	2.2
	Col Pct			1.2	5.0	2.8	
	Tot Pct			.4	.7	1.1	
Column Total		11	30	84	40	109	274
		4.0	10.9	30.7	14.6	39.8	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
2.94546	4	.5670	.241	5 OF 10 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V8 PREVIOUS BLOOD TRANSFUSION

V18	V8->	NO (0)	YES (1)	Row Total
NEGATIVE (0)	Count	240	28	268
	Row Pct	89.6	10.4	97.8
	Col Pct	98.0	96.6	
	Tot Pct	87.6	10.2	
POSITIVE (1)	Count	5	1	6
	Row Pct	83.3	16.7	2.2
	Col Pct	2.0	3.4	
	Tot Pct	1.8	.4	
Column Total		245	29	274
		89.4	10.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.00000	1	1.0000	.635	1 of 4 (25.0%)
.23983	1	.6243	(Before Yates Correction)	

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V9 ALCOHOLIC/SMOKING

V18	V9->	NO (0)	YES (1)	Row Total
NEGATIVE (0)	Count	251	17	268
	Row Pct	93.7	6.3	97.8
	Col Pct	97.7	100.0	
	Tot Pct	91.6	6.2	
POSITIVE (1)	Count	6		6
	Row Pct	100.0		2.2
	Col Pct	2.3		
	Tot Pct	2.2		
Column Total		257	17	274
		93.8	6.2	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.00000	1	1.0000	.372	1 of 4 (25.0%)
.40577	1	.5241	(Before Yates Correction)	

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V10 USE OF CONDOME

V18 / V10->	Count / Row Pct / Col Pct / Tot Pct	NO RESPO NSE 0	YES 1	NO 2	Row Total
0 NEGATIVE		5	28	235	268
		1.9	10.4	87.7	97.8
		100.0	96.6	97.9	
		1.8	10.2	85.8	
1 POSITIVE			1	5	6
			16.7	83.3	2.2
			3.4	2.1	
			.4	1.8	
Column Total		5	29	240	274
		1.8	10.6	87.6	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.33908	2	.8441	.109	3 OF 6 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V11 USE TRADITIONAL BARBERS

V18 / V11->	Count / Row Pct / Col Pct / Tot Pct	NO RESPO NSE 0	YES 1	NO 2	Row Total
0 NEGATIVE		1	32	235	268
		.4	11.9	87.7	97.8
		100.0	100.0	97.5	
		.4	11.7	85.8	
1 POSITIVE				6	6
				100.0	2.2
				2.5	
				2.2	
Column Total		1	32	241	274
		.4	11.7	88.0	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.83997	2	.6571	.022	3 OF 6 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V12 SHARE CLIPPERS

V18	V12->	NO RESPO NSE 0	YES 1	NO 2	Row Total
NEGATIVE	0	6 2.2 100.0 2.2	49 18.3 100.0 17.9	213 79.5 97.3 77.7	268 97.8
POSITIVE	1			6 100.0 2.7 2.2	6 2.2
	Column Total	6 2.2	49 17.9	219 79.9	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
1.54058	2	.4629	.131	3 OF 6 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V13 MANICURE/PENDICURE

V18	V13->	NO RESPO NSE 0	YES 1	NO 2	Row Total
NEGATIVE	0	1 .4 100.0 .4	17 6.3 100.0 6.2	250 93.3 97.7 91.2	268 97.8
POSITIVE	1			6 100.0 2.3 2.2	6 2.2
	Column Total	1 .4	17 6.2	256 93.4	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.43132	2	.8060	.022	3 OF 6 (50.0%)

Number of Missing Observations = 0

Crosstabulation: V18 HIGH LEVEL
By V14 ETHNIC GROUP

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V18	V14->	NO RESPONSE 0	IGBO 1	HAUSA/FULANI 2	YORUBA 3	OTHER NORTH 4	Row Total
0 NEGATIVE	Count	2	16	100	34	106	268
	Row Pct	.7	6.0	37.3	12.7	39.6	97.8
	Col Pct	66.7	100.0	97.1	100.0	98.1	
	Tot Pct	.7	5.8	36.5	12.4	38.7	
1 POSITIVE	Count	1		3		2	6
	Row Pct	16.7		50.0		33.3	2.2
	Col Pct	33.3		2.9		1.9	
	Tot Pct	.4		1.1		.7	
(Continued)	Column Total	3 1.1	16 5.8	103 37.6	34 12.4	108 39.4	274 100.0

Crosstabulation: V18 HIGH LEVEL
By V14 ETHNIC GROUP

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V18	V14->	OTHER SOUTHS 5	Row Total
0 NEGATIVE	Count	10	268
	Row Pct	3.7	97.8
	Col Pct	100.0	
	Tot Pct	3.6	
1 POSITIVE	Count		6
	Row Pct		2.2
	Col Pct		
	Tot Pct		
	Column Total	10 3.6	274 100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
15.23754	5	.0094	.066	7 OF 12 (58.3%)

Number of Missing Observations = 9

Crosstabulation: V18 HIGH LEVEL
By V15 HAVE YELLOW EYES

V18	Count / Row Pct / Col Pct / Tot Pct	NO RESPONSE 0	YES 1	NO 2	Row Total
NEGATIVE	0	1	27	240	268
		.4	10.1	89.6	97.8
		100.0	100.0	97.6	
		.4	9.9	87.6	
POSITIVE	1			6	6
				100.0	2.2
				2.4	
				2.2	
	Column Total	1	27	246	274
		.4	9.9	89.8	100.0

Chi-Square	D.F.	Significance	Min E.F.	Cells with E.F.< 5
.69822	2	.7053	.022	3 OF 6 (50.0%)

Number of Missing Observations = 0

This procedure was completed at 10:28:07
FINISH.

End of Include file.