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Overview of Hepatitis B virus (HBV) Infection

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Abstract. hepatitis B is a virus that can harm the liver in both the short and long term. The virus can also be transmitted through unsafe injections, unsafe sex, or accidental skin puncture with a sharp object, among other means. The virus is most commonly passed from mother to child during labor and delivery. According to the World Health Organization (WHO), 296 million people will have chronic hepatitis B infection in 2019, with 1.5 million new cases each year. The vast majority of the estimated 820,000 hepatitis B-related deaths in 2019 are due to cirrhosis and hepatocellular carcinoma. (Primary liver cancer). Hepatitis B can be avoided with the help of safe and effective vaccines. The hepatitis B virus causes a liver infection that, if left untreated, can be fatal (HBV). The entire world's health is jeopardized. It can result in a long-term infection and an increased risk of dying from cirrhosis or liver cancer. You can get a vaccine that protects against hepatitis B and has a high success rate (98% to 100%). A major goal is to prevent long-term health problems and liver cancer, both of which can result from hepatitis B infection. According to the World Health Organization, there are 116 million and 81 million infected people worldwide. According to statistics, chronic hepatitis B infection is most common in the Western Pacific and African regions. The WHO Eastern Mediterranean Region has 60 million cases, South-East Asia has 18 million, Europe has 14 million, and the Americas have 5 million.
Keywords: Hepatitis B, virus, HBV, Infection, **Treatment,** DNA, gene

I. Introduction

Introduction

The term "hepatitis" refers to liver inflammation. The liver is a vital organ that detoxifies the blood, processes nutrients, and protects the body from infections. When the liver is inflamed or damaged, its function can be compromised. Hepatitis can be caused by excessive alcohol consumption, toxins, certain medications, and medical conditions. Hepatitis viruses are a major public health concern on a global scale. The five viruses most commonly responsible for infecting the liver and causing hepatitis are known as Hepatitis A, B, C, D, and E. Infections with the hepatitis B, C, or D virus (HBV, HCV, or HDV) can result in chronic liver diseases and its long-term complications, such as cirrhosis and hepatocellular carcinoma (HCC). Hepatitis A virus (HAV), hepatitis B virus (HBV), and hepatitis C virus (HCV) are the three most common types of viral hepatitis found worldwide. In Saudi Arabia, this disease poses a significant threat to the general population's health. According to data from the Saudi Ministry of Health (MOH), viral hepatitis was the second most common viral disease reported in 2007, trailing only chickenpox. In that year, nearly 9000 new cases of viral hepatitis were diagnosed, with HBV accounting for 52%, HCV accounting for 32%, and HAV accounting for 16%, [1]. In Saudi Arabia, the human papillomavirus (HPV) and the human hepatitis C virus (HCV) are major causes of hepatocellular carcinoma and diseases requiring liver transplantation. As a result, there is a high demand for medical resources [2]. Significant shifts in the epidemiology of viral hepatitis have occurred

in Saudi Arabia over the last two to three decades, coinciding with significant socioeconomic developments in the country. Saudi Arabia has been identified as a region with a high prevalence of HBV infection since the 1980s **[3].**

HBV, or hepatitis B virus, is a DNA virus that almost always replicates in the liver. HBV infection remains a major global health issue, with acute and chronic HBV infection causing approximately 1 million deaths each year. Despite the availability of effective recombinant vaccines, HBV infection continues to be a major global health issue, [4]. The hepatitis B virus is a worldwide public health concern. The World Health Organization (WHO) estimates that approximately 350 million people have chronic hepatitis B virus infection (HBV). This infection's prevalence varies greatly by region, with Western Europe, North America, New Zealand, Australia, and Japan having the lowest rates (2%), and Africa, Southeast Asia, and China has the highest rates (>8%). The condition affects approximately 8% of the Brazilian population. There are currently eight genotypic variants ranging from A to H, as well as four major surface antigen subtypes denoted by the letters ADW, law, ADR, and ayr. There has been a lot of interest in determining the geographical distribution and prognosis associated with the various genotypes and subtypes.

The prevalence of HBV infection varies greatly around the world, with rates ranging from 0.1 percent to 20% in various regions **[5].** The Amazon basin, parts of the Middle East, the Far East, and Sub-Saharan Africa are all examples of regions with "high" prevalence, defined as an HBsAg positivity rate of more than 8%. These are all areas where the viral infection is common. The overwhelming majority of people in these areas have anti-hepatitis B core antigen (anti-HBc) or anti-hepatitis B surface antigen (anti-HBs) positivity, which is a serologic indicator of a previous HBV infection, **[6].**

Although the infection is usually asymptomatic in early childhood, it frequently results in a chronic carrier state for the rest of the child's life. More than 2 million people who are still alive today are thought to have had HBV infection at some point in their lives. Because they will continue to be infected, approximately 350 million of these people will carry the virus for the rest of their lives, **[7].** The vast majority of the world's population, roughly 75%, lives in areas with high rates of infectious disease. Each year, there are over 4 million acute clinical cases of HBV, and approximately one-quarter of HBV carriers, or one million people, die from chronic active hepatitis, cirrhosis, or primary liver cancer.

The detection of serological markers is the primary method for establishing a diagnosis of HBV infection, and the most accurate marker of HBV carriage is the HBV surface antigen (HBsAg) in serum. Most of the time, the HBV e antigen, also known as HBeAg, is used as a secondary marker to indicate high levels of the virus in the blood. Chronic HBV carriers who can be tested for HBeAg have a significantly increased risk of developing advanced liver disease and eventually dying from liver failure, **[8].** Monitoring the presence of hepatitis B virus DNA in the serum is just as important as using serological markers in predicting the clinical outcome of an infection. In recent years, the use of molecular diagnostic methods has enabled the quantification of HBV DNA levels in serum as a marker of viral replicate activity.

The use of PCR-based methods has resulted in a significant increase in the sensitivity of HBV DNA detection, and the widespread adoption of PCR-based methods (such as those provided by HBV Monitor and Roche Diagnostic Systems), which has resulted in the commercialization of PCR-based methods, has resulted in widespread adoption of the

methodology. Recently, the development of real-time PCR methodology has increased the ease with which HBV DNA levels can be monitored and the range over which such levels can be accurately quantified. This is because real-time PCR has expanded the range over which such levels can be accurately quantified, **[9].** HBV, or hepatitis B virus, is a DNA virus that almost always replicates in the liver. Despite the availability of highly effective recombinant vaccines, HBV infection continues to be a major health concern for people all over the world. Each year, acute and chronic HBV infections kill approximately one million people **[4, 10, 11].** This work aims to overview Habtaites B virus (HBV) Infection.

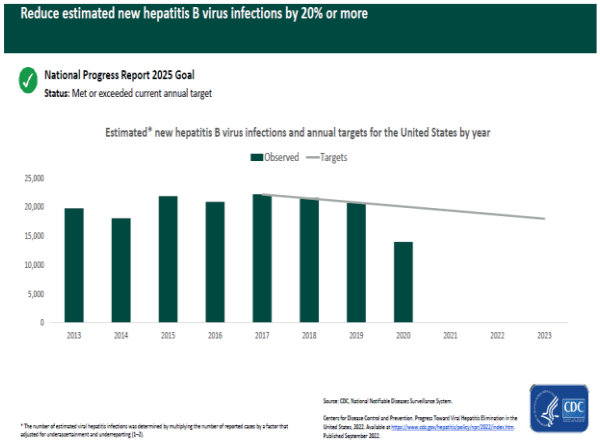

Fig1. Viral Hepatitis National Progress Report (2022)**, [12].**

Reduce estimated new hepatitis B virus infections by 20% or more

National Progress Report 2025 Goal

Status: Met or exceeded current annual target

Estimated* new hepatitis B virus infections and annual targets for the United States by year

	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023
Observed	19,800	18,100	21,900	20,900	22,200	21,600	20,700	14,000			
Targets					22,200	21,500	20,800	20,100	19,400	18,700	18,000

Table, 1. Viral Hepatitis National Progress Report (2022), [12].

Hepatitis B Can Exist in Several Forms

The Hepatitis B virus (HBV) is regarded as the model virus for the Hepadnaviridae family and the Orthohepadnavirus genus. It is an agent that is preferentially active in the liver and was originally known as serum hepatitis. The term "hepatology" refers to how the virus enters and, in some cases, remains dormant within the hepatocytes. The result is a type of necroinflammatory hepatitis that can vary in duration and severity, **[13].** The woodchuck hepatitis virus is a virus in the Hepadnaviridae family that is related to HBV. It was discovered in eastern woodchucks. A hepatitis-causing ground squirrel virus has been discovered in Beechy ground squirrels. Tree squirrel hepatitis and duck hepatitis B virus (DHBV) were isolated using Pekin ducks. They all share genomic and structural characteristics, such as virion size and ultrastructure, which consists of an envelope encasing a spherical nucleocapsid containing a viral DNA genome that is comparable in terms of organization, size, and structure. HBV has unique characteristics, many of which are shared by retroviruses. It replicates using an RNA intermediate and can integrate into the host genome. Its one-of-a-kind replication process involves the reverse transcription of an RNA strand that is longer than the genome, using a protein primer encoded by the virus. Finally, hepadnaviruses have a moderately limited host range, typically being restricted to each species alone**, [14].**

Hepatitis B is being transmitted all over the world.

On a global scale, the prevalence of hepatitis B can be divided into three categories, each determined by the proportion of a given population infected with the disease. In Southeast Asia, China, the Philippines, the Middle East, Africa, the Amazon Basin, the Pacific Islands, and the Arctic, the percentage of people who have ever been infected with the disease exceeds 60%. A chronic infection

affects between 8% and 15% of the population in these areas. Twenty to sixty percent of people in Eastern and Southern Europe, Central Asia, Japan, Israel, and South America are infected, and two to seven percent of those infected are chronically ill. In most Western European countries, North America, Australia, and New Zealand, the infection rate is less than 20%. Chronic infection affects less than 2% of the total population in these regions, as illustrated in Figure 2. **[15].**

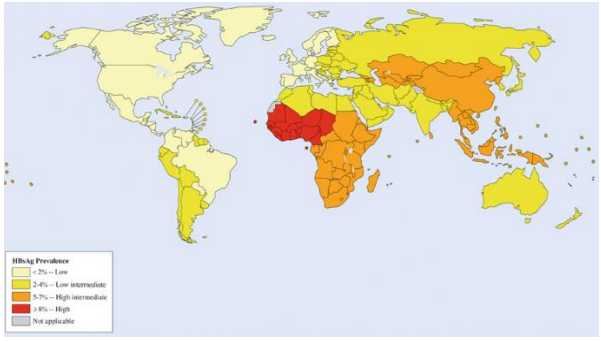

Figure 2 Global Hepatitis B Prevalence 2005 (adults 19–45 years old), [15].

The societal prevalence of HBV

Because more than 2 billion people worldwide are currently infected with hepatitis B, this disease has a significant impact on public health **[16].** It is responsible for approximately 40% of all hepatitis cases in the United States **[17].** Hepatitis B cases reported to the CDC in the United States can spread through a variety of routes, including heterosexual activity (36% of cases), intravenous drug use (13% of cases), homosexual activity (11% of cases), household contact (3% of cases), healthcare employment (2% of cases), and unknown causes (33% of cases), [18]. The actual percentages of each group vary from study to study, but the percentages presented here illustrate the broad patterns. High-risk groups include healthcare workers, children born to Hepatitis B-infected mothers, intravenous drug users, homosexual men, institutionalized populations, hemodialysis patients, recipients of certain plasma-derived products, household contacts and sexual partners of infected people, and people living in endemic areas. People aged 15 to 44 account for 89% of all HBV cases reported in the United States, **[19].** Even though vertical transmission is common, **[17].** Because the Hepatitis B virus kills an estimated 300 healthcare workers each year, it is an important virus for those who work in the medical field, **[17].** Blood and its derivatives are the most common mode of horizontal transmission. The risk of contracting hepatitis B from a blood transfusion is now extremely low (0.002%) thanks to comprehensive serological screening procedures introduced in 1972., **[19].**

Infections can be contracted by coming into contact with an infectious fluid through a minor, even inconspicuous, break in the skin. This can result in the spread of infection (Turgeon, 1996). An analysis revealed that disease transmission occurred in 6-24% of the cases where healthcare workers were exposed to hepatitis B via contaminated needle sticks. In order to cause infection, 1 x 10-6 ml of infected serum is required. Other bodily fluids that can contain infectious levels of the virus besides feces, saliva, urine, and serous fluids include vaginal secretions, sperm, and genital fluids, **[17].**

The hepatitis B virus is resistant to many of the commonly used laboratory treatments. These treatments include repeated freezing and thawing, a sixty-minute incubation at 37 degrees Celsius, desiccation, and ultraviolet light treatment. When the virus is in a solution with a high protein concentration, such as undiluted serum, it is stable to a 1:10 dilution of household bleach, which is commonly used for benchtop disinfection. This is significant because it indicates that the virus can resist being killed by bleach. Undiluted bleach from a household supply store can be used to eliminate the infectious potential of serum

spills contaminated with the Hepatitis B virus, **[20].**

The organizational structure of the HBV genome

The HBV genome is made up of approximately 3200 base pairs and is a type of DNA known as covalently closed circular DNA (cccDNA). It is a virus with an envelope and a genome that is uniquely organized in a partially double-stranded pattern. The genome encodes open reading frames (ORFs) that overlap and cover the entire genome. The genome contains four overlapping ORFs. ORFs are made up of the following regions: core (C) [pre-core/core], surface (S) [(pre-S1/S2/S)], polymerase (P), and HBX-encoding (X) **[21].** The minus strand is covalently attached to the viral polymerase at its 5' end and is approximately 3200 bases long. The minus strand is the longer of the two and contains the open reading frames (ORFs) that encode for viral proteins as well as the cis-elements that regulate HBV gene expression and replication. A plus strand of varying length maintains the cohesive hybridization's circular structure, which straddles the 5' and 3' ends of the minus strands. This strand is in charge of keeping the hybridization going. The four regions responsible for protein coding are depicted between the inner and outer circles. The pre-core/core gene, the polymerase gene, and the X gene are examples of these regions. The polymerase open reading frame (ORF) and the envelope genes pre-S1 (Large), pre-S2 (Middle), and small surface overlap (S or HBsAg). The locations of the two enhancers, ENI and ENII, as well as the two direct repeats, DR1 and DR2.

The HBV genes

Surface-related gene

The S ORF, which is structurally and functionally subdivided into pre-S1, pre-S2, and S regions, encodes the HBsAg proteins found on the surface of the viral envelope. Even though each has a distinct translation start codon (ATG), they all end with the same translation stop codon (TAA). The three HBsAg proteins that are produced are known as HBsAg, MHBsAg, and HBsAg, in that order. Each of these proteins is required for the formation of the HBV envelope, **[21].** The S gene, which codes for HBsAg, is considered the most important gene because it contains the epitope required for neutralization. It consists of 226 different amino acids. HBsAg is extremely important because it contains a major epitope capable of neutralizing the virus and is thus included in all commercially available hepatitis B vaccines. Only the presence of antibodies to HBsAg, also known as anti-HBs, can protect against HBV infection, **[22].**

The HBx gene is responsible for this.

The HBV genome contains four ORFs, the smallest of which is the X gene. It contains the genetic code for a polypeptide of 154 amino acids with a molecular weight of 17 kD. (1993, Lau and Wright). The X gene is thought to be an early gene, but it was the very last of the HBV genes to be discovered. Inadequate research has been conducted into the potential biological roles of HBxAg. In addition to its primary function of activating transcription, the X protein has been shown to transactivate a variety of cellular and viral promoters, [23].

HBV virus genotypes and subtypes

To classify HBV into its eight genotypes, which are denoted by the letters A through H, an intergroup divergence of 8% or more in the nucleotide sequences were used. Genotype A has been linked to Europe and Sub-Saharan Africa, genotypes B and C to East Asia, genotype D to the Mediterranean and Middle Eastern regions, genotype E to Western Africa, and genotype F to Americans. Individuals with the genotype G have been reported in both France and the United States, [24]. Previous research suggested that genotype A was the most common. Genotype C is the most common in South Africa, while genotype E is the most common in West Africa, **[25].**

Migration that occurs over time and across multiple regions can be used to explain the genotype distribution across those regions. They may also reveal the immigrants' countries of origin as well as other migration patterns. South Africa is an excellent example of this pattern because it has a diverse range of genotypes, including genotypes A and D. These genotypes are associated with migration from Northern Europe (the United Kingdom and the Netherlands), Southern Europe, and India. It has been established that recombination between genotypes can occur in South Africa, **[26].** It is unknown how these recombination events occurred in the HBV virus. Recent research has resulted in the identification of genotype H. **[27].**

HBV has been classified into nine distinct serological types in addition to its genotypes. This theory is based on the use of sub-specific antibodies against HBsAg, which reflect the genetic variability of HBV. One of the defined determinants is known as the "a determinant" because it is shared by all subtypes. There are also two pairs of common sub-determinants of the major envelope protein. They have been widely used in clinical, virological, and epidemiological research to distinguish between HBV strains. These serotypes can be found in a wide range of geographical areas. For example, ayw2 is prevalent in the Mediterranean region, while adw2 is found in the Pacific region. and can only be found in the Eastern region. The most common form of the disease in Northern Europe and Sub-Saharan Africa is serotype adw2. However, as one moves westward from East Africa into Central and West Africa, the prevalence of this serotype decreases, accompanied by an increase in the unique African serotype ayw4, **[28].**

The life cycle of the HBV virus

Coating access and removal

The HBV lifecycle is based on the model developed for the overall replicative cycle of hepadnavirus using DHBV. The attachment of mature viruses to host cell membranes initiates the HBV infection process. This process most likely involves the surface protein's pre-S domain On the other hand, the early stages of the viral life cycle, such as entry, uncoating, and viral genome delivery into the nucleus, are poorly understood. This is due in part to the lack of available cell lines susceptible to HBV infection. As a result of research aimed at determining the HBV receptor, numerous candidates, such as apolipoprotein H, have been discovered, **[29].** Despite these findings, the proteins and mechanisms used by HBV to enter cells have yet to be fully characterized. The DHBV system has demonstrated that the uncoating of the HBV genome occurs at the nuclear membrane, **[30].**

The replication of the HBV virus's genome

After HBV has entered the hepatocytes, the viral nucleocapsid releases circular DNA that is partially double-stranded and covalently closed. This DNA is then converted within the hepatocyte nucleus to covalently closed circular DNA. The cccDNA, which serves as a template for the transcription process, is used to generate functional genomic and subgenomic mRNAs. Surface proteins and the HBX protein are regulated by smaller subgenomic transcripts, which range in size from 2.4 to 2.1 to 0.9 kilobase pairs and function as messenger RNA. The longer genomic transcript is 3.5 kilobytes long, which is longer than one genome. This genomic transcript functions as messenger RNA (mRNA) in the synthesis of HBeAg, core, and polymerase proteins (Figure 3).

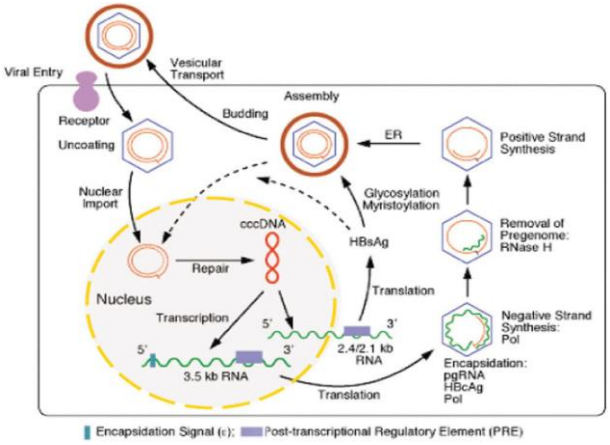

Figure 3. depicts a schematic representation of the HBV life cycle, **[31].**

The genomic transcripts are 3.5 kb long and made up of two species with distinct 5' ends. This is the case for genomic and pre-core RNAs. Pregenomic RNA serves as a template for HBV reverse transcription as well as the messenger RNA for core and polymerase (pgRNA). The precore direct RNA directs the translation of the pre-core gene product. The viral messenger RNAs are transported to the cytoplasm, which is where viral protein translation, nucleocapsid assembly, and virus replication occur. The replication process occurs within a nucleocapsid composed of the core protein, genomic RNA, and ribosome. along with the polymerase, **[32].** Encapsidation of pgRNA is thought to occur as a result of the polymerase protein's interaction with the stem loop**, [33].** A terminal repeat is incorporated into both the 5' and 3' ends of this longer-than-genome-length transcript. This repeat includes DR1 as well as the stem-loop RNA packaging signal. The polymerase interacts with the 5'-end of the packaging signal to prime reverse transcription. As soon as the polymerase binds to the stem loop and begins to transcribe the pgRNA template, reverse transcription of three to four bases can begin. The polymerase protein forms a chemical bond with the growing strand of DNA in the opposite direction. The polymerase acts as a primer at the start of the reverse transcription process. The polymerase protein initiates reverse transcription by following a bulge in the stem loop that serves as its template **[34].** When the complex of pgRNA and HBV polymerase combines with the core proteins, viral capsids are formed. Following the completion of the protein priming step, the subsequent steps of HBV reverse transcription are carried out within the viral capsid. This sequence of events, which includes template switches, DNA synthesis, and RNase degradation, culminates in the formation of the recognizable circular, partially incomplete strand of DNA.

DNA from a double-stranded hepadnavirus

The virus is not eliminated in chronic hepatitis patients because they do not produce an adequate protective immune response to it during the disease. There is a chance that the liver pathology will worsen, resulting in an increase in liver necrosis, a breakdown of the reticular framework, and fibrosis **[35].** Ground glass hepatocytes are hepatocytes that have an abnormal appearance due to the presence of cytoplasmic HbsAg. Post-necrotic liver necrosis is the medical term for this condition. Chronic infection is thought to be cured when a patient with chronic hepatitis becomes HBsAg and HBeAg negative. However, only a small percentage of patients experience this. This percentage can be increased by using interferon therapy, which will be discussed in greater detail in the following section. There is a link between chronic hepatitis and the development of hepatocellular carcinoma (HCC). It is currently unknown if the hepatitis B virus contains an oncogene. Because liver necrosis is followed by intense hepatocyte proliferation during the healing process, the link between hepatitis B and hepatocellular carcinoma may be indirect. This rapid proliferation could be linked to abnormal cell development **[35].** Hepatocellular carcinoma is the most common type of cancer and the

leading cause of death in Hepatitis B-endemic areas. People who were infected through the virus's transmission from mothers to their newborn children are most likely to develop hepatocellular carcinoma.

Hepatitis D, also known as the delta agent, is a virus with a genetic flaw that prevents it from infecting on its own and only occurs in the presence of hepatitis B. It is increasingly linked to hepatitis B cases that progress to fulminant or chronic disease **[36].** The results of the laboratory tests show an increase in liver enzymes as well as several serologic markers. These serologic markers are linked to each manifestation and will be discussed further below.

The acute infection

Acute HBV infection can result in a variety of outcomes, which can include either

Which is it: symptomatic or non-symptomatic? The incubation period can range from 6 weeks to 6 months, and the onset of clinical manifestations is highly dependent on the age of the patient. HBV infection causes a well-defined immunological response, which eventually leads to infection elimination and the development of protective immunity (also known as anti-HBs). The first serologic marker of HBV infection to appear after an infection caused by HBV is HBsAg. In most cases, the HBsAg will remain in the serum throughout the clinical illness. This antigen is commonly used in the diagnosis of acute HBV infection. The absence of HBsAg and the development of anti-HBsAg (anti-HBs) antibodies indicate the resolution of acute infection. In most cases, the appearance of anti-HBc coincides with the appearance of HBsAg. HBc IgM appears first but is eventually replaced by HBc IgG after some time.

In most cases, newborns show no clinical signs or symptoms, and only about 5-15 percent of children aged 1 to 5 years old develop a typical illness when infected, **[37].** Symptoms in symptomatic infections can range in severity from mild to severe, depending on the type of infection present. In older children and adults, symptomatic infections are more common.

Some of the clinical signs and symptoms of acute HBV infection include fever, anorexia, nausea, malaise, vomiting, jaundice, dark urine, clay-colored or pale stools, abdominal pain, and, in rare cases, arthralgias and arthritis. About one to two percent of people with acute hepatitis develop fulminant hepatitis, which has a case-fatality ratio ranging from 63 to 93%, **[38].**

Pathogenesis

It is well understood that the outcome of an HBV infection is determined by the dynamic interaction and equilibrium of the rate of viral replication and the host's immune response. The cellular and humoral immune responses to HBV infection are both complexes. Virus elimination necessitates contributions from both the humoral and cellular immune systems. Antibodies directed against each of the individual viral antigens, known as anti-HBs, anti-HBc, and anti-HBe, are a feature of hepatitis B virus (HBV) infection. The HBV infection is cleared as a result of a sufficient multispecific anti-HBV T cell response against HBV proteins. The persistence of HBV infection, on the other hand, is the result of an insufficient immune response. A robust polyclonal helper (Th) and cytotoxic T lymphocyte (CTL) response to multiple viral antigens in infected livers is linked to acute infection resolution, **[14].** Noncytolytical reduction of viral gene products in infected cells by cytokines such as interferon-gamma and tumor necrosis factor released by activated T lymphocytes has been shown to play an important role in infection termination. Even though the destruction of virally infected hepatocytes is visible during the resolution of acute infection, this is the case**.** HBV does not directly cause cytopathy in infected hepatocytes, according to the findings of the vast majority of studies, and the cellular

response to several viral proteins appears to correlate with the severity of clinical disease and viral clearance, **[39].** It is hypothesized that the production of antibodies in response to viral envelope antigens aids in viral elimination and that cytotoxic T cells kill infected cells to aid in viral elimination. It has also been shown that cytotoxic T lymphocytes can inhibit HBV gene expression by secreting antiviral cytokines and that the expression of these cytokines may be the primary factor in HBV gene expression inhibition. The mechanism of virus clearance during an HBV infection. Chronic infection is thought to be associated with a poor T-cell response to viral antigens, whereas neonatal immune tolerance to viral antigens appears to play an important role in viral persistence in people who were infected at birth. Both of these factors are thought to be at work. There is a lack of knowledge about the causes of poor T-cell response in adults, **[38, 10, 11].**

HBV; Transmisson

HBV is transmitted through the mucosal or percutaneous surfaces of the body being exposed to infected blood or other body fluids. HBV is transmitted via a wide range of human contact, including perinatal or mother-to-child contact, nonsexual household contact, sexual contact, needle sharing, occupational contact, and healthcare-related contact. Blood and serum have the highest levels of infectious HBV found anywhere in the body. However, the disease can be transmitted not only through serum but also through other body fluids derived from serum, such as sperm and saliva. Those who are infected with HBV are the primary reservoirs for transmission; however, anyone who tests positive for HBsAg has the potential to infect others through sexual or intimate contact, as well as those in their household. Because the virus can remain infectious and stable on environmental surfaces for at least seven days, HBV transmission can occur indirectly through contaminated surfaces and other objects.

Transmission of HBV from a chronically infected mother to her child during childbirth is both effective and one of the most common routes of HBV infection found worldwide. HBV transmission in utero is possible, but it is extremely rare, accounting for less than 2% of all perinatal transmissions. Perinatal transmission of HBV most commonly occurs during the birthing process. Babies born to mothers who tested positive for HBsAg have a 5-20% chance of perinatal infection, but the risk increases to 70-90% if the mother also tested positive for HBeAg. HBV infection can also be transmitted from person to person if there is frequent and prolonged close personal contact with an infected person. Before the implementation of universal infant hepatitis B immunization, it was estimated that approximately 16,000 children under the age of ten were infected with hepatitis B on an annual basis in the United States. This infection was caused by contact with HBsAg-positive household members or community contacts, **[40].**

Sexual contact is one of the most effective ways of transmitting HBV (Alter and Margolis, 1990). It has been demonstrated that sexual contacts of chronically infected individuals have a higher seroprevalence of HBV infection than control populations, including household (nonsexual) contacts of infected individuals. People with acute hepatitis B are more likely than controls to report having multiple heterosexual partners, and HBV seroprevalence correlates with a higher number of recent and lifetime heterosexual partners. Furthermore, MSM has consistently higher HBV seroprevalence rates than the general population, **[41].** Sharing needles, syringes, and other drug paraphernalia is common among injection drug users, putting them at high risk of contracting the herpes simplex virus (HBV). According to data

collected more than ten years ago, the majority of injection drug users in the United States and elsewhere have evidence of a previous or current HBV infection. This is true both in the US and elsewhere . However, the risk among injection drug users varies depending on the prevalence of chronic HBV infection in the community, as well as the practices used for drug sharing and preparation. In the mid-1990s, approximately 70% of drug users who used intravenous drugs in the United States were infected with HIV after 5 years of injection drug use **[42].**There have been reports of outbreaks linked to percutaneous exposures other than injection drug use. It has been demonstrated that the amount of blood and needles exposed to a healthcare worker is directly related to the risk of infection that the worker faces. The risk of contracting HBV after a needle stick varies according to the volume and viral concentration of the infectious fluid exposed. The risk of inoculation after a needlestick with HBeAg-positive blood is at least 30%, while the risk after a needlestick with HBeAg-negative blood is less than 6%, **[43, 10, 11].**

Patient-to-patient One of the most common ways for people in developing countries to become infected with HBV is through transmission. Percutaneous exposure to contaminated injection or other procedure equipment, as well as blood or mucosal exposure to HBV-infected medication, can result in HBV transmission from one patient to another. HBV can also be transmitted in the following ways: Exposure to contaminated therapeutic injection equipment is common in many developing-country settings. This is primarily due to a lack of awareness of infection control practices, a lack of resources for sterilization and the purchase of new disposable equipment, as well as economic incentives and cultural preferences favoring injection overuse. Contaminated injections were responsible for an estimated 21 million new HBV infections worldwide in 2000. This accounted for 32% of all new infections, [44]. Outbreaks involving this mode of transmission continue to be a problem in the developed world, and the majority of the time, they are caused by errors in infection control practice on the part of healthcare workers. Some of the vehicles implicated in virus transmission include multidose vials, finger-stick devices, acupuncture needles, and jet injection guns. **[45].** Contaminated environmental surfaces in healthcare settings have also served as a reservoir for HBV transmission, particularly in dialysis units (Recommendations for preventing transmission of infections among chronic hemodialysis patients, 2001). HBV transmission from a healthcare provider to a patient is extremely rare. The majority of incidents have been linked to invasive procedures performed by healthcare professionals, and the majority of these incidents occurred before the widespread use of the hepatitis B vaccine and the adoption of universal precautions as standard infection control practice, **[46].** Because of blood donor screening and the implementation of techniques that ensure viral inactivation of products made from blood, such as factor concentrates, the transmission of HBV through blood product transfusion has been significantly reduced or eliminated in the majority of regions around the world, **[47].**

Transmission during pregnancy

Despite high rates of prenatal HBsAg screening in the United States, identifying pregnant women who are chronic HBV carriers has been difficult. Although it is estimated that 23,000 HBsAg-positive women give birth each year, only 9,000 are identified and reported through prenatal screening (Euler et al., 2003). As a result of this disparity, it appears that there is a high prevalence of chronic HBV infection among the relatively small proportion of pregnant women who do not have prenatal HBsAg screening.

Furthermore, the patient's HBsAg status is frequently unknown, even when the patient is admitted to the hospital during the labor and delivery process without being screened (Thomas et al., 2004). According to data collected in 2000, only 33% of US newborns received a birth dose of the hepatitis B vaccine, which could serve as a safety net for infants whose mothers' HBsAg testing was not done or was incorrectly recorded. Even though the vaccine could provide a safety net for infants whose mothers' HBsAg testing was not performed or was recorded incorrectly**,[48].**

Local health departments are now identifying a much larger proportion (85 percent) of the expected number of perinatally exposed infants in their areas using enhanced case management systems to improve the detection and prevention of perinatal hepatitis B. Furthermore, local health departments are identifying far more perinatally exposed infants' household and sexual contacts for vaccination. In the context of addressing these issues, these encouraging results have been demonstrated, **[49].** In 2005, the Centers for Disease Control and Prevention's Advisory Committee on Immunization Practices recommended that specific policies and procedures be established in delivery hospitals to ensure the routine administration of a birth dose to all medically stable infants unless there is a physician's order to defer administration and a copy of the laboratory report. This recommendation was made to increase the number of infants who received their first dose of the hepatitis B vaccine at birth. This recommendation was made in 2005 to (A comprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States, 2005.

Molecular tests and analyses

The only marker that enables direct detection of HBV in the blood is the virus's genetic material or DNA. There is a correlation between the presence of HBV DNA in serum and the presence of an infectious virus. Consequently, the detection of HBV DNA in the blood is direct evidence of the presence of an infectious virus. Direct nucleic acid hybridization and nucleic acid amplification using polymerase chain reaction (PCR) or ligase chain reaction are some of the methods that can be used to detect HBV DNA. Other methods include indirect nucleic acid hybridization (LCR) (LCR). Methods that are based on direct hybridization have a detection limit of approximately 0.3 pg HBV DNA/ml, which allows for the detection of approximately 105 virion particles per ml. Despite the relatively high sensitivity of the direct hybridization methods, it is not possible to definitively rule out the presence of infectivity. In comparison to methods that rely on direct hybridization, techniques that are based on amplification, such as PCR and LCR, have the capability of detecting a very low number of DNA molecules with a sensitivity that is increased by 1,000 to 10,000 times, **[50].** PCR tests, on the other hand, frequently produce incorrect positive results. It has also been demonstrated that the branched-chain DNA assay is useful for the detection of HBV DNA, **[51].** The fact that molecular assays are only used for research and cannot be used routinely in clinical laboratories to screen for HBV infections is one of the limitations of these tests. Molecular assays are also quite expensive for clinical laboratories to use.

Other supplementary tests can be carried out, such as biochemical examinations of the functioning of the liver and histological examinations of any liver damage that may have occurred. Bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, gamma-glutamyl transpeptidase, albumin, and globulin are all examples of liver function tests. However, alkaline phosphatase (ALT) and aspartate aminotransferase (AST) are the two most important indicators of hepatocellular damage

in viral hepatitis. In chronic active HBV, ALT and AST levels may fluctuate or may remain consistently elevated. A bilirubin test, an alkaline phosphatase test, and a gamma-glutamyl transpeptidase albumin test are some examples of additional liver function tests. Globulin is an essential component in determining how effectively the liver can synthesize proteins, **[52].**

Prevention and regulating behavior

In South Africa, people are required to report cases of acute HBV when they see them. Multiple strategies are currently being utilized all over the world to reduce the number of people infected with hepatitis B, as well as to control and prevent HBV transmission. These include the testing of blood donors for HBV infection markers, the vaccination of those who are at risk, the administration of hepatitis B immunoglobulin (HBIG) (often in conjunction with HBV vaccines), universal precautions, and education of healthcare workers and high-risk groups regarding routes of transmission of the disease, [**52].** Since more than 20 years ago, a vaccine that protects against HBV that is both effective and safe has been commercially available, and many nations have included it in their Expanded Programs on Immunization (EPI) (EPI), the World Health **[53]. ,** and the **[54]** all recommended that the hepatitis B vaccine be incorporated into national immunization programs by the year 1995 in nations that have a high endemicity, and by the year 1997 in all of the other nations of the world. The primary objective of vaccination is to reduce the risk of acute symptomatic HBV infection as well as chronic HBV infection, chronic liver disease, cirrhosis, and HCC, as well as deaths caused by these complications; the ultimate goal of vaccination is to hopefully eradicate HBV in the future. In April 1995, South Africa included the hepatitis B vaccine as part of its Expanded Program on Immunization (EPI), **[55].** Over ninety percent of healthy adults younger than forty years old experience a protective antibody response after receiving the recommended series of three intramuscular doses of the hepatitis B vaccine. After the age of 40, the cumulative age-specific decline in immunogenicity drops below 90%, and by the age of 60 years, only 70% of people who are vaccinated develop protective levels of anti-HBs. Booster dose studies conducted on adults have shown that over ninety percent of vaccinees have such immune memory when challenged with the hepatitis B vaccine. This indicates that the immune system would be able to respond quickly to HBV exposure if it were to occur.

Conclusions

Infection with HBV is a major cause of morbidity and mortality on a global scale; however, tremendous progress has been made in the prevention and control of the disease over the past quarter century. The foundation is being laid for significant future burden reductions in hepatocellular carcinoma and cirrhosis as a result of increasing proportions of the global birth cohort being vaccinated each year. Not only will the currently high levels of infant vaccination coverage need to be maintained or even increased to eliminate HBV transmission in the United States, but specific efforts will also need to be made to vaccinate populations that are at high risk for transmission. This is required to eliminate HBV transmission. New cases of hepatitis B virus infection are becoming increasingly concentrated in certain populations in the United States. These populations include injection drug users, inmates, and people who are at risk for sexually transmitted diseases. These populations frequently have restricted access to the kinds of settings in which preventive care and immunization services are routinely offered. It will be necessary to expand programs such as perinatal case management and venue-based vaccination of high-risk adults, and a consistent commitment

of resources will be required to support this expansion. To ensure the continued success of hepatitis B vaccination, it is necessary to have the capability to measure the impact of vaccination and investigate potential threats to the program's ability to continue to be successful. Accurate surveillance data are necessary to characterize the population that is at risk and evaluates the effects of vaccination; this is a task that becomes increasingly difficult as the overall morbidity rate decreases. In the future, it will be necessary to evaluate potential new causes of vaccine failure, such as HBV variants. Additionally, the requirement for booster doses to maintain vaccine-induced immunity should be evaluated regularly as vaccinated cohorts get older.

In May 2016, the World Health Assembly adopted the first *Global health sector strategy on viral hepatitis,* 2016–2020. The strategy highlighted the critical role of universal health coverage and sets targets that align with those of the Sustainable Development Goals. The strategy proposed the elimination of viral hepatitis as a public health threat by 2030 (defined as a 90% reduction in new chronic infections and a 65% reduction in mortality, compared with the 2015 baseline), and included a roadmap towards elimination by implementing key prevention, diagnosis, treatment, and community interventions strategies. In May 2022 the 75th World Health Assembly noted a new set of integrated global health sector strategies on HIV, viral hepatitis, and sexually transmitted infections for the period of 2022–2030. Based on these previous and now new strategies, a broad range of Member States have developed comprehensive national hepatitis programs and elimination strategies guided by the global health sector strategy.

To support countries in achieving the global hepatitis elimination targets under the Sustainable Development Agenda 2030, WHO is working to: raise awareness, promote partnerships and mobilize resources. formulate evidence-based policy and data for action. increase health equities within the hepatitis response. prevent transmission. scale up screening, care, and treatment services. WHO organizes the annual World Hepatitis Day campaign (as 1 of its 9 flagship annual health campaigns) to increase awareness and understanding of viral hepatitis. For World Hepatitis Day 2022, the WHO focuses on the theme “Bringing hepatitis care closer to you” and calls for simplified service delivery of viral hepatitis services, bringing care closer to communities.

3. References

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كلية العلوم ، جامعة الملك عبد العزيز

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مستخلص. التهاب الكبد B هو فيروس يمكن أن يؤذي الكبد على المدى القصير والطويل. يمكن أن ينتقل الفيروس أيضًا من خلال الحقن غير الآمن ، أو الجنس غير الآمن ، أو ثقب الجلد العرضي بأداة حادة ، من بين وسائل أخرى. ينتقل الفيروس بشكل شائع من الأم إلى الطفل أثناء المخاض والولادة. وفقًا لمنظمة الصحة العالمية (WHO) ، اصيب ٢٩٦ مليون شخص بعدوى التهاب الكبد B المزمنة في عام ٢٠١٩ ، مع ١,٥ مليون حالة جديدة كل عام. الغالبية العظمى من الوفيات المرتبطة بالتهاب الكبد B المقدرة بـ ٨٢٠,٠٠٠ في عام ٢٠١٩ ترجع إلى تليف الكبد وسرطان الخلايا الكبدية. يمكن تجنب التهاب الكبد B بمساعدة لقاحات آمنة وفعالة. يتسبب فيروس التهاب الكبد B في حدوث عدوى في الكبد يمكن أن تكون قاتلة (HBV) إذا تُركت دون علاج. صحة العالم كله في خطر. يمكن أن يؤدي إلى عدوى طويلة الأمد وزيادة خطر الوفاة بسبب تليف الكبد أو سرطان الكبد. يمكنك الحصول على لقاح يقي من التهاب الكبد B وله نسبة نجاح عالية (٩٨٪ إلى ١٠٠٪). الهدف الرئيسي هو منع المشاكل الصحية طويلة المدى وسرطان الكبد ، وكلاهما يمكن أن ينتج عن عدوى التهاب الكبد .B وفقًا لمنظمة الصحة العالمية ، هناك ١١٦ مليونًا و ٨١ مليون مصاب في جميع أنحاء العالم. وفقًا للإحصاءات ، فإن عدوى التهاب الكبد B المزمن هي الأكثر شيوعًا في مناطق غرب المحيط الهادئ وأفريقيا. يوجد في إقليم شرق المتوسط التابع لمنظمة الصحة العالمية ٦٠ مليون حالة ، وجنوب شرق آسيا ١٨ مليون حالة ، وأوروبا ١٤ مليونًا ، والأمريكتان ٥ ملايين.

الكلمات الدالة: التهاب الكبد B ، الفيروس، الالتهاب الكبدي الوبائي، العدوى، العلاج، الحمض النووي، الجين