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# The Roles of Viral Structural Proteins gB, gC, gD, and Tegument Proteins on the Pathogenecity of Bovine Herpesvirus-1 Infections: A review

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## Abstract

Infectious bovine rhinotracheitis, which is caused by *bovine herpesvirus-1*, is one of the most common cattle diseases and is responsible for significant economic losses worldwide. The genome of the *BoHV-1* virus is a double-stranded DNA virus, and it is made up of two distinct sequences: unique long and unique short. The goal of this paper is to review the significance of viral structural proteins gB, gC, gD, and tegument proteins on the pathogenicity of *BoHV-1* infections, despite the fact that early detection of *BoHV-1* epidemic strains is essential for assessing the herd's health status, epidemiological research, vaccine development, and disease control. *BoHV-1* viral glycoproteins (gB, gC, and gD) interact with cellular membranes to allow for virus entrance and infection to begin. Membrane fusion and cell penetration are aided by the receptor-binding proteins *BoHV-1* gB and gD. Major histocompatibility complex class I and II proteins, along with the transporter associated with antigen processing, allow viruses to enter the cell. During entry into the host cells, *BoHV-1* loses the lipid envelope and sheds some of its tegument proteins. The capsid then releases the viral DNA into the nucleus through the nuclear pore complex, where the DNA replicates via a rolling circle mechanism. At the Golgi, the final maturation of the virus takes place after the addition of the lipid envelope and the glycoproteins. Furthermore, investigations that will deliver a more comprehensive understanding of the remaining viral glycoproteins and their molecular basis, which are responsible for the pathogenicity of *BoHV-1*, and the most effective preventive and control approach for the disease are recommended for the feature.

**Keywords:** BoHV-1, glycoproteins, pathogenicity, structural proteins, virulence factor

## 1. Introduction

Livestock plays a critical role in the welfare of the rural populations of the world and its economy. Different disease-related respiratory and reproductive disorders in dairy animals due to variousetiological agents have led to significant economic losses for dairy animals (Yadav *et al*., 2018). Among these agents that cause respiratory diseases; bovine respiratory disease (BRD) includes *bovine viral diarrhea virus* (BVDV), *bovine herpesvirus-1* (*BoHV-1*), *bovine parainfluenza-3 virus* (*BPI-3V*), and *bovine respiratory syncytial virus* (*BRSV)* (Guzman and Taylor, 2015; Abd El Fadeel *et al*., 2020) are those commonly listed. Out of the above-mentioned viral agents, *BoHV-1* is one of the most globally widespread respiratory and/or reproductive viral diseases of bovines (Ackermann and Engels, 2006).

The genome of the *BoHV-1* virus is about 135 kilobase pairs (kbp) long and is an enveloped double-stranded DNA virus(Engels *et al*., 1987). The viral genome is made up of two distinct sequences: unique long (UL) and unique short (US) (Schynts *et al*., 2003). Additionally, there are 73 open reading frames (ORFs) in the *BoHV-1* genome (Engels *et al*., 1986).According to Jones andChowdhury (2008), herpesviruses typically have 12 glycoproteins (gB, gC, gD, gE, gG, gH, gI, gK, gL, gM, gN, and Us9) that are encoded by the virus and are present in the virion envelope.However, according to Barber *et al*. (2017), the *BoHV-1* virion has nine glycoproteins:gB, gC, gD, gE, gG, gH, gI, gM, and gL, whereas the other two, gN and gK, were discovered in infected cells but not in the virion (Blewett and Misra, 1991).

Infection resulting in the genital forms of the disease is acquired through venereal transmission and the genital carrier state is the primary reservoir for outbreaks of genital *BoHV-1* disease. Venereal transmission may occur during natural services and/or artificial insemination. Virus shed in semen is due to viral replication in the mucosa of the prepuce, penis, and urethra rather than the gonads or accessory sex glands (Givens, 2018). Genital shedding of *BoHV-1* can be seen as early as two to seven days following infection and bulls may intermittently shed virus throughout life without showing any clinical signsof infection. Infection is not believed to negatively impact sperm motility or semen quality (Tanghe *et al*., 2005) directly but may cause changes as a result of generalized illness.Different diagnostic techniques, such as ELISA, are used to investigate virus infection in the given herds, whereas virus isolation via cell culture is the gold standard technique(Dima and Abdisa, 2022). Even though early detection of *BoHV-1* epidemic strains is crucial for evaluating the herd's health status, epidemiological research, vaccine development, and disease control, the objective of this paper is to review the importance of viral structural proteins on the pathogenicity of *BoHV-1* infections in cattle.

## 2. Literature Review

### 2.1 Description and history of the *BoHV-1*

The virus has been known to cause disease in cattle for many years, and the first report of the disease believed to be caused by *BoHV-1*was reported from the United States in the early 1950s (Schroeder and Moys, 1954). The *BoHV-1* virus is a member of the *Herpesviridae* family and the *Alphaherpesvirinae* subfamily in the genus *Varicellovirus*(Lugaj *et al*., 2020), which is an enveloped double-stranded DNA (dsDNA) virus(Engels *et al*., 1987).The total molecular size of the *BoHV-1* genome is about 135–140 kbp (Jones and Chowdhury, 2008), which is structurally made up of a 125nm-diameter icosahedral proteinaceous capsid (Sucharita *et al*., 2022). *BoHV-1* can be differentiated into three subtypes that belong to one single viral species, and they are recognized worldwide. These three subtypes are *BoHV-1.1*, *BoHV-1.2a*, and *BoHV-1.2b*. The *BoHV-1.2b* strains are less virulent than the other strains (*Gibbs, 1977*). The virus affects different organs such as the respiratory tract, ocular, reproductive, alimentary, integumentary, and central nervous systems, in addition to causing neonatal infections (Gibbs, 1981).

*Infectious pustular vulvo-vaginitis (IPV) in cows, infectious pustular balanoposthitis (IPB) in bulls, and infectious bovine rhinotracheitis (IBR), a highly contagious viral disease of cattle, are all caused by BoHV-1*(Keneisezo *et al*., 2019; Lugaj *et al*., 2020). This virus is also one of the most important viral infections of buffaloes all over the world (Woodbine *et al*., 2009; Lugaj *et al*., 2020), except in *BoHV-1*-free countries (OIE, 2010). The disease is commonly characterized by inflammation of the upper respiratory tract (Gibbs, 1981).

## 2.2 Morphology and structure of the *BoHV-1*

*Alphaherpesviruses* have their own typical and distinct virion morphology (Davison *et al*., 2009). The viral genome of *BoHV-1* consists of dsDNA that codes for about 70 proteins, including 33 structural proteins, 15 nonstructural proteins, and an envelope glycoprotein, which is located in the envelope on the surface of the virions(Gibbs, 1977). The nucleocapsid, which is made up of 150 hexagons and 12 pentagons, surrounds the linear dsDNA genome. The envelope, a lipid bilayer produced from the host cell, surrounds the tegument, a protein layer that contains the nucleocapsid (Mettenleiter *et al*., 2009).

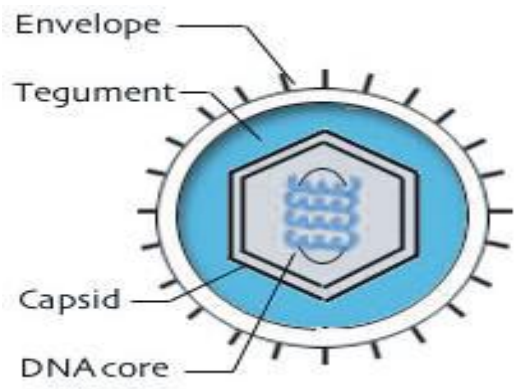


**Figure 1**: A schematic diagram of *BoHV-1* virion structure(Chatterjee *et al*., 2016)

## 2.3 The Role of Viral Structural Proteins for the Pathogenecity of *BoHV-1*

The *BoHV-1* viral genome consists of 12 enveloped glycoproteins, of which ten are glycosylated and two are non-glycosylated (Jones and Chowdhury, 2008). Following viral infections, these glycoproteins are essential for viral entry, pathogenesis, and the development of the host's immune system (Gibbs, 1977;Jones and Chowdhury, 2008). The significance of each viral glycoprotein for viral pathogenicity will be discussed below.

### 2.3.1 *BoHV-1* glycoproteins

#### 2.3.1.1 Glycoprotein (gB, gC and gD)

*BoHV-1* viral glycoproteins(gB, gC, and gD) interact with cellular membranes to allow for virus entrance and infection to begin. Membrane fusion and cell penetration are aided by the receptor-binding proteins *BoHV-1*gB and gD.Endosomal low pH and host cell endocytosis have recently been shown to be essential for *BoHV-1* entrance. Cellular membrane proteins, such as the putative gB-receptor for the *alphaherpesvirus*PILR, may also bind with *BoHV-1*gB or a gH/gL complex(Pastenkos*et al*., 2019).

#### 2.3.1.2 Tegument proteins

All *herpesviruses*' virion component consists of a bunch of proteins called a viral tegument that fills the gap between the nucleocapsid andenvelope. This protein layer is sometimes referred to as a viral matrix (Yu *et al*., 2011). The primary *BoHV-1* tegument protein, VP8 is crucial for viral replication in the host-infected cell (Sucharita *et al*., 2021).These proteins are often generated in the late stages of viral infectious cycles, following viral gene replication. Soon after infection, the tegument is typically released into the cytoplasm. The tegument normally contains proteins that support viral DNA replication and immune response avoidance, frequently via inhibiting immune system signaling and activating interferons (Brian *et al*., 2007). The tegument is a crucial layer because tegument proteins can play a variety of essential roles in the replication, virus assembly, and immune evasion phases of the *herpesvirus* life cycle (Sucharita *et al*., 2022).The most prevalent tegument protein in *BoHV-1* is

VP8, which is produced by the $UL_{47}$ gene and has a crucial role in both viral replication and the activation of the host's immune system (Lobanov*et al*., 2010). Additionally, it affects how host defense mechanisms respond and how host cells undergo apoptosis(Afroz *et al*., 2018).During early infection, nuclear localization signals (NLS)allow VP8 to enter the nucleus. As a result, VP8 recruits and redistributes promyelocytic leukemia protein into the nucleus, and host defenses against viruses are suppressed (Sucharita *et al*., 2022).

### 2.3.2 Viral Replication

Initiating with receptor-mediated endocytosis, which results in the fusion of the viral envelope and the endocytic membrane as a result of host cell molecules and glycoprotein interactions, the life cycle of *BoHV-1* begins with entrance into the host cell (Muylkens *et al*., 2007;Sucharita *et al*., 2021).The mucosal epithelia of the respiratory and genital tracts are the first to become infected by *BoHV-1*(Muylkens *et al*., 2007).During entry into the host cells, *BoHV-1* loses the lipid envelope and also sheds some of its tegument proteins. The capsid then releases the viral DNA into the nucleus through the nuclear pore complex, where the DNA replicates via a rolling circle mechanism (Sucharita *et al*., 2021).Inside the nucleus, the freshly formed viral DNA is packaged into the capsid, which is then ready to leave the nucleus through a budding process. The capsid gains a primary envelope from the inner nuclear membrane during egress and buds into the perinuclear space (Mettenleiter *et al*., 2009).The tegument then undergoes changes in composition as the virus particle moves through the cytoplasm towards its site of maturation at the Golgi, as a result of protein loss and/or addition (Granzow *et al*., 2001).

At the Golgi the final maturation of the virus takes place after the addition of the lipid envelope and the glycoproteins (Mettenleiter *et al*., 2009).Furin also cleaves the majority of herpesvirus conserved glycoprotein B (gB), which is an important component of the respective virions and required for virus entrance and direct propagation from cell to cell (Kopp *et al*., 1994; Spear and Longnecker, 2003).Tegument proteins are added to *alphaherpesviruses* during primary envelopment, which occurs when the virus moves from the nucleus into the perinuclear region for the duration of its egress (Granzowel*et al*., 2001); in the cytoplasm, following its exit from the nucleus and movement towards the Golgi; and at the Golgi, where the final maturation occurs (Mettenleiter *et al*., 2009).

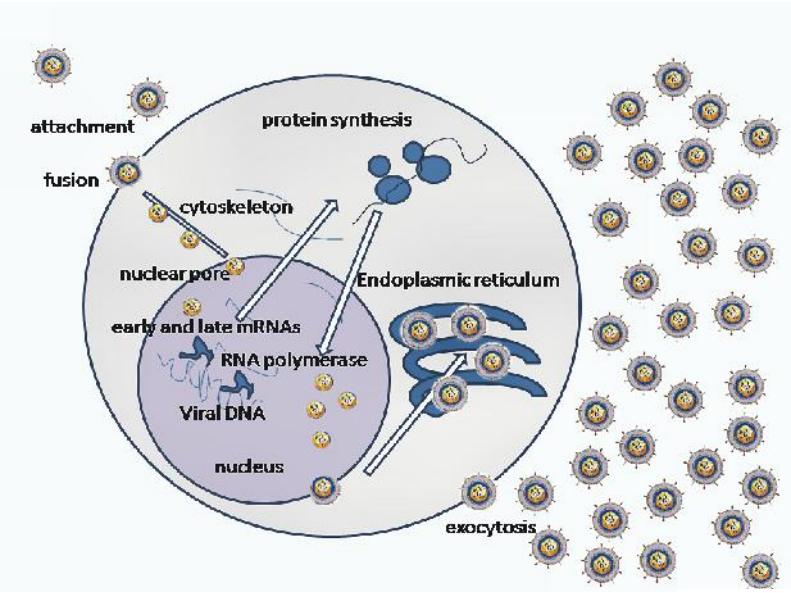


**Figure 2:** The general overviews of the *BoHV-1* replication cycle (Chatterjee *et al*., 2016)

### 2.3.3 The Strain of *BoHV-1* for Pathogenecity

There are three subtypes of *BoHV-1*: *BoHV-1.1*, *BoHV-1.2a*, and *BoHV-1.2b*. The *BoHV-1.1* is usually associated with respiratory infections and abortions, whereas the *BoHV-1.2a* is for genital tract infections. A third subtype of *BoHV-1.2b*is not associated with abortion. In trials,both*BoHV-1.1*and *BoHV-1.2a*can result in genital and respiratory infections, respectively. Although *BoHV-1.1*, *BoHV-1.2a*, and *BoHV-1.2b* have similar antigenic characteristics, restriction enzyme fragment polymorphisms (REFPs) allow for their differentiation (Muylkens *et al.*, 2007).

### 2.3.4 *BoHV-1* Pathogenecity

*BoHV-1* infects epithelial cells in the upper respiratory airway and genital tract mucosa, and it establishes a productive infection on the mucosal surfaces. Interactions between viral glycoproteins and host cell receptors allow viruses to enter the cell (Ellis, 2009).On the other hand, the effective synergy between innate and adaptive immune responses relies heavily on antigen-presenting cells such as macrophages and dendritic cells. These cells phagocytose invading pathogens and present specific antigens via major histocompatibility complex (MHC) class I andII proteins, along with the transporter associated with antigen processing (TAAP) (Abele and Tampe, 2004; Gaudino and Kumar, 2019). *BoHV-1* actively disrupts this communication by infecting and down-regulating the expression of signaling molecules within lymphocytes. As a result, this viral infection is known to inhibit the translocation of internalized MHC class I receptors to the surface of infected cells (Koppers-Lalic *et al*., 2003), and MHC receptor expression is also intrinsically down-regulated (Nataraj *et al*., 1997).

Those newly synthesized MHC receptors are found in abundance in the endoplasmic reticulum of *BoHV-1*-infected cells, suggesting that interference with TAAP-dependent transport mechanisms is responsible for the lack of lymphocyte activation during infection (Hinkley *et al*., 1998; Koppers-Lalic *et al*., 2003). Infected animals shed virus from respiratory mucous membranes and secretions or genital mucous membranes and secretions for 8–16 days after exposure (Dinter and Morein, 1990).Latently infected animals do not consistently shed virus and do not always have detectable levels of neutralizing antibodies in serum. Therefore, detection of infection in these animals is achieved by viral detection following administration of dexamethasone,according to the concepts of experimental research (Marin *et al*., 2016; Marawan *et al*., 2021), and recrudescence of the virus**.** Libido in bulls may be decreased and resumption of normal breeding behavior delayed for several weeks (Nandi *et al*., 2009), which means that the semen production and distribution center will be paid extravagantly for feedings and health care.

## 2.4 Prevention and control of IBR

The incidence of *BoHV-1* infection has drawn considerable attention in developing countries owing to its effect on the internal movement of livestock and germplasm. The presence of *BoHV-1*-specific antibodies in the serum of infected animals can be used to identify them. Because of the risk of reactivation of the latent virus, every affected animal must be identified and culled from a herd in order to eradicate *BoHV-1*(Lugaj *et al*., 2020)and even prevent the spread of the virus from these infected herds to the neighboring dairy and beef farms.

### 2.4.1 Management strategies

This management approach must aim to lessen productivity losses brought on by various diseases and poor management, all the while ensuring the best care and well-being of dairy cattle. Intermingling or mixing of subclinically infected animals poses the greatest risk for the first introduction and transmission of many infectious diseases into a herd; however, wildlife carriers pose some risk as well (Sibhat *et al*., 2018). As a result, closed farming systems, avoiding bull sharing for natural services, routine surveillance (Jones, 2019), and culling PI animals from herds have been put in place (Lugaj *et al*., 2020).

### 2.4.2 Biosecurity

The primary implications of a dairy farm's biosecurity are mainly focused on the reduction and prevention of the introduction of new diseases from outside sources, as well as the reduction and/or prevention of the movement of infectious diseases on the farm (Baraitareanu and Vidu, 2020).Therefore, biosecurity action plans must be implemented primarily on large dairy farms, where disease agents can be introduced through a variety of means, including labor, advisers, replacement cattle, supplies, feedstuffs, and vehicles (Villarroel *et al*., 2007).

### 2.4.3 Vaccination

Different vaccine types are currently available in different countries and are used for the prevention and control of the disease(Iscaro *et al*., 2021). Vaccination is frequently used to clinically protect cattle from infection and significantly reduce viral shedding. Herd immunity is provided through vaccination with inactivated and live attenuated vaccines, which lowers the likelihood of an animal coming into contact with an infected animal. The typical duration of immunity is six months to a year. The typical duration of immunity is six months to a year. To distinguish infected animals from those who have received vaccinations, it is also possible and recommended to use marker vaccines, which are also known as the differentiation of infected from vaccinated animals (DIVA) test(Strube*et al*., 1996; Bosch *et al*., 1997).

## 3. Conclusion and Recommendations

Infectious bovine rhinotracheitis is caused by *BoHV-1*, which is a double-stranded DNA virus. The viral glycoproteins gB, gC, and gD interact with cellular membranes to allow for virus entrance and infection to begin. During entry into the host cells, the virus loses the lipid envelope and sheds some of its tegument proteins. The capsid then releases the viral DNA into the nucleus through the nuclear pore complex, where the DNA replicates via a rolling circle mechanism. The effective synergy between innate and adaptive immune responses relies heavily on macrophages and dendritic cells, but this viral infection disrupts this communication by infecting and down-regulating the expression of signaling molecules within lymphocytes. In this paper, I discuss how the three glycoproteins mentioned above are required for viral infections to occur in susceptible hosts, and how the tegument protein VP8 is required for viral replication in the host infected cell. Therefore, for disease prevention and control purposes, vaccination is used to protect cattle from infection and reduce viral shedding. Based on the above conclusion, the following recommendation is forwarded. Furthermore, the investigations that will deliver a more comprehensive understanding about the remaining viral proteins and their molecular basis that are responsible for the pathogenicity of *BoHV-1* and the most effective preventive and control approach for the disease are recommended for the feature.

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