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Correlation between viral infections in male semen and infertility: a literature review

Yan Guo[1,4,5†], Yunhua Dong[2,3,4†], Runzi Zheng[2,3,4,5], Jiacong Yan[2,3,4], Weiyuan Li[2,3,4], Ya Xu[6], Xuelan Yan[1,4], Yunmei Ke[1,4], Yantao Li[1,4] and Lifeng Xiang[2,3,4*]

Abstract

Infertility affects approximately one-sixth of couples globally, with the incidence of male infertility steadily increasing. However, our understanding of the impact of viral infections on fertility remains limited. This review consolidates findings from previous studies, outlining 40 viruses identified in human semen and summarizing their key characteristics, modes of transmission, and their effects on both the reproductive and endocrine systems. Furthermore, it elucidates potential pathogenic mechanisms and treatment prospects of viruses strongly associated with male infertility. This synthesis will enhance our comprehension of how viral infections influence male reproductive health, offering valuable insights for future research as well as the diagnosis and treatment of infectious infertility.

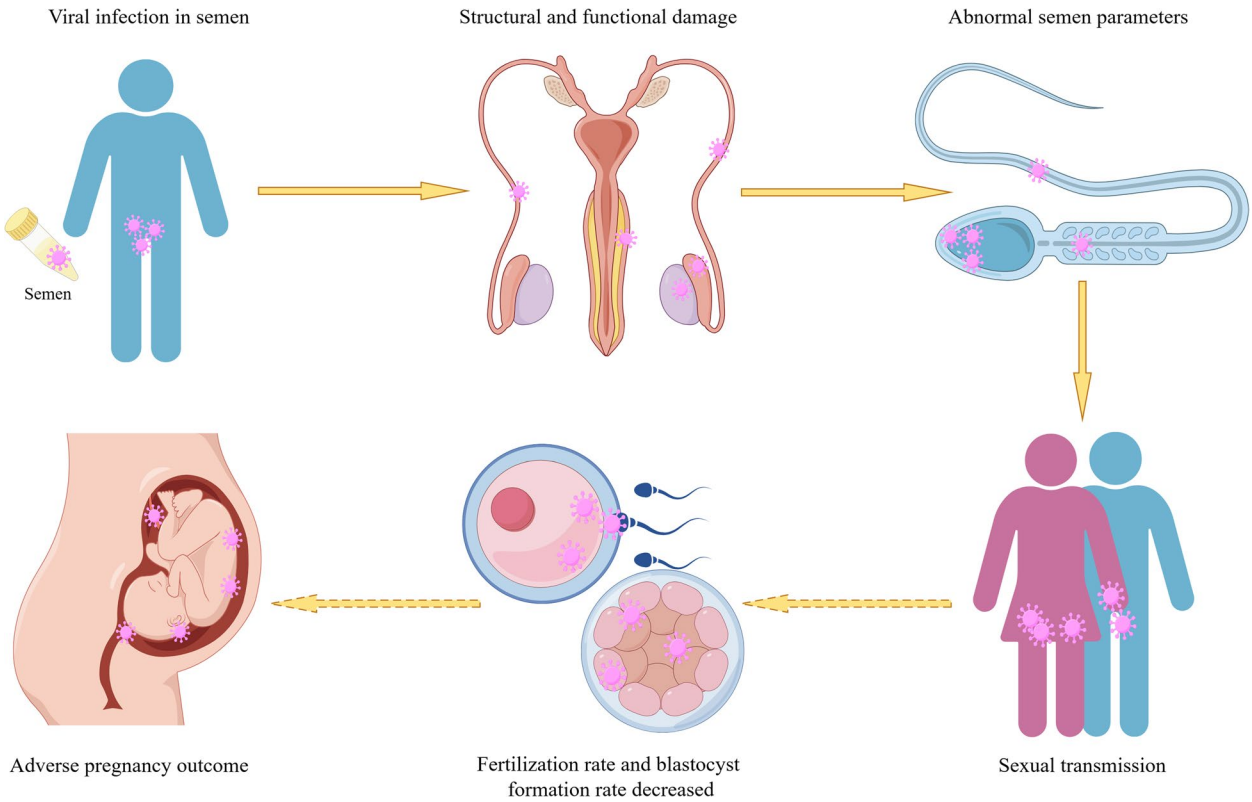
Keywords Virus, Semen, Infection, Infertility, Reproduction, Sexual transmission

†Yan Guo and Yunhua Dong contributed equally to this work.

*Correspondence:
Lifeng Xiang
xlflotus@aliyun.com
Full list of author information is available at the end of the article

Graphical Abstract

Abbreviated summary: Semen viral infections can damage the male reproductive system, further affecting semen quality and causing infertility, as well as sexually transmitted infections to partners and vertical transmission resulting in poor pregnancy outcomes.



Introduction

The WHO explicitly defines infertility as the inability to conceive after at least 12 months of regular, unprotected sexual intercourse [1], which may stem from factors related to female, male, or both [2]. It's reported that about 17% of couples globally grapple with infertility [3], with male infertility constituting half of all cases [4]. In recent years, male infertility has garnered increasing attention. A global burden of disease study encompassing 204 countries and territories revealed that in 2019, the prevalence of male infertility was approximately 56 million, marking a 76.9% increase since 1990 [5].

The reasons for decreased male fertility vary but may be linked to congenital, acquired, or idiopathic factors affecting sperm production [6], among which reproductive tract infections are considered to be one of the most influential [7, 8]. A survey involving over 4000 infertile men showed a prevalence rate of genitourinary tract infections in males as high as 35% [9]. Furthermore, 20% of primary infertile men exhibited asymptomatic semen infections, resulting in varying degrees of abnormal sperm concentration [10].

Previous research has compiled a list of 38 viruses detected in the male reproductive tract and semen, many of which exhibit a strong affinity for the male reproductive organs, particularly the testes [11]. Moreover, amidst the global coronavirus disease 2019 (COVID-19) pandemic, accumulating studies have indicated that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) can directly or indirectly impair the male reproductive system, thereby heightening the risk of male infertility [12–14]. This resurgence of concern highlights the potential impact of viral infections on male fertility. Within semen, viruses may infect sperm or precursor cells, attaching to molecules on the sperm surface as free viral particles or residing within immune cells, leading to pathology in the reproductive system, abnormalities in semen parameters, and declined sperm quality. This

poses a significant threat to individual fertility and overall health. Furthermore, this may also result in virus-induced mutations transmitted to future generations [15].

Despite recent advancements in the study of semen viruses, a definitive correlation between viral infections and fertility remains unclear. This review seeks to summarize the primary discoveries regarding viruses in human semen, delineate the diversity of viruses present, identify the key viral species linked to male infertility, and discuss potential avenues of virus research with clinical relevance and implications for fertility guidance.

Methods

To investigate the presence of viruses in semen, we conducted an unrestricted search on PubMed, Embase and Web of Science using the terms "semen, sperm, virus," and we obtained 4815 results. Through screening titles, abstracts, and full-text articles, we gathered data describing viral infections in semen. Subsequently, we conducted another search using the term "(virus name) and sexual transmission" to find relevant evidence of sexual transmission.

Results

Our findings revealed the presence of 40 viruses in human semen, with some lacking data on sexual transmission. Among these 40 viruses, many cause chronic or latent infections such as herpes viruses and human immunodeficiency virus (HIV), while others cause acute infections such as zika virus (ZIKV), ebola virus (EBOV), and chikungunya virus (CHIKV). Among the viruses causing acute infections, only ZIKV and EBOV have been systematically studied beyond case reports. Table 1 summarizes the main characteristics, clinical manifestations, and key information related to male fertility for the 40 viruses. In the following sections, we will provide detailed explanations regarding viruses highly relevant to male reproductive health.

Human papillomavirus

Human papillomavirus (HPV) is a virus species with double-stranded circular DNA, and it belongs to the *Papillomaviridae* family. It is classified into high-risk and low-risk types based on its carcinogenicity [139]. HPV is one of the most common sexually transmitted pathogens worldwide, with approximately 12% of the population infected and over 6.2 million new cases reported annually [140, 141]. Sexual contact is the primary route of HPV transmission. Males not only serve as carriers but also play a crucial role as transmitters in the epidemiological chain of HPV [142]. High-risk HPV is associated with anal cancer, penile cancer, and some head and neck cancers in males [143]. However, HPV may also exist in asymptomatic males [144]. The prevalence of HPV DNA in semen has been reported to range from 0.0% to 46.2%, with an average of 17.1% (95% CI = 14.1 to 20.1%). The prevalence in fertility clinics (20.4%, 95% CI = 16.2 to 24.6%) is significantly higher than that in the general population (11.4%, 95% CI = 7.8 to 15.0%) ($P < 0.001$), and the prevalence of high-risk HPV (15.5%, 95% CI = 11.4 to 19.7%) is significantly higher than that of low-risk HPV (10.3%, 95% CI = 6.8 to 13.9%) ($P < 0.001$) [145]. Notably, the detection rate of HPV in donor sperm from sperm banks ranges from 3.1% to 16.7%, significantly impacting the clinical pregnancy rate in assisted reproduction. Therefore, it is recommended that donor sperm should be tested for HPV before being used for insemination [146].

The mechanisms by which sperm function may be impaired after HPV infection are still poorly understood. HR-HPV proteins cause inflammation and increase reactive oxygen species (ROS) levels in host cells, leading to oxidative stress (OS) [147]. Additionally, HPV infection directly inhibits the function of aquaporins (AQPs), making sperm cells more sensitive to OS. This inhibition reduces the AQP-mediated detoxification mechanism, resulting in sperm distress and impaired sperm function [148]. Research indicates that the presence of glycosaminoglycans or other soluble substances on the surface of sperm facilitates HPV attachment to the equatorial region of the sperm head [149]. When the equatorial region fuses with the oocyte plasma membrane [150], HPV may adversely affect fertilization. Certain HPV genotypes are associated with sperm DNA fragmentation [151], decreased motility and fertilization potential [152], abnormal sperm quality [19], and the formation of antisperm antibodies (ASAs) [153]. HPV transinfection may reduce the ability of trophoblasts and embryonic membranes to invade, leading to placental dysfunction and adverse pregnancy outcomes [154]. Viral genes may be transmitted to oocytes and embryo cells, causing DNA fragmentation and apoptosis, ultimately resulting in pregnancy loss [155, 156].

The guidelines issued by the European Society of Human Reproduction and Embryology (ESHRE) state that HPV in semen is a viral factor highly associated with assisted reproduction outcomes. They recommend targeted counseling for couples undergoing assisted reproductive therapies (ART) who are infected [157]. For couples with fertility problems, if they have unexplained infertility, a history of HPV infection, show related clinical manifestations, or if there is the presence of ASA and asthenospermia, HPV DNA testing and genotyping are recommended for the male partner. When HPV is detected, fluorescence in situ hybridization (FISH) analysis is recommended to check for the

Table 1 Summary of virus species detected in semen

Virus	Family	Genus	Genome	Clinical presentation	Sexual transmission reported (Y/ N/ NS)	Effect on male reproductive health	References
Human papillomavirus (HPV)	*Papillomaviridae*	*α-,β-, γ-Papillomavirus*	dsDNA	Warts, anal cancer, penile cancer, head and neck cancer	Y	Abnormal semen parameters, increased sperm DNA fragmentation index, formation of anti-sperm antibodies, placental dysfunction, pregnancy loss	[16–34]
Herpes simplex virus-1 (HSV-1)	*Herpesviridae*	*Simplexvirus*	dsDNA	Skin lesion	Y	Average sperm count decreased	[35–47]
Herpes simplex virus-2 (HSV-2)	*Herpesviridae*	*Simplexvirus*	dsDNA	Genital, anal blisters or ulcers	Y	Hematospermia, decreased mean semen volume	
Varicella zoster virus (VZV)	*Herpesviridae*	*Varicellovirus*	dsDNA	Chickenpox, shingles	Y	NS	[41, 45, 48]
Epstein-Barr virus (EBV)	*Herpesviridae*	*Lymphocryptovirus*	dsDNA	Infectious mononucleosis	Y	Leukocyte spermatozoa and average sperm count increased	[38, 39, 41, 45, 49–51]
Cytomegalovirus (CMV)	*Herpesviridae*	*Cytomegalovirus*	dsDNA	Congenital viral infections	Y	Normal-form sperm count and motility decrease, increasing the risk of early miscarriage and fetal birth defects	[38, 41, 47, 49, 51–55]
Human herpesvirus-6 (HHV-6)	*Herpesviridae*	*Roseolovirus*	dsDNA	Infantile roseola	Y	NS	[39, 41, 49, 56, 57]
Human herpesvirus-7 (HHV-7)	*Herpesviridae*	*Roseolovirus*	dsDNA	Rash, fever without rash, febrile convulsions, persistent status epilepticus	NS	NS	[57]
Kaposi sarcoma-associated herpesvirus (KSHV)	*Herpesviridae*	*Roseolovirus*	dsDNA	Kaposi's sarcoma, primary exudative lymphoma, multicenter Castleman's disease	Y	NS	[58–62]
Hepatitis B virus (HBV)	*Hepadnaviridae*	*Orthohepadnavirus*	dsDNA (RT)	Hepatitis, cirrhosis, hepatocellular carcinoma	Y	Decrease in sperm concentration, (progressive motile sperm) PMS, sperm motility, sperm viability and morphologically normal sperm (MNS)	[50, 63–68]
Hepatitis C virus (HCV)	*Flaviviridae*	*Hepacivirus*	ssRNA (+)	Hepatitis, cirrhosis, hepatocellular carcinoma	Y	Decreased sperm count and sperm motility, abnormal hormone levels, erectile dysfunction	[69–76]
Hepatitis D virus (HDV)	*Arenaviridae*	*Delta virus*	ssRNA (-)	Hepatitis, cirrhosis, hepatocellular carcinoma	NS	NS	[77]
Hepatitis E virus (HEV)	*Hepeviridae*	*Orthohepevirus, Piscihepevirus*	ssRNA (+)	Acute hepatitis, jaundice	Y	NS	[78]
Hepatitis G virus (HGV)	*Flaviviridae*	*Flavivirus*	ssRNA (+)	Hepatitis, liver fibrosis	NS	NS	[79, 80]

Table 1 (continued)

Virus	Family	Genus	Genome	Clinical presentation	Sexual transmission reported (Y/ N/ NS)	Effect on male reproductive health	References
JC polyomavirus (JCPyV)	*Polyomaviridae*	*Betapolyomavirus*	dsDNA	Progressive multifocal leukoencephalopathy (pML), chronic meningoencephalitis	Y	Sperm motility decreased and sperm morphology changed	[81–83]
Merkel cell polyomavirus (MCPyV)	*Polyomaviridae*	*Alphapolyomavirus*	dsDNA	Syphilocellular carcinoma	Y	NS	[81]
MW polyomavirus (MWPyV)	*Polyomaviridae*	*Deltapolyomavirus*	dsDNA	NS	Y	NS	[81]
BK polyomavirus (BKPyV)	*Polyomaviridae*	*Betapolyomavirus*	dsDNA	Hemorrhagic cystitis	Y	NS	[84]
Simian virus 40 (SV40)	*Polyomaviridae*	*Betapolyomavirus*	dsDNA	Brain tumor	Y	NS	[85]
Mumps virus (MuV)	*Paramyxoviridae*	*Orthomyrovirus*	ssRNA (-)	Mumps	NS	Orchitis, decreased sperm count, abnormal sperm morphology, anti-sperm antibody production	[86]
Human immunodeficiency virus (HIV)	*Retroviridae*	*Lentivirus*	ssRNA (RT)	Acquired immunodeficiency syndrome (AIDS)	Y	Abnormal morphology, semen pH value increases and the number of round cells increases, sperm motor capacity, sperm count and ejaculation volume decreases, testicular morphology and sperm abnormalities, hormone secretion disorders	[87–93]
Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)	*Coronaviridae*	*Betacoronavirus*	ssRNA (+)	Pneumonia	Y	Semen concentration, sperm count and motility decreased, and testosterone secretion decreased	[94–101]
Adeno-associated virus (AAV)	*parvoviridae*	*Dependent virus*	sDNA (+)	Nonpathogenicity	Y	Decreased sperm motility	[48, 102–104]
Zika virus (ZIKV)	*Flaviviridae*	*Flavivirus*	ssRNA (+)	Guillain–barre syndrome (GBS), myelitis, meningoencephalitis, congenital microcephaly	Y	Hematospermia, decreased sperm count, abnormal hormone secretion, cell death, destruction of spermatogenic tubules, and testicular injury	[105–113]
Monkeypox virus (MPXV)	*Poxviridae*	*Orthopoxvirus*	dsDNA	Monkeypox	Y	Decreased sperm quality	[114–116]
Human T-lymphotropic virus (HTLV)	*Retroviridae*	*Deltaretrovirus*	ssRNA (RT)	T-cell leukemia/lymphoma, HTLV-1-associated myelopathy, HTLV-1-associated mastitis	Y	NS	[117–119]
Ebola virus (EBOV)	*Filoviridae*	*Ebolavirus*	ssRNA (-)	Hemorrhagic fever	Y	Decreased sexual function	[120–122]

Table 1 (continued)

Virus	Family	Genus	Genome	Clinical presentation	Sexual transmission reported (Y/ N/ NS)	Effect on male reproductive health	References
West-Nile virus (WNV)	*Flaviviridae*	*Flavivirus*	ssRNA (+)	Influenza-like symptoms, meningitis, encephalitis, encephalomyelitis	Y	NS	[123]
Dengue virus (DENV)	*Flaviviridae*	*Flavivirus*	ssRNA (+)	Dengue fever, dengue hemorrhagic fever, dengue shock syndrome	Y	Sperm count and motility decreased	[124]
Chikungunya virus (CHIKV)	*Togaviridae*	*Alphavirus*	ssRNA (+)	Chikungunya, hepatitis, hemorrhagic meningitis, myocarditis, acute respiratory distress syndrome	Y	NS	[125, 126]
Coxsackie virus (CV)	*Picornaviridae*	*Enterovirus*	ssRNA (+)	Hand, foot and mouth disease, mumps, myocarditis, meningitis	NS	Orchitis	[127]
Marburg virus (MARV)	*Filoviridae*	*Marburgvirus*	ssRNA (-)	Marburg hemorrhagic fever	Y	NS	[128]
Rift Valley fever virus (RVFV)	*Bunyaviridae*	*Phlebovirus*	ssRNA (-)	Rift valley fever	NS	NS	[129]
Adenovirus (Ad)	*Adenoviridae*	*Mastadenovirus*	dsDNA	Conjunctivitis, respiratory or gastrointestinal syndrome, meningoencephalitis, urinary tract infection	NS	NS	[130]
Chapare virus (CHAPV)	*Arenaviridae*	*Chaparevirus*	ssRNA (-)	Hemorrhagic fever	NS	NS	[131]
Toscana virus (TOSV)	*Bunyaviridae*	*Phlebovirus*	ssRNA (-)	Toscana virus disease	Y	Orchitis, decreased sperm motility	[132]
Lassa virus (LASV)	*Arenaviridae*	*Arenavirus*	ssRNA (-)	Lassa fever(LF)	Y	Epididymitis	[133–135]
Severe fever with thrombocytopenia syndrome virus (SFTSV)	*Bunyaviridae*	*Phlebovirus*	ssRNA (-)	Severe fever with thrombocytopenia syndrome (SFTS)	Y	NS	[136]
Nipah virus (NiV)	*Paramyxoviridae*	*Henipavirus*	ssRNA (-)	Acute respiratory disease, fatal encephalitis	NS	NS	[137]
Yellow Fever Virus (YFV)	*Flaviviridae*	*Flavivirus*	ssRNA (+)	Yellow fever	NS	NS	[138]

Abbreviations: *Y* yes, *N* no, *NS* not specified

presence of HPV DNA on the sperm surface, and colposcopy is suggested to rule out subclinical genital lesions [158]. Since some infertile couples cannot afford to delay conception and standard washing procedures in ART are insufficient to reduce semen viral load, two potential strategies are proposed for infertile couples with HPV-infected semen: special sperm washing and HPV adjunctive vaccination. Research indicates that applying a sperm washing procedure based on hyaluronidase (IALu) helps remove HPV viral particles from the sperm surface [159]. Additionally, vaccination of infected patients has been shown to shorten the viral clearance time in semen [160], reduce the risk of recurrence [160], and improve sperm quality [144].

Herpesviruses

Members of the *Herpesviridae* family are enveloped spherical viruses with linear double-stranded DNA genomes, and their size ranges from 125 to 241 kbp, containing 70 to 170 genes [161]. Nine types of herpesviruses infect humans, categorized into three subfamilies: *α-herpesviruses*, *β-herpesviruses*, and *γ-herpesviruses*, all capable of persisting in host cells and causing recurrent infections [162]. Members of the herpesvirus family have been detected in numerous semen studies and can be transmitted through sexual intercourse or from mother to baby during pregnancy and childbirth [15, 163]. PCR assay is the preferred method for distinguishing type-specific HSV. Antiviral medications can effectively reduce the severity, duration, and recurrence of the disease, as well as prevent transmission to uninfected partners [164].

Herpes simplex virus-1/2

Herpes simplex virus-1/2 (HSV-1/2) belongs to the *α-herpesvirus* subfamily. HSV-1 primarily causes oral and labial herpes, while HSV-2 mainly causes genital herpes. Both HSV-1 and HSV-2 can be transmitted sexually [163], with a mother-to-child transmission rate of approximately 1:1,400–30,000, sometimes leading to life-threatening widespread infections in newborns [165].

There is significant variation in the reported infection rates of HSV-1 and HSV-2 in semen. One study revealed detection rates of 22.9% for HSV-1 and 14.3% for HSV-2 in semen samples from infertile men, with all HSV-positive samples exhibiting abnormal semen parameters [43]. Another study indicated a detection rate of HSV in 10.7%, with 7.5% positive for HSV-1 and 3.2% positive for HSV-2. HSV-1 infection was associated with decreased sperm count, while HSV-2 infection was associated with hematospermia [44]. Two other studies reported detection rates of HSV at 2.5% and 3.2%, respectively, with no observed impact on semen parameters [41, 57]. Evidence from experiments has shown that components in semen such as prostatic acid phosphatase (PAP), seminalplasmin (SEM), and seminal plasma (SP) can promote the formation of HSV particles and accelerate virus replication, indicating that semen is an important target for HSV [166]. In addition, HSV-2 can internalize into the head of sperm, potentially influencing pregnancy outcomes [167].

Varicella-zoster virus

Varicella-zoster virus (VZV) belongs to the *α-herpesvirus* subfamily and is responsible for causing chickenpox and shingles. Research on VZV in semen is relatively scarce. One study detected VZV in patients with teratozoospermia but did not explicitly state its correlation with the condition [45]. Other studies reported detection rates of VZV in semen ranging from 1.2% to 4%, but no association with semen quality was found [41, 48]. Additionally, some studies did not detect VZV in semen samples [57, 168, 169].

Epstein-barr virus

Epstein-barr virus (EBV) belongs to the *γ-herpesvirus* subfamily and is a lymphotropic herpesvirus. Infection can occur in individuals at different times, primarily through saliva transmission, but can also be transmitted through genital secretions or blood. Approximately 3% of EBV-positive mothers may transmit the virus to their babies [170]. Studies have shown that the prevalence of EBV in semen ranges from 0.4% to 45% [45, 50, 51] and may be associated with leukocytospermia [41]. Semen samples from infertile individuals have a significant capacity to induce early antigen (EA) of EBV, which may be related to reproductive damage mediated by immune responses and tumor development [171, 172].

Cytomegalovirus

Cytomegalovirus (CMV) belongs to the *β-herpesvirus* subfamily and is the largest and most genetically variable virus among human herpesviruses, with a global infection rate of approximately 66% to 90%. CMV can establish latency in long-lived cells and reactivate periodically, posing a risk of severe disease in immunocompromised individuals and being one of the most common causes of congenital disabilities [173]. Transmission mainly occurs through direct contact with body fluids such as saliva, urine, or semen [174].

Since CMV presence in semen was first reported in the United States in 1974 [54], relevant studies have continuously emerged. The prevalence of CMV in semen ranges approximately between 6% and 56.9% [51], and it has been associated with decreased sperm count and motility, as well as an increased failure rate in assisted reproduction [49, 52, 53]. However, some studies suggest

that the presence of CMV in semen is not related to male infertility [41, 55]. It has been reported that CMV in semen can be transmitted to partners through sexual intercourse [175] and can infect the endometrium, leading to early miscarriage and fetal birth defects [176].

Human herpesvirus 6A/B

Human herpesvirus 6 (HHV-6) belongs to the *β-herpesvirus* subfamily and includes HHV-6A and HHV-6B. HHV-6B infects almost 100% of humans, typically before the age of 4, and is the pathogen of exanthema subitum (infant roseola). Little is known about the transmission routes and prevalence of HHV-6A, but recent research suggests a link between high levels of HHV-6A antibodies and multiple sclerosis [177]. HHV-6 typically integrates its genome into the telomeres of host cells to establish latency. Additionally, HHV-6A/B can integrate into the chromosomes of germ cells, leading to offspring carrying copies of the viral genome, a condition known as inherited chromosomally integrated HHV-6 (iciHHV-6) [178].

One study indicated a detection rate of HHV-6 in semen of 5.6% and a higher prevalence among infertile males with inflammatory diseases of the reproductive tract (19%) [49]. Two other studies reported detection rates of HHV-6 in semen at 4% and 70%, respectively, but found no correlation with semen quality [41, 57].

Human herpesvirus-7

Human herpesvirus-7 (HHV-7) belongs to the *β-herpesvirus* subfamily, with a global infection rate exceeding 90%. Primary infection often occurs in early childhood, manifesting with different clinical presentations such as rashes, fever, and febrile seizures [179]. There is limited research on HHV-7 in semen. One study found no evidence of HHV-7 infection in 172 semen samples [41], while another study detected only 1 case (0.4%) out of 252 semen samples [57]. Some research has detected HHV-7 in placental samples [180], but its mechanisms of sexual and mother-to-child transmission require further confirmation.

Human herpesvirus-8

Human herpesvirus-8 (HHV-8), also known as Kaposi's Sarcoma-Associated Herpesvirus (KSHV), belongs to the *γ-herpesvirus* subfamily and is an oncogenic pathogen that causes Kaposi's sarcoma (KS) [181]. HHV-8 primarily spreads through saliva and enters latency after cell infection, with reactivation leading to disease occurrence [182]. In the general population, the prevalence of HHV-8 is low, with most studies unable to detect HHV-8 in semen from healthy donors or infertile males [38, 57]. However, the detection rate of HHV-8 in semen from KS patients is 12% [59], and HHV-8 has also been detected in semen from HIV-1-infected individuals [62]. The prevalence of HHV-8 in semen from healthy males has not yet been determined.

Hepatitis viruses

Hepatitis B virus

Hepatitis B virus (HBV) belongs to the *Hepadnaviridae* family and affects nearly 400 million people worldwide with chronic hepatitis B, posing a significant global healthcare challenge [183]. It can be transmitted through body fluids such as blood, semen, and vaginal secretions. Most individuals with normal immune function can spontaneously clear the infection, but some may develop acute or chronic hepatitis or even progress to liver cirrhosis and hepatocellular carcinoma [184].

Studies have found HBV in semen [50, 68], suggesting the reproductive tract may serve as an independent reservoir for the virus, transmitting it through sexual intercourse [65]. The HBV genome can also integrate into sperm chromosomes, leading to chromosomal aberrations and even hereditary effects through vertical transmission [185]. HBV infection in semen can induce abnormal cytokine expression [186], trigger cell apoptosis, sperm DNA fragmentation, and reduce fertilization capacity [187]. HBV infection in males has been observed to decrease the success rates of assisted reproduction [188], although some studies suggest HBV positivity in semen has no impact on assisted reproduction and pregnancy outcomes [189]. In male HBV patients, sperm washing procedures can effectively reduce the risk of vertical transmission and prevent HBV from entering the oocyte during intracytoplasmic sperm injection (ICSI). Additionally, in vitro studies have shown that the risk of infected sperm cells acting as carriers in IVF is no different from that in ICSI for male HBV patients. Therefore, when the male partner is a chronic HBV carrier, there is no reason to exclude a couple from undergoing ICSI [190].

Hepatitis C virus

Hepatitis C virus (HCV) belongs to the *Flaviviridae* family [191] and is a major cause of liver cirrhosis and hepatocellular carcinoma. It is estimated that approximately 56.8 million people worldwide are infected with HCV, with a prevalence rate of 0.7% [192]. HCV is primarily transmitted through blood but can also be transmitted through other body fluids such as saliva, urine, and semen, indicating the possibility of sexual transmission [193]. Several studies have also demonstrated the presence of HCV in semen [72–74], correlating with decreased semen quality, abnormal hormone levels, and erectile dysfunction [75, 76]. Medications used to treat

HCV fall into two main categories: immunomodulators and antiviral agents. However, interferon therapy can reduce male fertility due to its gonadotoxic effects [194].

Hepatitis E virus

Hepatitis E virus (HEV) belongs to the *Hepeviridae* family and is a common pathogen causing acute hepatitis and jaundice. Globally, around 200,000 people are infected with HEV annually, with at least five genotypes capable of human infection. HEV-1 and HEV-2 are primarily transmitted through the fecal–oral route, while HEV-3, HEV-4, and HEV-7 are often associated with zoonotic transmission and may spread through food and blood transfusions [195]. Research on HEV in the male reproductive system is limited and controversial. One study found a prevalence of HEV-4 in semen samples from infertile men, with 28.11% (52/185) of samples testing positive for HEV RNA. It was confirmed that HEV-4 infection could disrupt the blood-testis barrier (BTB), infect testicular cells, and reduce sperm quality [78]. However, other studies have failed to detect HEV in semen samples [196–198].

Polyomaviruses

Polyomaviruses (PV) belong to the *Polyomaviridae* family, a group of small, non-enveloped, double-stranded DNA viruses named for their association with various tumors. The Polyomaviridae family is divided into six genera, comprising 117 different viruses, with 14 known to infect humans [199]. The most relevant ones to human disease are the BK virus (BKPyV) and JC virus (JCPyV) [200]. These two viruses are widespread among the global population, with primary infection typically occurring in childhood or adolescence, establishing persistent or latent infections that may reactivate when the immune system is compromised. Blood, oral-fecal, urine, and sexual contact may be routes of transmission for PV [201, 202]. Studies have found the presence of JCPyV, Merkel cell polyomavirus (MCPyV), MW polyomavirus (MWPyV), STL polyomavirus (STLPyV), and Simian virus 40 (SV40) in semen [81, 85, 203]. Comar et al. first reported an infection rate of 24.5% for JCPyV in semen samples from infertile men, significantly associated with decreased sperm motility and morphological changes [82]. In addition, Rotondo et al. also identified JCPyV DNA in semen samples [83].

Mumps virus

The mumps virus (MuV) is a negative-sense RNA virus belonging to the *Paramyxoviridae* family. MuV is the pathogen of mumps, primarily transmitted through direct contact or respiratory droplets [204]. MuV also demonstrates a high affinity for the testes, leading to orchitis, which is a common cause of viral orchitis and male infertility [205]. In adult mumps patients, approximately 20–30% may develop orchitis, and among affected testes, 30–50% may experience testicular atrophy [206]. During the early stages of MuV infection, the virus can induce parenchymal inflammation, lymphocytic infiltration, and damage to the seminiferous tubules, thereby affecting testicular function and hormone levels [207]. A study found that MuV can be detected in patients' semen, resulting in decreased sperm count, abnormal morphology, and the production of anti-sperm antibodies, which may potentially have long-term adverse effects on fertility [86]. The diagnosis of MuV primarily relies on clinical complications and laboratory tests. The characteristic manifestation of orchitis is testicular swelling and pain. Laboratory diagnosis depends on MuV culture, viral RNA detection, or more commonly, serological confirmation by measuring immunoglobulin antibody levels [206]. MuV infections are mostly self-limiting, and there is currently no specific antiviral therapy. Treatment for mumps orchitis typically includes bed rest, scrotal support, and the use of analgesic and anti-inflammatory medications [208].

Human immunodeficiency virus

Human immunodeficiency virus (HIV) is a virus species with single-stranded positive RNA, and it belongs to the *Retroviridae* family. Acquired immunodeficiency syndrome (AIDS) is a major global public health concern, with an estimated 39 million people infected with HIV as of 2022. HIV is primarily transmitted through unprotected sexual intercourse, with semen serving as the primary carrier of transmission [209].

HIV infection can contaminate semen at any stage [89], existing in the form of both free viral particles and infected cells [90]. There may be genetic variations in viral RNA and DNA sequences in semen during acute and chronic infection, indicating independent viral replication in semen [91]. The impact of HIV infection on semen parameters mainly includes decreased sperm motility, reduced quantity, increased abnormal morphology, decreased ejaculate volume, elevated semen pH, and increased round cell count [92, 93]. Chronic orchitis may occur in AIDS patients, affecting testicular function and hormone production [210]. Antiretroviral therapy can reduce viral load in semen, thereby lowering the risk of transmission [211]. However, antiviral drugs also pose a risk of reducing sperm quality. This negative impact can be mitigated by using ICSI [212]. The effectiveness of sperm washing in eliminating viral particles from semen remains controversial, and improvements to washing procedures may be needed in the future [212–214].

Severe acute respiratory syndrome coronavirus 2

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a single-stranded positive-sense RNA virus belonging to the *Coronaviridae* family, primarily transmitted through respiratory droplets. The COVID-19 pandemic has led to a massive outbreak worldwide, infecting billions of people and causing millions of deaths [215].

Coronaviruses bind to host cells via the spike (S) protein and the cell receptor angiotensin-converting enzyme 2 (ACE2), and entry into host cells is facilitated by the S protein activated by type II transmembrane serine protease (TMPRSS2) [216]. The high expression of ACE2 and TMPRSS2 in the testes suggests they are target organs for SARS-CoV-2 [217]. Autopsies of individuals infected with SARS-CoV-2 revealed testicular findings such as interstitial edema, congestion, and erythrocyte extravasation. There was an increased proportion of CD3 + and CD68 + leukocytes in the testicular interstitium. Additionally, there were increases in OS, as well as elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and monocyte chemoattractant protein-1 (MCP-1) [218, 219]. Studies have confirmed severe damage to the testes following SARS-CoV-2 infection, including testicular atrophy, inflammatory cell infiltration, germ cell apoptosis, and microthrombosis in testicular blood vessels [220]. Regarding the presence of SARS-CoV-2 in semen, research results vary. Some studies suggest the virus is present in the semen of infected individuals [95, 97–99], while others have failed to detect it [221, 222]. Most studies have shown a decline in sperm quality [100] and impaired endocrine function in the testes following SARS-CoV-2 infection [101]. Due to the limited understanding of these emerging viruses, many drugs are currently being developed and evaluated to combat and mitigate the impact of SARS-CoV-2. Vaccination is considered a crucial pathway to help end this devastating pandemic. Most studies suggest that vaccination does not affect semen parameters [223] and can reduce the incidence of orchitis and/or epididymitis [224]. Overall, the current data seem to indicate that vaccination does not negatively impact fertility in either sex [224–226].

Adeno-associated virus

Adeno-associated virus (AAV) belongs to the *Parvoviridae* family and is a type of defective virus with simple structured single-stranded DNA. AAV requires helper viruses (such as adenovirus, herpesvirus, and HPV) to participate in replication, either establishing latent infections in the absence of helpers [227] or integrating into the chromosome DNA of the host [228].

In 1999, Rohde et al. first detected AAV DNA in semen samples of infertile men, which was associated with decreased sperm vitality [104]. Erles et al. found a higher detection rate of AAV DNA (38%) in semen samples of patients with semen abnormalities compared to those with normal semen (4.6%), and AAV DNA was also detected in testicular tissue, suggesting that AAV may contribute to male infertility by interfering with sperm development [102]. Several other studies have confirmed the presence of AAV DNA in semen samples but have not found a significant correlation with fertility [48, 103].

Zika virus

Zika virus (ZIKV) is a virus species with positive-sense single-stranded RNA and belongs to the *Flaviviridae* family, first discovered in 1954 [229]. Infection can lead to symptoms such as rash, fever, arthritis, Guillain-Barré syndrome (GBS), myelitis, meningitis, and congenital microcephaly [230]. ZIKV can be transmitted through mosquito bites, blood transfusions, breastfeeding, vertical transmission, and sexual contact [231, 232].

ZIKV was the first arthropod-borne virus detected in human semen [109]**.** Subsequent studies have confirmed the presence of ZIKV in semen [110, 232]. ZIKV RNA can persist in semen for more than six months in some infected males [111]. ZIKV infection can also cause local inflammation and tissue damage in the reproductive organs, resulting in symptoms such as hematospermia, ejaculatory pain, reduced sperm count, and abnormal secretion of reproductive hormones [112, 113]. ZIKV exhibits tropism for various cell types in the reproductive tract [233], and experimental studies have confirmed that ZIKV exhibits a preference for infecting cells within the testes, resulting in cell death and disruption of the seminiferous tubules, ultimately leading to severe testicular damage and infertility [234]. ZIKV infects testicular macrophages, triggering the upregulation of various inflammatory factors. Within seminiferous tubules, sertoli cells express high levels of the Axl receptor, facilitating ZIKV entry and replication. This infection induces the overexpression of antigen presentation genes, pro-inflammatory cytokines, and transcription factors, along with the release of inflammatory cytokines and chemokines. Inhibin-B, crucial for follicle-stimulating hormone (FSH) regulation, is downregulated by ZIKV, while ZIKV increases vascular cell adhesion molecule-1 (VCAM-1) expression, aiding immune cell adhesion. ZIKV can infect various cell types within the tubules but has a lower impact on interstitial cells. Testosterone production, primarily by interstitial cells, is also disrupted by ZIKV, affecting endocrine function [235, 236]. Sexual transmission of Zika virus and persistence of the virus in male reproductive tract (MRT) are the biggest challenges to outbreak control, vaccine and antiviral drug development. DNA-based vaccination and/or ZIKV attenuated live vaccines have shown high

efficacy against ZIKV-induced MRT injury in animal trials and may be an important tool for future prevention of ZIKV-induced male infertility [234]. Besides, the best way to prevent transmission is to abstain from sex or use protective measures against suspected infections [237].

Ebola virus

Ebola virus (EBOV) is a linear, non-segmented, single-stranded negative-sense RNA virus belonging to the *Filoviridae* family. Over 17,000 people survived the 2013–2016 Ebola virus disease outbreak in West Africa, which was the largest outbreak since the virus was first identified in 1976 [238]. This substantial number of survivors has enabled the implementation of several cross-sectional and longitudinal studies, leading to a better understanding of the long-term clinical sequelae in survivors and the persistence of the Ebola virus in biological fluids, particularly in semen [120, 121, 239].Studies have shown that the detection rate of EBOV RNA in the semen of Ebola survivors ranges from 8.1% to 9.8%, with viral shedding persisting for an extended period [240]. One cohort study found that viral RNA could remain in semen for up to 40 months [239]. Dyal et al. suggest that age, disease severity, and immune response may be risk factors for the prolonged presence of EBOV in semen [121]. Significant progress has been made in the treatment and prevention of Ebola virus disease. Currently, many vaccines and specific drugs targeting EBOV have been approved and are in use. However, there are still many issues to be addressed in reducing mortality, preventing viral escape, and improving safety [240].

Discussion

This study conducted a comprehensive and systematic analysis of the correlation between viral infections in male semen and infertility. It summarized 40 viruses found in human semen, with most viruses closely associated with male fertility. The main findings indicate that many viruses can be detected in semen, including those causing acute infections like ZIKV and EBOV, as well as those causing chronic infections like HPV and HIV. Some viruses, especially those showing strong tissue tropism for the male reproductive tract (such as SARS-CoV-2 and MuV), may spread through sexual transmission, which could be an important route of transmission. Viruses can potentially impact semen quality and fertility through various pathways, including direct infection of sperm or germ cells, disruption of testicular functions, induction of reproductive tract inflammation, and triggering immune responses.

Previous studies have investigated the impact of certain viral infections on male infertility. A meta-analysis indicated that individuals infected with HPV had decreased sperm motility, increased abnormal sperm morphology and DNA fragmentation index (DFI) compared to the uninfected group [241]. In contrast, our study included all types of viruses associated with male infertility, conducting a comprehensive analysis of the 40 viruses detected in semen, covering various virus families and potential transmission routes. This provided a comprehensive perspective on the relationship between male reproductive health and viral infections. We found the presence of some viruses, such as ZIKV and ZBOV, in the reproductive system of males and emphasized their potential impact on reproductive health, providing new directions for future research. In viral infections, changes in sperm and semen parameters can arise from (Fig. 1): (1) Genital tract inflammation: leukocyte infiltration and cytokine release. (2) Viral replication: viral replication in MGT cells alters their function and integrity, impacting sperm quality through various means like disrupted endocrine function and sperm DNA damage. (3) Systemic effects: acute infections raise testis temperature, hindering spermatogenesis. Chronic infections increase OS, harming sperm function. Altered hormone release from the pituitary reduces sperm counts by affecting testosterone and inhibin B secretion, crucial for spermatogenesis [11]. Among them, we found that OS is an important way to affect male fertility after virus infection. According to the literature, OS is responsible for 80% of male infertility cases [242]. While small amounts of reactive oxygen species (ROS) play crucial roles in sperm capacitation, acrosome reaction, and hyperactivation, excessive ROS production and OS can severely impair sperm structure and function. The sperm plasma membrane, which is rich in polyunsaturated fatty acids, is particularly susceptible to lipid peroxidation. Additionally, the sperm nuclear and mitochondrial epigenomes are vulnerable to elevated ROS levels, resulting in DNA damage through fragmentation, microdeletions, and mutations [243]. Consequently, OS induced by viral infections can degrade sperm quality, leading to reduced fertilization rates, poor embryo development, recurrent miscarriages, genetic mutations, and overall unfavorable outcomes in ART [242]. This highlights the importance of medical attention to these viruses and emphasizes the need for effective prevention and treatment strategies. Furthermore, protective measures can be developed, such as the use of antioxidants, to reduce the damage caused by oxidative stress in testicular tissues.

However, we must recognize that many viral infections in semen (such as HHV-6, JCPyV, and BKPyV, etc.) may remain asymptomatic or latent within the host and may not necessarily lead to impaired reproductive function. Conversely, some viruses (such as influenza viruses) may never be detected in the male reproductive

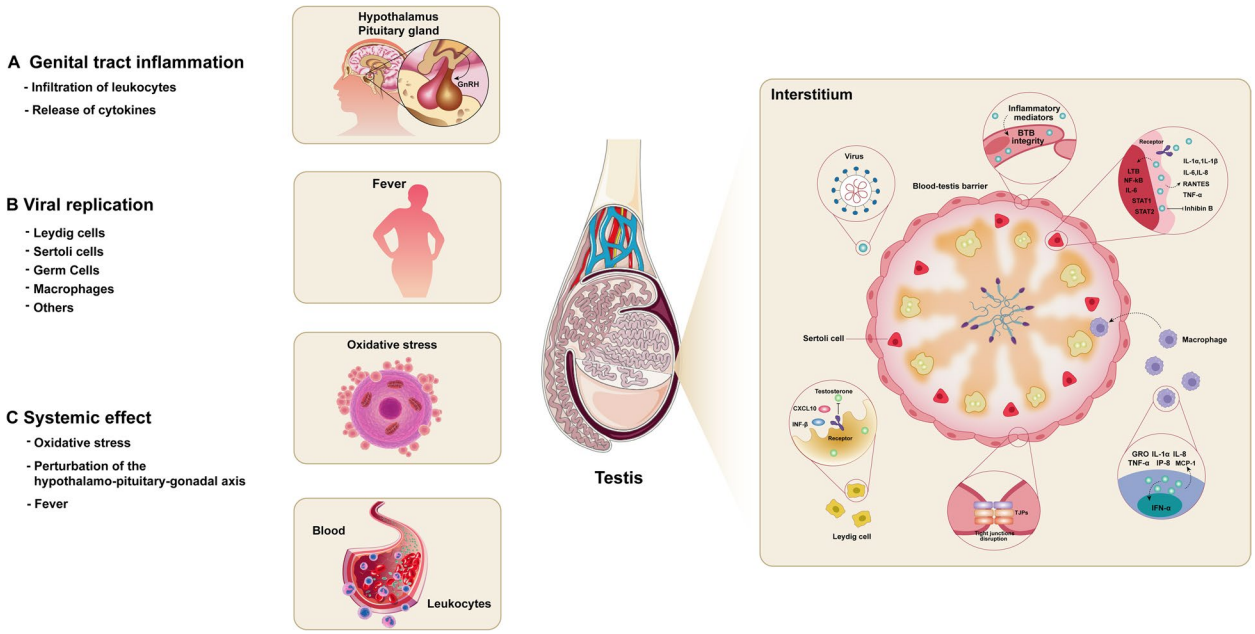


Fig. 1 The mechanism of seminal parameter change caused by viral infections. **A** Genital tract inflammation: Leukocyte infiltration leads to fibrosis in testicular tissues; Cytokines released during inflammation trigger germ cell apoptosis and disrupt cell functions. **B** Viral replication: Leydig cells: viral replication affects leydig cells, altering hormone production; Sertoli cells: viral infections disrupt sertoli cells, affecting the nourishment and development of germ cells; Germ cells: direct viral damage to germ cells impacts sperm quality and DNA integrity; Macrophages: viral interactions with these immune cells induce local inflammatory responses; Others: other testicular cell types may also be affected by viral infections. **C** Systemic effect: Oxidative stress: chronic infections increase oxidative stress, harming sperm function; Perturbation of the hypothalamo-pituitary–gonadal axis: viral infections disrupt this axis, altering hormone release and affecting testosterone and inhibin B secretion, crucial for spermatogenesis; Fever: acute infections induce fever, raising testis temperature and impairing spermatogenesis. These mechanisms collectively explain the multifaceted impact of viral infections on male fertility, demonstrating how local and systemic effects lead to significant changes in sperm and semen parameters

system [240] yet still cause apoptosis and decreased fertility due to fever or direct DNA damage [244]. Influenza virus infection can alter sperm morphology, reducing sperm count, motility, and ability to penetrate the egg [245, 246]. In addition, many infectious diseases are not caused by a single factor but rather by mixed infections involving multiple viruses, virus-bacteria co-infections, virus-fungus co-infections, or even virus-fungus-bacteria co-infections [81, 247–250]. Studies have indicated that lower viral diversity in semen samples is significantly positively correlated with successful pregnancy rates [81]. The microbiota is also highly associated with viral infections (such as HIV and HPV) [251, 252] and disease states. Therefore, considering viruses as the sole basis for disease is not entirely accurate; attention should be paid to the complex interactions in the microenvironment (including different pathogens, resident microbiota, and immune responses) and how they collectively affect reproductive function [253, 254]. Furthermore, the presence of viruses in semen may negatively impact the efficacy of ART [188, 241]. Viral infections in semen can also lead to congenital malformations and/or chronicity of viruses in fetuses [255–257]. Thus, even during natural conception, vertical transmission of viruses is a matter of concern [11].

Our study also has some limitations: it primarily relies on published literature and known data, which may overlook some viral infections or case reports. Although we summarized the presence of viruses in semen and their potential effects, the lack of support from large-scale clinical studies necessitates more experiments and clinical data to validate our findings.

Conclusion

Viral infections in males can potentially have detrimental effects on semen parameters and the efficacy of ART, serving as potential risk factors for male infertility. It is advisable for infertility patients, particularly those with abnormal semen parameters, to consider the potential impact of viral infections. Timely detection and treatment of viral infections are expected to enhance semen parameters and improve the success rate of ART. These insights offer valuable guidance for refining treatment strategies for male infertility patients in the future.

Abbreviations

COVID-19	Coronavirus disease 2019
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
HIV	Human immunodeficiency virus
ZIKV	Zika virus
EBOV	Ebola virus
CHIKV	Chikungunya virus
HPV	Human papillomavirus
HSV-1/2	Herpes simplex virus-1/2
VZV	Varicella zoster virus
EBV	Epstein-barr virus
CMV	Cytomegalovirus
HHV-6/7	Human herpesvirus-6/7
KSHV	Kaposi sarcoma-associated herpesvirus
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HDV	Hepatitis D virus
HEV	Hepatitis E virus
HGV	Hepatitis G virus
JCPyV	JC polyomavirus
MCPyV	Merkel cell polyomavirus
MWPyV	MW polyomavirus
BKPyV	BK polyomavirus
SV40	Simian virus 40
MuV	Mumps virus
AAV	Adeno-associated virus
MPXV	Monkeypox virus
HTLV	Human T-lymphotropic virus
WNV	West-Nile virus
DENV	Dengue virus
CV	Coxsackie virus
MARV	Marburg virus
RVFV	Rift valley fever virus
Ad	Adenovirus
CHAPV	Chapare virus
TOSV	Toscana virus
LASV	Lassa virus
SFTSV	Severe fever with thrombocytopenia syndrome virus
NiV	Nipah virus
YFV	Yellow fever virus
PMS	Progressive motile sperm
MNS	Morphologically normal sperm
pML	Progressive multifocal leukoencephalopathy
LF	Lassa fever
SFTS	Severe fever with thrombocytopenia syndrome
ASAs	Antisperm antibodies
PAP	Prostatic acid phosphatase
SEM	Seminalplasmin
SP	Seminal plasma
iciHHV-6	Inherited chromosomally integrated HHV-6
KS	Kaposi's sarcoma
BTB	Blood-testis barrier
PV	Polyomaviruses
AIDS	Acquired immunodeficiency syndrome
ACE2	Angiotensin-converting enzyme 2
TMPRSS2	Type II transmembrane serine protease
GBS	Guillain-barré syndrome
DFI	DNA fragmentation index
ART	Assisted reproductive therapies
FISH	Fluorescence in situ hybridization
ICSI	Intracytoplasmic sperm injection
IALu	Hyaluronidase
ESHRE	European Society of Human Reproduction and Embryology
OS	Oxidative stress
ROS	Reactive oxygen species
AQPs	Aquaporins
IL-6	Interleukin-6
TNF-α	Tumor necrosis factor-alpha
MCP-1	Monocyte chemoattractant protein-1
FSH	Follicle-stimulating hormone
VCAM-1	Vascular cell adhesion molecule-1
MRT	Male reproductive tract

Authors' contributions

YG and YYD performed the literature search, prepared the figures, and wrote the first draft; RZZ and JCY performed the literature search and data analysis; WYL and YX wrote the first draft; XLY, YMK and YTL supervised and reviewed the manuscript; LFX: developed the idea for the article, review and editing of the final manuscript. All authors contributed to the final version of the manuscript.

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Availability of data and materials

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The data used in this article were all sourced from published literature. Therefore, it is not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Reproductive Gynecology, NHC Key Laboratory of Healthy Birth and Birth Defect Prevention in Western China, the First People's Hospital of Yunnan Province, Kunming, Yunnan, China. [2]Department of Reproductive Medicine, NHC Key Laboratory of Healthy Birth and Birth Defect Prevention in Western China, the First People's Hospital of Yunnan Province, Kunming, Yunnan, China. [3]KUST-YPFPH Reproductive Medicine Joint Research Center, Medical school of Kunming University of Science and Technology, Kunming, Yunnan, China. [4]The Affiliated Hospital of Kunming University of Science and Technology, Kunming, Yunnan, China. [5]Faculty of Life Science and Technology, Kunming University of Science and Technology, Kunming, Yunnan, China. [6]Department of Clinical Laboratory, the First People's Hospital of Yunnan Province, Kunming, Yunnan, China.

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