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Current scientific perspectives on Mpox: a review of recent research

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Abstract: The review article presents the epidemiological, etiopathogenetic, clinical, and diagnostic aspects of monkeypox (MPOX), as well as possible measures for the prevention and treatment of this infectious disease. The causative agent, the monkeypox virus (MPXV), is transmitted in various ways: sexually, through household contact, and airborne droplets, which determines the clinical features of patients and a high mortality rate. Currently, a limited number of drugs are used that affect viruses of the Poxviridae family. Therefore, MPXV retains the status of a priority object for research in virology and epidemiology.

Keywords: mpox, monkeypox virus, MPXV, virus clade, skin rashes, DNA virus.

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Introduction. Mpox (MPOX) is an acute zoonotic infectious disease caused by the DNA-containing enveloped monkeypox virus (MPXV), which belongs to the genus *Orthopoxvirus* of the family *Poxviridae* [1]. In the Russian Federation, according to current sanitary regulations (SP 3.3686-21), the mpox pathogen is classified as a Pathogenicity Group I agent. It is noteworthy that this classification was established within the national regulatory framework as early as 1994 (SP 3.4.035-94), underscoring the consistently high priority of epidemiological surveillance for this infection in Russia [2]. The first officially confirmed case of human infection with mpox was recorded in 1970 in the Democratic Republic of the Congo (DRC); subsequently, for several decades, the disease remained primarily endemic to countries in Central and West Africa [3]. The first occurrence of the infection outside the African continent was reported in 2003 in the United States. This outbreak was linked to the importation of a shipment of infected Gambian pouched rats from Ghana, which, in pet store settings, infected local prairie dogs that became the direct source of infection for their owners. Between 2018 and 2021, sporadic imported cases of MPOX were documented in the United Kingdom, Israel, Singapore, and the USA. However, a radical shift in the global epidemiological landscape occurred in May 2022 with the onset of a large-scale outbreak in non-endemic countries across Europe and the Americas, characterized predominantly by human-to-human transmission. In July

2022, the World Health Organization (WHO) declared the mpox epidemic a Public Health Emergency of International Concern (PHEIC). In 2024, the situation escalated due to the spread of a new, more contagious variant—Clade Ib. Consequently, on August 14, 2024, the WHO re-declared a PHEIC, highlighting the global threat of the infection spreading beyond its epicenter in the DRC [4].

Structure of Viral Particles and the Monkeypox Virus Genome. The mpox virion has a brick-shaped or ovoid morphology with dimensions of 220–450 by 140–260 nm [5]. The viral supercapsid consists of a lipid bilayer with an embedded protein matrix, D13. The structural subunits of the capsid are assembled according to capsomeric principles [6]. From a pathogenetic perspective, the most significant envelope proteins are A26, A27, A29, H3, D8, and B5R. A27 proteins are involved in viral adhesion to host cells. The glycoprotein B5R, modified by lipid chains (palmitoylation), facilitates virus packaging and eases the spread of infectious units [7]. Within the nucleoprotein complex lies a biconcave or cylindrical core containing genomic viral DNA and proteins. The cylindrical (dumbbell-shaped) core of orthopoxviruses is represented by a single molecule of linear double-stranded DNA with a length of 130–375 kb, the ends of which are covalently closed into terminal hairpins, forming a continuous single-stranded structure at the termini [8]. Genes encoding replication enzymes E9, D4, D5, and A20, as well as transcriptional and structural proteins, are concentrat-

ed in the central part of the genome, as confirmed by orthopoxvirus architecture analysis [9]. A characteristic feature of the monkeypox virus is the presence of the specific modifying protein C3L, which blocks complement activation. This protein binds to C3b/C4b, preventing the lysis of infected cells, along with E3L [10]. The RNA-modifying enzyme VP39 is localized in the cytoplasm and associated with viroplasm. Its functions include acting as an RNA-cap-specific 2'-O-methyltransferase and a cofactor for the poly(A) polymerase VP55. Conformational stability of the virions is ensured by the A10L protein, while nucleoprotein dimerization is mediated by the I7L protein [8]. Contagiousness is determined by envelope proteins (A26L, H3L) that regulate cellular attachment [11].

Based on genomic nucleotide sequences, the monkeypox virus is classified into two genetic clades: Clade I (Central African) and Clade II (West African). Clade I is divided into Clade Ia (the endemic Congo strain) and Clade Ib (or Ib-MLC, "Mpox-Like Clade," the new highly contagious variant that triggered the 2024 emergency). Clade II includes IIa (historical West African variant, low lethality), IIb (the primary variant of the 2022–2023 outbreak), and IIb-C.1 (a subclade with the F105L mutation that dominated in Europe in 2022). Subclade IIa was identified in 2003 in the USA, while subclade IIb caused the outbreak in Nigeria in 2017–2019 [12]. In 2024, subclade Ib-MLC caused the global spread of the infection among the African population, while subclade IIb-C.1 continued

to circulate in countries across Europe, Asia, and the Americas [13].

Epidemiology. The monkeypox virus was first identified in 1958 in monkeys transported from Africa to Copenhagen (Denmark) for research purposes, which gave the disease its name [14]. However, rodents—specifically the rope squirrel (*Funisciurus pyrropus*) and the Gambian pouched rat (*Cricetomys gambianus*)—are considered the primary natural reservoirs in Central and West Africa [15]. According to epidemiological investigation data, the spread of the virus in the USA resulted from the importation of a shipment of African rodents from Ghana, including Gambian pouched rats (*Cricetomys gambianus*). These infected animals were kept in close contact with local prairie dogs, leading to interspecies transmission. Despite the animals' unhealthy appearance, characterized by conjunctivitis and tongue ulcerations, sales continued across the states of Wisconsin, Indiana, Illinois, Ohio, Kansas, and Missouri [16]. Animal-to-human transmission in this region occurred via the contact-household route through bites, scratches, and direct contact with contaminated biological fluids [17]. MPXV is transmitted from human to human through various routes: sexual, contact-household, and respiratory droplets. Sexual transmission occurs via infected biological fluids (semen, vaginal secretions). The contact-household route is realized through household items, clothing, bedding, and other objects contaminated with biological fluids (vesicle and pustule contents, saliva, sputum, urine, excrement); consequently, health-care workers and individuals living with infected persons constitute the primary high-risk groups [18].

Pathogenesis. The entry of the virion into the cell is a multi-stage process involving adhesion, hemifusion, and endocytosis or pinocytosis [19]. Primary replication occurs in lymphoid tissue, where target cells include T and B lymphocytes, macrophages, dendritic cells, and endothelial cells [20]. Replication of the monkeypox virus takes 6 to 13 days and occurs in the host cell cytoplasm, where specific complexes known as Guarnieri bodies are formed [21]. The monkeypox virus produces two distinct forms of infectious particles: intracellular mature virus (IMV), which plays a key role in transmission, and extracellular enveloped virus (EEV), which facilitates dissemination within body fluids. IMV enters the cell via macropinocytosis, while the enveloped virion (EEV) enters through membrane fusion with target cells [22]. In the early stages of replication, complex-

es of overlapping oligonucleotides—concatemers—are formed. Utilizing these, viral particles migrate to the internal cell membrane; upon fusion, they induce actin polymerization and the formation of filaments that assist the virion in exiting the cell [23]. Secondary replication occurs in regional lymph nodes, to which the virus migrates via lymphogenous dissemination. Subsequently, the virus enters the systemic circulation, causing a stage of generalized viremia that affects lung tissue, the parenchyma of the liver and kidneys, the ovaries, the pancreas, and the gray matter of the brain and spinal cord [24].

Following viral entry, the first line of immune defense is activated, involving macrophages and dendritic cells. These not only identify viral antigens through pattern recognition receptors (membrane-bound TLRs 1-11, intracellular NLRs, and RLRs) but also produce cytokines (IL-6, IL-12, IFN- γ) and chemokines (CXCL 8, CXCL 2) that recruit other immune cells to the site of inflammation. Viral replication is limited by Type I interferons (IFN- α/β) [25]. Antigen-presenting cells phagocytose viral particles and display their peptides on the surface of Major Histocompatibility Complex Class II (MHC II), which activates the differentiation of CD4+ T cells (Treg, Th1, Th2). T-regulatory cells (Treg) control the intensity and duration of the immune response. T-helper 1 (Th1) cells stimulate cell-mediated immunity and contribute to hypercytokinemia. T-helper 2 (Th2) cells, interacting with B lymphocytes, facilitate the activation of humoral immunity and the production of specific IgM and IgG [26]. The monkeypox virus possesses mechanisms for immune evasion (inhibition of IFN- α/β synthesis, blocking of antigen presentation, and induction of apoptosis in immunocompetent cells), which allows it to persist in the human body for extended periods [27].

Clinical Manifestations. There are four periods of the clinical course of mpox. The incubation period depends on the virus clade and transmission route, lasting from 4 to 21 days (averaging 5 to 13 days), and corresponds to the primary replication of the virus in lymphoid tissue [28]. The prodromal period lasts from 1 to 4 days. Initial manifestations of the disease are non-specific and may include malaise, chills, headache, dizziness, myalgia, and arthralgia [29]. A specific manifestation of the prodromal period is lymphadenopathy (involvement of cervical, inguinal, and axillary lymph nodes). After 24–48 hours, involvement of the mucous membranes of the genitals, anus,

and oral cavity appears in the form of enanthema [30]. The period of cutaneous eruptions lasts from 14 to 28 days and is characterized by the appearance of exanthema accompanied by itching and soreness [31]. Clade II is characterized by localized lesions in the genital area, perianal zone, and lower extremities, while Clade I is associated with generalized skin lesions. In most cases, eruptions are localized on the face and also affect the palms of the hands and soles of the feet, appearing on the trunk in rare cases [32]. The reconvalescence period depends on the severity of the course and the state of the immune system, lasting from 2 to 4 weeks and usually ending favorably [33]. Local complications, limited to the perineal region, include proctitis, paraproctitis, pararectal fistulas, edema of the penis and scrotum, and balanoposthitis. Systemic complications manifest as the development of secondary bacterial skin infections, primarily caused by *S. aureus*, *S. pyogenes*, *E. coli*, *P. aeruginosa*, and *Cl. perfringens* [34].

Diagnosis. The period of active virus spread to non-endemic countries from 2018 to 2022 became pivotal for the development and market release of mpox rapid diagnostic panels. In laboratory differential diagnosis of the mpox pathogen, nucleic acid amplification testing (NAAT) using real-time PCR is employed. Samples for testing include venous blood, swabs and scrapings from damaged skin areas, crusts taken from more than one lesion site, or exudate [35]. One of the leading research institutions involved in developing rapid mpox diagnostic methods during this period was the State Research Center of Virology and Biotechnology "VECTOR" in the Russian Federation. The "Vector-MPCRv-Ospa" test system includes a species-specific primer and a fluorescently labeled probe for diagnosing the DNA nucleotide sequence of the monkeypox virus [36]. In the same year, a similar test system, the RealStar® Orthopoxvirus PCR Kit 1.0, was developed in Germany by Altona Diagnostics [37]. In 2022, some of the first diagnostic panels patented on the global market included the RealTime® Monkeypox Virus PCR Kit (Jiangsu BioPerfectus Technologies Co., Ltd., China) and Viasure® RealTime PCR Detection Kits (Jant Pharmacal Corporation, USA). However, the highest demand was seen for the GeneXpert® Mpox test (Cepheid, USA), distinguished by its reliability and high result speed, and the LightMix® Modular Orthopox/Monkeypox Virus Kits (TIB MOLBIOL GmbH, Germany), which possess high sensitivity and specificity

for clades I and II of the monkeypox virus [38]. In October 2024, the World Health Organization (WHO) included the Alinity® m MPXV AMP Kit (Abbott Molecular Inc., USA) and the GeneXpert® Mpx test (Cepheid, USA) in the list of medical products approved for use in emergency situations [39]. A promising direction in mpox diagnostics is sequencing, which allows for the detection of virus mutations during an epidemic. Currently, isothermal sequencing methods (for example, based on Phi29 polymerase and "multiple displacement amplification" – MDA technology) are being actively developed to improve the coverage and accuracy of MPXV genome analysis, including clades I and II. These approaches are considered promising for accelerated genomic epidemiology [40]. Specific IgG and IgM against individual surface proteins, including the A29L protein of the monkeypox virus, can be detected 5–8 days after infection using enzyme-linked immunosorbent assay (ELISA) with the Monkeypox Virus (MPXV) IgG ELISA Kit® (Elabscience, USA) [41].

Treatment. To date, a limited number of medicinal products acting against the genus *Orthopoxvirus* are utilized. Cidofovir® was used as an antiviral drug until 2017 in endemic countries. This medication acts by inhibiting viral DNA polymerase, thereby hindering viral replication. Brincidofovir® is a modified version of this drug, possessing a reduced spectrum of side effects on the human body [42]. The drug of choice for the past several years has been Tecovirimat® (Tecovirimat, ST-246, TPOXX), an antiviral agent that inhibits the activity of the envelope protein p37 (F13L/VP37). By blocking p37 function, Tecovirimat® prevents its interaction with cellular factors (including Rab9 and TIP47), which disrupts the wrapping of intracellular mature virions and their release, thus reducing intracellular and intercellular viral spread [43]. In the Russian Federation, NIOCH-14—a direct domestic analog of tecovirimat—has been developed and registered, created by specialists from the State Research Center of Virology and Biotechnology "Vector" and the N.N. Vorozhtsov Novosibirsk Institute of Organic Chemistry. Possessing an identical mechanism of action, NIOCH-14 effectively blocks the exit of viral particles from infected cells, allowing it to be considered a key element of the national biosafety system. NIOCH-14 successfully passed clinical trials and received a registration certificate in the RF in 2022 [44]. Monoclonal antibodies (mAbs) are being studied as potential therapeutic strategies for

mpox. In 2022, Wenlong Zhu and Mengjia Zhang (China), utilizing hybridoma technologies, developed specific monoclonal antibodies 1E12, 4B2, and α-A29-MPXV against the A29L protein [45]. In 2023, Jennifer A. Melendez, Haiyan Sun, James Bonner, Qiang Chen, and co-authors (USA) discovered monoclonal antibodies synthesized by glycoengineered plants targeting extracellular enveloped virions (EEV) of the monkeypox virus [46]. Additionally, symptomatic therapy is used to alleviate symptoms and support the overall condition of the infected individual [47].

Prevention. The World Health Organization (WHO) recommends targeted vaccination against mpox among high-risk groups, including contacts [48]. Although mass specific vaccination against MPXV was limited in non-endemic countries until 2022, population protection was provided by three generations of vaccines based on the vaccinia virus: Generation I (dermal): live vaccines based on Lister strains (Lister-Elstree or Elstree), such as the live smallpox vaccine (L-IVP) (JSC NPO Microgen, Russia); live vaccines based on NYCBH strains (New York City Board of Health), such as the live attenuated vaccine Dryvax® (Wyeth Laboratories, USA); live vaccines based on the "Heavenly Temple" (Tiantan) strain, such as the smallpox vaccine based on the Tiantan strain (Beijing Institute of Biological Products, China); live vaccines based on the Paris strain, such as the smallpox vaccine (Institut Pasteur, France); live vaccines based on the Ankara strain (CVA), such as the live smallpox vaccine (Turkish Vaccine Institute, Turkey); and live vaccines based on the Dairen strain (Dairen-I), such as the live smallpox vaccine (Chiba Serum Institute and National Institute of Infectious Diseases, Japan). Generation II (cell-culture based): vaccines based on cell cultures (Vero cell line: African green monkey kidney cells), such as the replication-competent adapted live vaccine ACAM2000® (Acambis, USA); and vaccines based on cell cultures (primary chicken embryo fibroblasts – CEF), such as the replication-competent live vaccine Elstree-BN® (Bavarian Nordic, Denmark, Germany). During the international emergency in September 2022, the WHO approved the third-generation live attenuated, non-replicating vaccine based on the Ankara strain, MVA-BN® (Imvanex/Jynneos, Denmark), which became the first in the world approved for use against mpox. In November of the same year, the live attenuated, replication-competent third-generation vaccine based on

the Lister-Elstree strain—LC16m8® (KM Biologics Co., Ltd., Japan)—was added to the list of medical products approved for use in emergency situations [49]. In the Russian Federation today, the following smallpox vaccines are registered and ready for use: live smallpox vaccine, L-IVP strain (JSC NPO Microgen); dry inactivated smallpox vaccine—Tevrivak® (JSC NPO Microgen); dry live embryonic smallpox vaccine—TEOpak® (JSC NPO Microgen); fourth-generation recombinant vaccine—Orthopoxvac® (FBUN SRC VB "Vector" of Rospotrebnadzor); and live oral smallpox vaccine (FSBI "48th Central Research Institute" of the Ministry of Defense of Russia / JSC NPO Microgen) [50]. Smallpox vaccines were also successfully used: the live attenuated vaccine ACAM2000™ and the recombinant vaccine based on a weakened virus—JYNNEOS™ [51]. During the international emergency in September 2022, the WHO approved the MVA-BN™ vaccine (Imvanex/Jynneos, Denmark), which became the first in the world approved for use against mpox. In November of that year, the LC16m8™ vaccine (KM Biologics, Japan) was added to the list of medical products approved for emergency use [52]. For emergency prophylaxis and the treatment of vaccination complications in individuals immunized against mpox, human anti-smallpox immunoglobulin has been developed (JSC NPO Microgen, Russia), along with its foreign analog VIGIV (Cangene Corporation, USA) [53, 54].

Conclusion. Mpox, as a highly dangerous viral disease, is a significant subject of study in the fields of virology and infectious diseases. Virus clades I and II, identified through scientific research, highlight its genetic variability, high virulence, and contagiousness. The need for global monitoring becomes particularly urgent in light of recent outbreaks. Sustainable cooperation between scientists, medical institutions, and research institutes will allow for the development of effective specific prevention methods to help prevent the spread of the monkeypox virus.

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Predictors of HBV-associated hepatocarcinoma: a scientific review

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Abstract. The urgency of the problem of chronic hepatitis B (CHB) is caused by the annual detection of a large number of new cases of infection and a significant number of patients with a long-term course of the disease which can lead to serious complications, including cirrhosis of the liver and hepatocellular carcinoma (HCC).

The purpose of this work was to analyze and systematize the data of modern scientific literature on predictors of HCC development in chronic hepatitis B.

Studies show that the most significant risk factors for developing HCC are old age, male gender, genetic predisposition, presence of metabolic syndrome, liver fibrosis, smoking, alcohol consumption, exposure to hepatotropic toxins and environmental pollution. Of particular importance is the identification of mutant forms of hepatitis B virus, such as A1762T/G1764A, E64D, E77Q, and A80I/T which increase the risk of developing HCC.

Despite significant progress in the diagnosis and treatment of CHB, the problem of prevention and early diagnosis of HCC remains relevant. The development of universal and accessible diagnostic models that make it possible to identify patients at increased risk of developing HCC in a timely manner is a priority task for improving the quality of medical care and improving the effectiveness of combating this serious disease.

Conclusions. Thus, the study confirms the importance of further studying the mechanisms of HCC development and introducing new approaches to the treatment and prevention of hepatitis B complications.

Keywords: Hepatitis B, HBV, chronic hepatitis, replicative activity, liver fibrosis, liver cirrhosis, hepatocellular carcinoma, predictors, Republic of Sakha (Yakutia).

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Relevance. Despite significant advances in the prevention, diagnosis and treatment of infectious diseases, about 1.2 million new cases of hepatitis B infection are detected annually in the

world and the total number of patients with HBV infection has exceeded 250 million people. In the absence of therapy, chronic viral hepatitis often leads to cirrhosis of the liver (LC) and hepatocel-

lular carcinoma (HCC), from which, in turn, more than 1 million people die annually [1].

The purpose of the work: to analyze and systematize the data of modern sci-