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Review Article

Molecular epidemiology and genomic surveillance of human pegivirus: current insights and future directions

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ABSTRACT

Human pegivirus (HPgV), formerly termed GB virus C (GBV-C), is a widely circulating, non-pathogenic RNA virus within the Flaviviridae family. Although highly prevalent globally, HPgV remains insufficiently characterized despite growing evidence of its immunomodulatory effects, particularly among individuals co-infected with HIV or HCV. Improved understanding of its genetic diversity, transmission patterns, and evolutionary dynamics is crucial to elucidate its potential biomedical relevance. The contemporary evidence on the molecular epidemiology and genomic surveillance of HPgV, emphasizing research gaps, geographic disparities, and priorities for future study. A systematic peer-reviewed literature and publicly available genomic repositories such as GenBank and GISAID was conducted for studies published between 1995 and 2025 using terms including "HPgV," "GBV-C," "molecular epidemiology," "genomic surveillance," and "genotype distribution." Extracted data were classified by region, genotype, and study design to evaluate trends in prevalence, genetic variability, sequencing coverage, and the strength of surveillance systems. The review reveals substantial global genetic diversity, comprising at least seven recognized genotypes with distinct regional distributions. Despite diversity, HPgV remains underrepresented in genomic studies, particularly in low- and middle-income countries. Limitations include scarce full-genome sequencing, inconsistent genotype classification, and limited integration into global pathogen surveillance platforms. While co-infection with HIV and HCV poses analytical challenges, it also offers opportunities to investigate HPgV-associated immune modulation. Although clinically benign, HPgV's widespread distribution and possible immunological influence highlight the need for enhanced genomic monitoring, standardized nomenclature, and expanded sequencing capacity. Coordinated global efforts are essential to strengthen HPgV surveillance and advance understanding of its evolutionary behavior and public health implications.

Keywords: Human pegivirus, Molecular epidemiology, Genomic surveillance, Flaviviridae, Viral evolution

INTRODUCTION

Human pegivirus (HPgV), which was once referred to as GB virus C or hepatitis G virus, is an RNA virus that can be found all over the globe, belonging to the Flaviviridae family. Since it was first identified in the mid-1990s, HPgV has caught the attention of researchers because of its widespread presence, remarkable genetic diversity, and its interesting effects on the immune system, especially in people who are also infected with HIV.[1] Unlike many other viruses in the Flaviviridae family, HPgV is generally seen as non-pathogenic, meaning it doesn't directly cause liver disease. This makes it a fascinating subject for studies in molecular epidemiology and virology. This review aims to

give a thorough global perspective on the molecular epidemiology and genomic monitoring of HPgV, focusing on what we currently know about its genetic diversity, evolutionary trends, transmission methods, and clinical importance. We also point out the gaps in global monitoring, the challenges of standardization, and the future paths for research and public health collaboration.

TAXONOMY AND GENOMIC ORGANIZATION

HPgV is classified under the genus pegivirus within the Flaviviridae family. It shares some structural characteristics with other flaviviruses, including a single-stranded positive-sense RNA genome that is about 9.3 kb long. This genome contains a single open reading frame (ORF) that is translated into a polyprotein, which is then cleaved into structural proteins.[2] and nonstructural proteins (NS2, NS3, NS4A, NS4B, NS5A, NS5B).[3] The virus has various genotypes and subtypes that are spread across different geographical areas, reflecting the evolutionary pressures and history of transmission. Currently, at least seven distinct genotypes (1-7) have been identified, with genotypes 1 and 2 being common in Africa and the Americas, while genotypes 3 to 7 are primarily found in Asia and other regions.[4]

HPGV MOLECULAR BIOLOGY AND GENOMICS

Genomic structure

HPgV has a genome that's about 9.4 kb long and made up of RNA. It features a single open reading frame (ORF) that's flanked by untranslated regions (UTRs) on both the 5′ and 3′ ends. This ORF codes for a polyprotein, which is then cleaved into structural proteins (E1, E2) and non-structural proteins (NS2–NS5B) after translation. While HPgV's genomic layout is like that of the hepatitis C virus (HCV), it doesn't carry the same pathogenic risks associated with its relatives in the Flaviviridae family.

Genotypes and genetic diversity

So far, researchers have identified seven main genotypes of HPgV (from HPgV-1 to HPgV-7), each with different regional distributions; HPgV-1: mostly found in North America and Europe, HPgV-2: primarily seen in West Africa, HPgV-3: common in Asia and HPgV-4-7: found in specific areas; more studies are needed to understand their distribution and clinical relevance.

HPgV shows considerable sequence variability, particularly in the E2 envelope protein region, which plays a role in immune evasion and potential immune-modulation.

Mechanisms of evolution

HPgV has a high mutation rate due to its error-prone RNA-dependent RNA polymerase, leading to significant genetic diversity. There's also evidence of recombination events and the dynamics of intra-host quasispecies, which make phylogenetic classification more complex. These evolutionary traits highlight the need for genomic surveillance to monitor transmission patterns and identify new variants.

Comparison with related flaviviruses

HPgV is closely related to other pegiviruses and shares some structural features with HCV. However, unlike HCV, HPgV doesn't lead to hepatitis or chronic liver disease. Its relatively harmless nature has prompted researchers to explore its potential positive effects, particularly in the context of HIV co-infection, where it has been linked to slower disease progression.

Global molecular epidemiology of pegivirus

The global landscape of HPgV infection is quite extensive, with its prevalence differing across various populations and regions. Studies on seroprevalence show that as many as 20% of the general population and a staggering 70% of high-risk groups—like intravenous drug users and those co-infected with HIV and HCV show signs of current or past infection.[5]

Molecular epidemiological research, which often involves partial sequencing of conserved regions such as the 5'UTR and E2, has uncovered unique genotype distributions that align with geographical locations and modes of transmission. For example, genotype 1 is predominantly found in West Africa, while genotype 2 is more common in North America and Europe. Meanwhile, genotypes 3 to 5 have mainly been identified in Asia.[6] These findings hint at a historical spread of the virus that corresponds with human migration and trade routes.

HPgV shows a wide global reach, infecting various populations with significant differences in prevalence and genotype distribution. Studies in molecular epidemiology have highlighted intricate patterns influenced by geography, the characteristics of host populations, and the routes of transmission.[7]

PREVALENCE AND POPULATION DISTRIBUTION

When it comes to prevalence and population distribution, both serological and molecular screening studies have consistently found HPgV infections in healthy individuals as well as those at higher risk. In the general population, prevalence rates can vary dramatically, ranging from 1% to 20%, depending on the region and the diagnostic methods employed.[8] For instance in West Africa, prevalence often surpasses 15% among healthy individuals.[9] In Europe and North America, prevalence rates for blood donors typically fall between 1% and 5%.[10] In Asia, prevalence can be quite variable, often around 5% to 10%, with certain hotspots linked to intravenous drug use and high-risk sexual behavior.[11] Among high-risk

groups—including intravenous drug users, patients co-infected with HIV or HCV, and those receiving blood products—HPgV RNA prevalence can soar to as high as 70% (Table 1).[12]

Genotypic diversity and geographic distribution

HPgV showcases a significant amount of genetic variation, which is categorized into seven main genotypes (1-7) through phylogenetic analysis of partial E2 or NS5B gene sequences.[13] These genotypes have unique yet overlapping geographic distributions (Table 2).

The widespread distribution and overlapping presence of different genotypes hint at a historical spread of the virus through human migration and trade routes, which has been further complicated by modern travel and blood transfusion practices.[14,15]

Phylogenetic and evolutionary insight

When we look at phylogenetic and evolutionary insights, the analyses show that HPgV genotypes form strong clusters with high bootstrap support, pointing to well-defined genetic lineages. Studies using molecular clocks suggest that the common ancestor of today's HPgV genotypes appeared several centuries ago, with a substitution rate comparable to other RNA viruses, around $1–2\times10^{-3}$ substitutions per site per year.[16]

There are also genotype-specific evolutionary patterns, such as varying rates of amino acid substitution in envelope proteins. These variations might reflect how the virus adapts to the immune responses of its hosts and differences in how efficiently it spreads. For instance, genotype 2 isolates show more genetic conservation compared to genotype 3 strains, which could be tied to their specific epidemiological niches.[14]

Transmission clusters and network analysis

In terms of transmission clusters and network analysis, advanced molecular epidemiology techniques, including phylogeography and network analysis, have started to shed light on transmission clusters within certain populations. For example; intravenous drug user groups in Europe display tight genotype 2 clusters that align with recent local transmission events.[6] Blood donor screening programs have uncovered occasional multi-genotype infections, indicating co-circulation and dynamics of superinfection.[10] In Asia, molecular tracing has revealed that genotype 3 is spreading along drug trafficking routes (Table 3).[11]

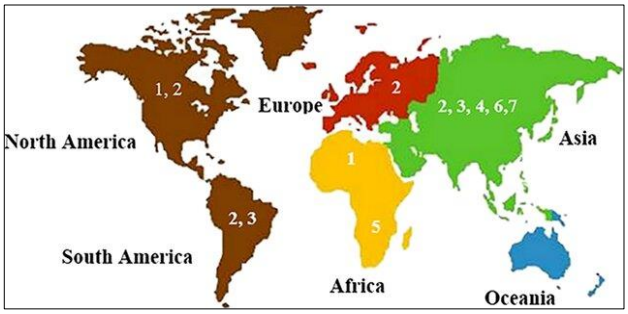

Figure 1: Geographical distribution of different HPgV-1 genotypes.

Table 1: Summary of HPgV prevalence studies by region and population.

Region	Population type	Sample size	Prevalence (%)	Genotypes detected	Reference
West Africa	General population	500	18	1	13
Europe	Blood donors	1000	3	2	2
Asia	Iv drug users	300	45	3,4	6
North America	HIV co-infected	250	55	2	11
South Africa	General population	200	10	5	16

Table 2: Geographical distribution and genotypes.

Genotype	Geographic distribution	Notes
1	West and Central Africa	Most common in West Africa; prevalent in blood donors and general population
2	North America, Europe	Widely distributed in Western countries
3	Asia (Japan, China, Southeast Asia)	Frequently isolated in Asia; possible link to local transmission networks
4	Southeast Asia (Vietnam, Thailand)	Less common; regional clusters reported
5	South Africa	Detected mainly in southern Africa
6	Indonesia and parts of Asia	Regionally localized genotype
7	Recently described in China	Emerging genotype with limited data

Table 3: Molecular characteristics of HPgV genotypes.

Genotype	Geographic region	Key genetic markers (e.g., 5'UTR, E2 variations)	Known recombination events	Estimated evolutionary rate (substitutions/site/year)
1	West Africa	Specific nucleotide polymorphisms in E2	Rare	~1×10^{-3}
2	Europe, N. America	Distinct 5'UTR sequence variants	Occasional	~5×10^{-4}
3	Asia	Unique E2 glycoprotein motifs	Not reported	~8×10^{-4}
4	SE Asia	Limited data	Unknown	Unknown
5	Central Africa	Partial sequencing data available	Unknown	Unknown
6	Central Africa	Limited data	Unknown	Unknown
7	SE Asia	Limited data	Unknown	Unknown

These insights underscore the importance of combining molecular and epidemiological data to guide targeted public health interventions.

The limited data available from many low- and middle-income countries (LMICs) means that the diversity of HPgV in these areas is often overlooked, which in turn affects our global understanding of the virus's epidemiology.

GENOMIC SURVEILLANCE APPROACHES

When it comes to genomic surveillance, the initial detection of HPgV typically relies on molecular assays, especially reverse transcription polymerase chain reaction (RT-PCR). This method targets conserved regions of the 5′ untranslated regions (UTR) or the NS5B gene. While serologic assays that look for anti-E2 antibodies can show past exposure, they fall short in identifying current viremia, which is crucial for genomic surveillance and studies on transmission.[11]

Next-generation sequencing (NGS) has really broadened the horizons of HPgV research. It allows for whole-genome sequencing from clinical samples, helps identify new genotypes and recombination events, and even enables metagenomic profiling in both human and non-human hosts.[4]

Particularly, metagenomic NGS (mNGS) has been instrumental in revealing HPgV diversity in asymptomatic individuals and environmental reservoirs.[7] However, it's important to note that the global distribution of sequencing capabilities is quite uneven. Most of the HPgV genomes we have so far come from high-income countries, which limits our understanding of the virus's diversity in less represented regions.

Proposed categorization of GB viruses. Maximum likelihood tree (Tamura 3-parameter nucleotide model and gamma distribution, T92 + G) depicting phylogenetic relationship of Human Pegiviruses with other main members of Flaviviridea family. The phylogenetic tree was constructed based on the complete coding sequence (CDS) of these viruses. The values at the tree branches are the bootstrap support values calculated from 1000 replicates. Scale bar indicates an evolutionary distance of 0.5 substitutions per position in the sequence. GB virus type A, B, C, D (GBV-A, GBV-B, GBV-C, GBV-D), simian Pegivirus (SPgV), human Pegivirus (HPgV), Bat Pegivirus (BPgV), CPZ: chimpanzees (Figure 3).

Bioinformatic and phylogenetic tool

On the bioinformatics side, advancements have made it easier to analyze HPgV genetic data effectively. Commonly used tools include PhyML and RAxML for maximum likelihood phylogenetic reconstruction, BEAST for Bayesian evolutionary analysis and molecular clock modeling, and SimPlot and RDP4 for spotting recombination events.[16] HPgV genotyping usually focuses on partial sequences from the E2 or NS5B regions, but there's no universal agreement on a standardized framework for genotyping. This lack of consensus has resulted in inconsistencies in classification and has made it tough to compare findings across different studies.[16,11]

Public databases and data sharing

When it comes to public databases and data sharing, the NCBI GenBank database is the main hub for HPgV sequences, but submissions tend to be irregular and often biased by region. Unlike HIV and SARS-CoV-2, HPgV doesn't have the advantage of a centralized, curated global surveillance platform (like GISAID), which makes coordinated epidemiological tracking a challenge.

Recently, there have been efforts to implement findable, accessible, interoperable, reusable (FAIR) data principles in virology, highlighting the importance of better metadata reporting. This includes details like host demographics, coinfection status, and geographic location, all of which help to provide context for sequence data.[14]

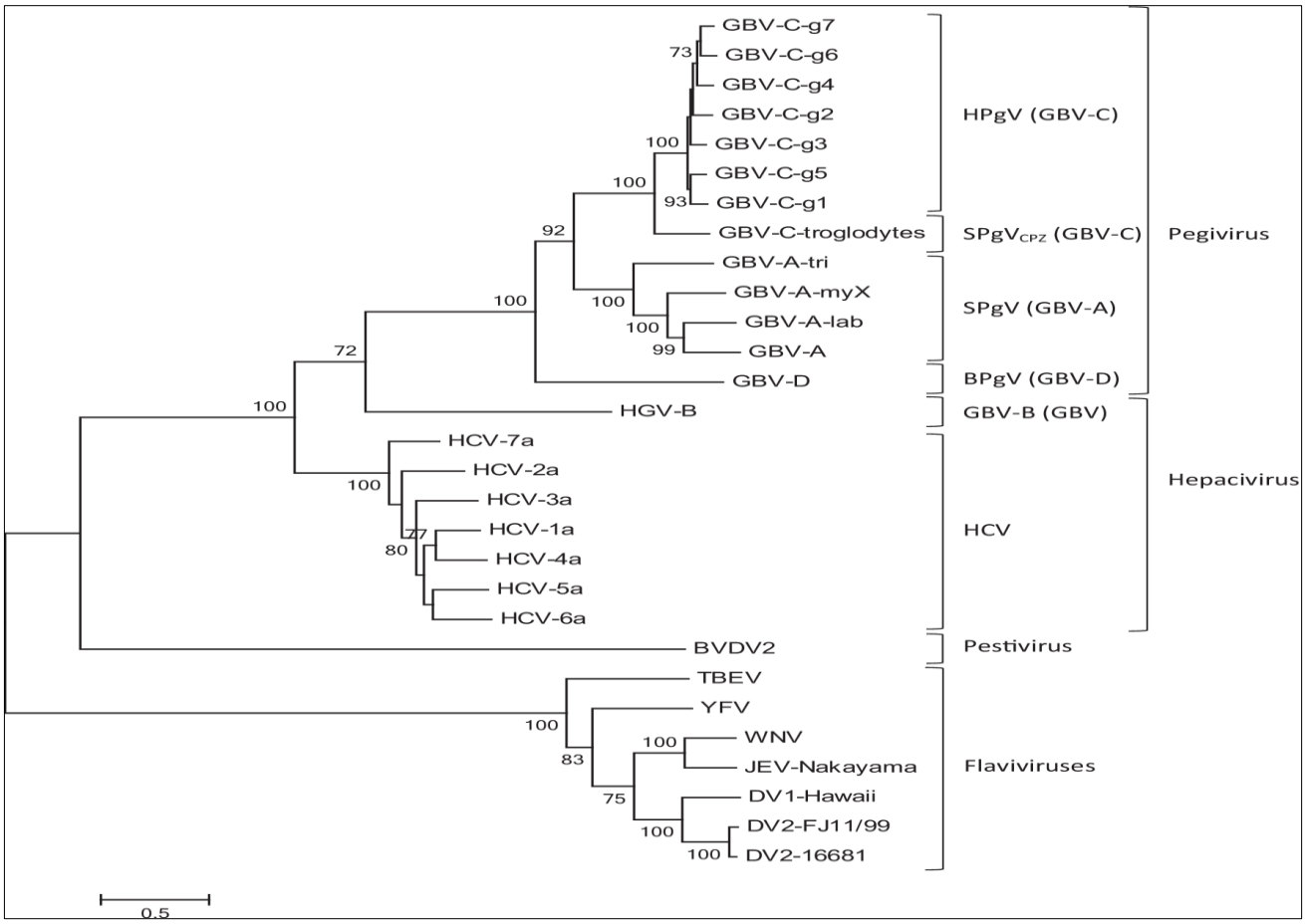

Figure 2: Phylogenetic tree.

EVOLUTIONARY DYNAMICS AND TRANSMISSION PATTERNS

Molecular evolution and quasi species diversity

Looking at evolutionary dynamics and transmission patterns, HPgV shows a significant level of genetic variability, mainly due to the error-prone nature of its RNA-dependent RNA polymerase. Like other RNA viruses, HPgV exists as a quasi species—a mix of related but genetically distinct viral genomes within a single host.[11] This genetic diversity allows the virus to quickly adapt to the host's immune responses, helping it persist without causing overt disease.

Longitudinal studies have demonstrated that viral populations within a host evolve over time, especially in the hypervariable regions of the E2 envelope protein, which faces immune pressure.[1,4] These evolutionary dynamics are reminiscent of those seen in hepatitis C virus (HCV), though HPgV tends to have a more benign clinical profile.

Recombination events

Recombination between different HPgV strains have been observed and play a crucial role in the virus's evolution. This process can affect viral fitness, help the virus evade the immune system, and influence genotype classification.[14]

Recombination hotspots have been spotted in the E2 and NS5 regions, but we still have a lot to learn about how this affects viral epidemiology. To detect recombination, we rely on full-genome sequencing and advanced bioinformatics techniques, like bootscanning and programs such as RDP4 and SimPlot. Unfortunately, the limited availability of sequences, particularly in low- and middle-income countries (LMICs), has hindered our ability to fully understand the global landscape of recombinant HPgV lineages.[16]

Transmission pathway

When it comes to transmission pathways, HPgV mainly spreads through parenteral routes, including blood transfusions, injection drug use, and organ transplants. There have also been reports of sexual and vertical transmission, but these methods are generally less effective.[9,8]

Coinfections with HIV, HCV, and HBV are quite common, as these viruses share similar transmission routes. Interestingly, HPgV coinfection seems to influence the

progression of HIV, possibly by reducing immune activation markers and inhibiting HIV replication.[8,13] These effects are particularly noticeable when HPgV viremia is persistent.

Moreover, HPgV has been found in non-human primates, hinting at the possibility of cross-species transmission and a more complex viral ecology.[14]

Clinical implications and immunological effects of HPgV

Now, let's talk about the clinical implications and immunological effects of HPgV. Generally, Human Pegivirus is viewed as a non-pathogenic virus, with no direct link to liver disease or other significant health issues.[12] Even though it's widely spread, HPgV rarely leads to symptoms, and its clinical importance has often been overlooked.

Immunomodulatory effects in HIV coinfection

Numerous studies have shown that HPgV can influence the progression of HIV infection. Persistent HPgV viremia has been linked to a slower progression to AIDS reduced viral loads, increased CD4+ T-cell counts, and lower immune activation levels.[1,4,6,12] It suggests that HPgV may interfere with HIV entry receptors, modulate cytokine profiles, and induce antiviral chemokines like RANTES and MIP-1β.[7,11] However, we still haven't seen clinical trials that take advantage of these potential benefits.

Potential effects on another viral coinfection

When it comes to other viral coinfections, the role of HPgV alongside HCV, HBV, and others is still a bit murky. Some research hints at possible positive effects similar to those seen in HIV coinfection, while other studies show no significant impact on disease progression.[3] Clearly, we need more research to clarify these connections.

Implications for blood safety and transfusion medicine

In terms of blood safety and transfusion medicine, HPgV poses a potential challenge due to its high prevalence among blood donors and the risk of transmission through transfusions. Although routine screening isn't currently in place, the risk of transfusion-transmitted infections is something we need to consider, especially for those who are immunocompromised.[2]

CHALLENGES AND FUTURE DIRECTIONS

Challenges

Limited global surveillance infrastructure

There's a notable absence of structured and coordinated HPgV surveillance programs, especially in low- and middle-income countries.

The fact that HPgV infections are often asymptomatic means they tend to be overlooked in public health discussions.

Scarcity of full-genome sequences

Most studies on HPgV focus on partial regions (like E2 or 5'UTR), leading to a shortage of complete genome sequences.

This limitation makes it difficult to conduct thorough phylogenetic and evolutionary studies that are essential for understanding transmission dynamics.

Underrepresentation in genomic databases

Public databases, such as GenBank, contain relatively few HPgV sequences compared to other viruses like HCV or HIV.

Additionally, many studies are geographically biased, with a disproportionate number coming from high-income countries.

Inconsistent naming and classification

The differences in naming conventions (like HPgV-1 versus GBV-C) and the lack of consistent genotype classifications make it tough to conduct comparative epidemiological studies.

The complexity of co-infections

HPgV often shows up alongside viruses such as HIV, HCV, or HBV, which complicates our ability to separate its unique epidemiological patterns.

There's also ongoing debate about its effects on the immune system, especially regarding HIV progression.

Challenges with technology and analysis

Limited access to next-generation sequencing (NGS) tools and bioinformatics resources hampers real-time genomic monitoring. Technical hurdles, such as low viral loads and sequence variability, make it difficult to detect and sequence HPgV.

Lack of funding and research focus

HPgV isn't seen as a priority pathogen, which restricts funding opportunities and collaborative research efforts.

Future directions

Enhancing global genomic surveillance

We should integrate HPgV into current viral surveillance systems like GISAID, GLOPID-R, or the WHO's GISRS.

It's important to promote active surveillance in underrepresented areas, including sub-Saharan Africa, Southeast Asia, and Latin America.

Creating standardized genotyping guidelines

Let's establish consensus guidelines for HPgV genotyping, similar to those for HIV-1 or HCV.

We should encourage the collection of standardized metadata, such as age, geography, and co-infection status.

Advancements in sequencing technology

We can utilize portable and affordable platforms (like Oxford Nanopore) for on-the-ground surveillance.

Metagenomic approaches should be applied to discover new HPgV strains and variants.

Longitudinal and cohort research

Investing in prospective cohort studies will help us better understand HPgV transmission, persistence, and the effects of co-infections.

We need to explore vertical transmission and pediatric epidemiology, which are still not well understood.

Interdisciplinary and one health strategies

Let's investigate the zoonotic origins or animal reservoirs of HPgV using One Health frameworks.

Let's work together across different fields like virology, immunology, and epidemiology to get a complete picture of the situation.

Functional genomics and host interaction studies

We need to clarify how HPgV influences the immune responses of hosts, particularly in populations co-infected with HIV.

By utilizing CRISPR screening, transcriptomics, and proteomics, we can pinpoint the host factors that contribute to HPgV persistence.

Open-access data sharing and collaboration

It's essential to motivate international groups to share sequence data and analytical tools freely. We can take advantage of platforms like Nextstrain to keep track of HPgV evolution in real-time.

Gaps in molecular surveillance

Even with the progress in sequencing technologies, HPgV is still not well-studied in many regions, especially in LMICs. The limited access to sequencing tools and a lack of epidemiological data hinder our global understanding of HPgV diversity and how it spreads.[16]

Standardization of genotyping and nomenclature

The absence of a universally accepted genotyping system makes it tough to compare findings across different studies and regions. We urgently need international efforts to create standardized molecular markers and classification criteria.[11]

Understanding clinical impact

While we know that HPgV has a beneficial effect in HIV co-infection, we still need to explore its potential roles in other diseases and its long-term health effects. Large-scale prospective studies and in-depth research into host-virus interactions should be top priorities.[12]

Integrating HPgV surveillance into public health programs

Currently, HPgV isn't routinely screened in blood banks or public health monitoring. By incorporating HPgV genomic surveillance into existing viral hepatitis and HIV programs, we could enhance detection and gain valuable epidemiological insights.[2]

Potential for therapeutic and vaccine development

Given its ability to modulate the immune system, HPgV or its components could lead to innovative therapeutic strategies for HIV and other immune-related diseases. Translating these observations into clinical interventions is still a major hurdle.[4]

CONCLUSION

Human pegivirus remains a globally prevalent yet understudied member of the *Flaviviridae* family, characterized by broad genetic diversity and intriguing immunological effects, particularly in the context of HIV co-infection. This review consolidates existing molecular, epidemiological, and genomic evidence to provide a clearer and more integrated understanding of HPgV's evolutionary dynamics, genotype distributions, and potential clinical relevance. By synthesizing disparate findings across regions and research disciplines, this work advances current knowledge by highlighting the substantial gaps in genomic data, the lack of standardized genotyping systems, and the underrepresentation of low- and middle-income countries in global datasets.

Importantly, our analysis underscores that HPgV offers a unique model for studying virus–host interactions due to its non-pathogenic nature and measurable immunomodulatory effects. Recognizing this potential reframes HPgV from a neglected viral entity to a biologically informative system that may yield insights

relevant to HIV pathogenesis, immune regulation, and viral evolution.

Looking forward, scaling up global genomic surveillance, harmonizing classification frameworks, and deepening research on HPgV's immunological mechanisms are essential next steps. Integrating HPgV monitoring into broader pathogen surveillance platforms will not only improve our epidemiological understanding but also create opportunities to explore its translational potential in immunotherapy and HIV management. By identifying these research priorities and synthesizing the current landscape, this review provides a foundational roadmap to guide future scientific inquiry into this overlooked but scientifically valuable virus.

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REFERENCES

1. Alter HJ, Prati D, Muerhoff AS, et al. A multicenter study of the prevalence of GB virus C/hepatitis G virus among US blood donors. N Engl J Med. 2002;336(12):747-54.
2. Bhattarai N, Stapleton JT. GB virus C: the good boy virus? Trends Microbiol. 2012;20(3):124-30.
3. Greninger AL, DeRisi JL, DeSalle R, et al. Metagenomic sequencing as a diagnostic tool for infectious diseases: a primer for physicians. JAMA. 2017;318(13):1271-2.
4. Heringlake S, Ockenga J, Tillmann HL, Trautwein C, Meissner D, Stoll M, et al. GB virus C/hepatitis G virus infection: a favorable prognostic factor in human immunodeficiency virus-infected patients? J Infect Dis. 1998;177(6):1723-6.
5. Muerhoff AS, Smith DB, Leary TP, Erker JC, Desai SM, Mushahwar IK. Identification of GB virus C variants by phylogenetic analysis of 5'-untranslated and coding region sequences. J Virol. 1997;71(9):6501-8.
6. Niel C, Chevalier M, Devaney J, et al. High diversity and evolutionary dynamics of GB virus C/hepatitis G virus in HIV-infected patients. J Virol. 2004;78(1):259-67.
7. Simons JN, Pilot-Matias TJ, Leary TP, Dawson GJ, Desai SM, Schlauder GG, et al. Identification of two flavivirus-like genomes in the GB hepatitis agent. Proc Natl Acad Sci U S A. 1995;92(8):3401-5.
8. Sibley SD, Pinto AK, Babady NE, et al. Metagenomic investigation of plasma samples from febrile patients reveals diverse microbial sequences including a novel GB virus C genotype. PLoS One. 2014;9(5):e96436.
9. Smith DB, Bukh J, Kuiken C, Muerhoff AS, Rice CM, Stapleton JT, et al. Expanded classification of hepatitis C virus into 7 genotypes and 67 subtypes: updated criteria and genotype assignment web resource. Hepatology. 2014;59(1):318-27.
10. Stapleton JT, Foung S, Muerhoff AS, et al. The GB viruses: a review and proposed classification of GBV-A, GBV-C, and GBV-D in genus Pegivirus within the family Flaviviridae. J Gen Virol. 2011;92(Pt 2):233-46.
11. Tacke F, Blessing K, Janssen RW, et al. GB virus C/hepatitis G virus infection in patients with chronic hepatitis C: evidence for hepatotropism and viral persistence in the liver. J Hepatol. 2003;39(3):442-8.
12. Tillmann HL, Heiken H, Knapik-Botor A, Heringlake S, Ockenga J, Wilber JC, et al. Infection with GB virus C and reduced mortality among HIV-infected patients. N Engl J Med. 2001;345(10):715-24.
13. Viazov S, Blum H, Timm J, et al. Transmission of hepatitis G virus via blood transfusion in immunocompromised patients. Lancet. 1999;353(9161):2165-9.
14. Wilkinson MD, Dumontier M, Aalbersberg IJ, Appleton G, Axton M, Baak A, et al. The FAIR Guiding Principles for scientific data management and stewardship. Sci Data. 2016;3:160018.
15. Williams CF, Klinzman D, Yamashita TE, Xiang J, Polgreen PM, Rinaldo C, et al Persistent GB virus C infection and survival in HIV-infected men. N Engl J Med. 2004;350(10):981-90.
16. Xiang J, Wünschmann S, Diekema DJ, Klinzman D, Patrick KD, George SL, et al. Effect of coinfection with GB virus C on survival among patients with HIV infection. N Engl J Med. 2001;345(10):707-14.

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