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# Global Prevalence of Human Pegivirus-1 (HPgV-1) Infection among HIV-Positive Patients and the Impact of HPgV-1 on HIV Viral Load and CD4 Cell Count: A Systematic Review and Meta-Analysis

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**ABSTRACT**

**Background:** Human pegivirus-1 (HPgV-1) was first identified in 1996. The impact of HPgV-1 on HIV viral load and CD4 cell count is controversial. This study aimed to assess the global prevalence of HPgV-1/HIV coinfection and the impact of HPgV-1 on HIV viral load and CD4 cell count.

**Materials & Methods:** A literature search was performed in PubMed, Scopus, EMBASE, Web of Science, ProQuest, Cochrane, and Global Index Medicus (GIM) up to January 2026. The $I^2$ statistic and Cochran's Q test were used to assess heterogeneity. Subgroup analyses were carried out to find potential causes of heterogeneity. Funnel plot and Egger's test were used to assess publication bias.

**Findings:** A total of 86 studies with 19050 HIV-positive cases, published from 1997 to 2023 and covering 36 different countries, were included in this meta-analysis. The overall prevalence of HPgV-1 among HIV-positive patients was 25.3%. The prevalence of HIV/ HPgV-1 coinfection in different continents was as follows: 26.5% in Africa, 21.9% in Asia, 26.7% in Europe, 24.6% in North America, and 25.1% in South America. The overall mean of CD4 cell count was significantly different between HPgV-1-positive and HPgV-1-negative patients, while the overall mean of HIV viral load was not significantly different.

**Conclusion:** In this meta-analysis, the global prevalence of HPgV-1/HIV coinfection was 25.3%, with coinfected patients showing higher CD4 cell counts ($p$= .004) but no impact on HIV viral load. The results suggest that HPgV-1 may modulate HIV progression, though mechanisms remain unclear. Larger longitudinal studies are needed to confirm clinical significance and explore genotype-specific effects.

**Keywords:** CD4 lymphocyte count, Human pegivirus-1, Prevalence, HIV, Viral load

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## Introduction

Human pegivirus-1 (HPgV-1) is an enveloped, single-stranded RNA virus classified in the genus *Pegivirus* and the family *Flaviviridae* [1]. This virus was first identified in 1996 and named GB-C virus [2]. The association of HPgV-1-1 with hepatitis disease remains unknown, but the virus is a rare cause of hepatic inflammation. Most immunocompetent healthy persons clear HPgV-1-1 viraemia within 2 years of HPgV-1 infection, but infection may persist for years in some individuals [3]. There is no significant evidence indicating that HPgV-1-1 is associated with chronic liver disease [4].

HPgV-1 has seven genotypes with distinct geographical distributions as follows: genotype 1 in Japan, USA, and West Africa; genotype 2 in East Africa, USA, and Europe; genotype 3 in Asia; genotype 4 in Vietnam, Australia, and Myanmar; genotype 5 in Japan and South America; genotype 6 in southeast Asia and Indonesia; and genotype 7 in China and Qatar [5].

HPgV-1 is widespread across the world. It is estimated that one-sixth of the world's population is HPgV-1-1 seropositive, and around 750 million people have HPgV-1-1 RNA. The prevalence of HPgV-1-1 varies from 0.8 to 44.6% among the general population but from 1.8 to 75.3% among at-risk populations [2, 6]. Because HPgV-1 is easily transmitted through percutaneous injuries and blood transfusion, a significant prevalence of this viral infection is detected among healthy blood donors [7]. Intravenous drug users (IDUs) are the major high-risk group for this viral infection and show a high HPgV-1-1 prevalence. HPgV-1-1 may spread through sexual contact and vertical mother-child transmission. Men who have sex with men (MSM) and commercial sex workers (CSWs) are also high-risk populations for HPgV-1-1 infection. Because HPgV-1-1 has the same transmission route as HBV, HCV, and HIV, a high prevalence of HPgV-1 co-infection (3.2-47.9%) is frequently recorded in patients who are positive for the mentioned viruses [6].

Different studies have shown that approximately 5-47.9% of HIV-positive patients are co-infected with HPgV-1-1. Some of these studies have reported that HIV/HPgV-1-1 co-infected patients have slower AIDS progression and prolonged survival [8, 9]. Both HIV viral load and CD4 + T cell count are important indicators of AIDS disease progression. Some studies have reported that HPgV-1 viral load is positively correlated with $CD4^+$ T cell count and negatively correlated with HIV viral load. These results suggest that HPgV-1-1 infection may have a beneficial effect on HIV/AIDS [10-12]. There are studies with conflicting results in this field because some researchers have not find a significant relationship between the presence of HPgV-1-1 in HIV-positive patients and its beneficial effect on reducing HIV viral load and increasing CD4+ T cell count [13-15]. Therefore, it is necessary to conduct a meta-analysis study that combines the findings of independent studies and determines whether HPgV-1-1 slows AIDS progression. In addition, although many studies have reported HPgV-1-1/HIV coinfection in different countries, a comprehensive study on the global prevalence of HPgV-1-1 among HIV-positive patients has not yet been carried out.

**Objectives:** Therefore, this systematic review and meta-analysis was conducted to assess the global prevalence of HPgV-1-1/HIV coinfection and the impact of HPgV-1-1 on HIV viral load and CD4 cell count.

## Materials and Methods

This systematic review and meta-analysis was conducted according to the guidelines of the preferred reporting items for systematic reviews and meta-analyses (PRISMA). The study protocol was registered in PROSPERO

with code CRD42020178233.

**Search strategy:** A comprehensive search for published literature was conducted in electronic databases, including PubMed, Scopus, EMBASE, Web of Science, ProQuest, Cochrane, and Global Index Medicus (GIM). In addition, Google Scholar was used as a secondary source. The search included all manuscripts published from the earliest possible start date to January 2026 without language restrictions. The search strategies were made using the following keywords: "hepatitis G virus" OR "HPgV-1-1", "GB virus C" OR "GBV-C" OR "Pegivirus", and "HIV" OR "AIDS" OR "human immunodeficiency virus". Two reviewers evaluated the reference lists of the articles to find additional relevant manuscripts that might have been missed during the initial literature search.

**Study selection based on inclusion and exclusion criteria:** After removing duplicate citations, two reviewers independently assessed the remaining publications to find studies that matched the inclusion criteria. Eligible studies were selected in two stages. First, the title and abstract of each manuscript was reviewed to exclude articles that did not meet the inclusion criteria. Second, the full text of the remaining articles was reviewed, and those meeting the inclusion criteria were selected**.** To determine the global prevalence of HIV/HPgV-1-1 coinfection, all original articles that reported the prevalence rates of HPgV-1-1 among HIV-positive patients were included, and review articles were excluded. To determine the impact of HPgV-1-1 on HIV viral load and CD4 cell count, only studies that compared HIV viral load and CD4 cell count between HIV/HPgV-1-1 coinfected patients and HIV patients were selected. For the prevalence meta-analysis, no restriction was applied regarding ART (antiretroviral therapy) use. For the meta-analyses of HIV viral load and CD4 cell count, only studies explicitly reporting ART-naïve status were included; studies involving ART-treated or mixed/unknown populations were excluded from these analyses. Articles whose samples showed HIV co-infection with other viruses were excluded.

**Data extraction:** The following variables were extracted from the selected studies: first author; publication year; country; study design; sample size; number of patients with HIV/HPgV-1-1 coinfection; diagnostic method; patient age, sex, CD4 cell count, and HIV viral load; HPgV-1 genotypes; and use of antiretrovirals. Two reviewers collected data in extraction forms, and any disagreements were resolved by the decision of a third independent reviewer.

**Evaluation of study quality and risk of bias:** The methodological quality and risk of bias of all studies included in this review were assessed using the Joanna Briggs Institute (JBI) critical appraisal checklist for studies reporting prevalence data [3]. This tool evaluated the quality of studies using nine questions about sample frame, sample size, study setting, data analysis, validation, measurement, and response rate. Each question was answered with yes, no, unclear, or not applicable. The ratio of the number of yes responses to the total responses was calculated as a percentage and considered as the final quality score for each study. Scores below 60% were considered to be low-quality, while scores above 80% were considered to be high-quality. Low-quality studies were excluded from the meta-analysis. Two independent reviewers evaluated the quality of the studies, and disagreements were resolved by the decision of a third reviewer.

**Performing meta-analysis:** In the present review study, Comprehensive Meta-Analysis software Version 3.0 (CMA, Bio Stat, Englewood, CO, USA) was used to perform meta-analyses. Event rates, 95% confidence intervals (CI), and *p*-values were determined

for all studies. Study heterogeneity was assessed by Cochran's Q test and the $I^2$ index. For Cochran's Q test, $p < .10$ was considered significant heterogeneity. If heterogeneity was not obtained, a fixed effects model was used. If the $I^2$ statistic result was over 50% with a $p$-value < .05, heterogeneity was considered high, and a random effects model was performed. To explore potential sources of heterogeneity, subgroup analyses were performed by continent, type of study, year of publication, use of antiviral therapy, and risk factor group. Sensitivity analysis was carried out to identify whether excluding high-risk studies significantly changed the effect estimate. Publication bias was evaluated by funnel plots, and statistical significance was calculated using Egger and Begg's tests. A $p$-value of less than 0.05 was considered statistically significant. To assess the robustness of the findings, sensitivity analyses were performed by sequentially excluding each study and re-analyzing the pooled effect size.

## Findings

**Characteristics of the selected studies:** The results of the literature search and the study selection process are shown in Figure 1. A total of 1788 studies were collected through literature search. After removing duplicate and ineligible abstracts, the full-text of 109 articles was reviewed. A total of 95 studies met the inclusion criteria for further analysis. Table 1 presents the basic characteristics of the selected studies. Among them, 68 studies were cross-sectional, and 27 studies were designed as cohorts. The selected studies were published from 1997 to 2023 and performed in 36 different countries. The number of studies conducted in different continents was as follows:22 studies in Asia, 31 studies in Europe, 17 studies in North America, 13 studies in South America, and 12 studies in Africa. The total number of HIV-positive patients coinfected with HPgV-1 was 14976, with a range of 19 to 1406 per study. HPgV-1 genotypes were reported in 17 studies among a total of 1036 HPgV-1/HIV coinfected patients. The most common HPgV-1 genotype reported in the studies was HPgV-1-2 (48.65%), followed by HPgV-1-3 (25.68%), HPgV-1-7 (11.40%), HPgV-1-4 (5.30%), HPgV-1-1 (4.93%), HPgV-1-5 (1.44%), and HPgV-1-6 (2.60%).

**Prevalence of HPgV-1 in HIV-positive patients:** The overall prevalence of HPgV-1 among HIV-positive patients in 86 studies was 25.3% (95% CI 23.6–27%) (Figure 2). A significant heterogenicity was observed in prevalence estimates ($I^2$=88.9%; $Q$-statistic=763.9, $p < .0001$). The HPgV-1 prevalence was estimated to be 26.5% (95% CI 21.4–32.3%) in Africa, 21.9% (95% CI 18–26.3%) in Asia, 26.7% (95% CI 23.8–29.8%) in Europe, 24.6% (95% CI 19.6–30.4%) in North America, and 25.1% (95% CI 22.3–28.3%) in South America. Subgroup analyses (Table 2) revealed that the pooled prevalence of hepatitis G virus did not differ significantly by continent ($Q$= 3.57, $df$= 4, $p$= 0.467), study design ($Q$= 0.02, $df$= 2, $p$= .991), or reported antiviral drug use ($Q$= 0.20, $df$= 2, $p$= .904). However, significant subgroup differences were observed for year of publication ($Q$= 6.91, $df$= 2, $p$= .032), with the highest prevalence in studies published after 2010 (28.1%; 95% CI 24.9–31.6) and the lowest prevalence in those published from 2000–2010 (22.1%; 19.3–25.2). A marked difference was also found across risk factor groups ($Q$= 13.55, $df$= 4, $p$= .009): the prevalence was the highest among people who injected drugs (29.3%; 24.0–35.3) and the lowest among transfusion recipients (16.1%; 9.1–26.7) and heterosexual individuals (18.0%; 14.7–22.0).

In meta-regression analyses (Table 3), the sample size did not affect the heterogeneity of the studies ($p$= .40), but publication year

**Table 1)** Basic characteristics of the selected studies

| Autor/Publish Year | Country | Continent | Study Design | Study Period | Diagnostic Method | ART status | Sample Size | HGV Prevalence (%) | | | Age of Patients with HIV/ HGV (year) | HGV Genotypes (N) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Total | Male | Female | | |
| Fiordalisi (1997) | Italy | Europe | Cross-sectional | NR | RT-PCR | NR | 93 | 32/3 | NR | NR | NR | NR |
| Heringlake (1997) | Germany | Europe | Cross-sectional | 1993-1994 | RT-PCR | NR | 197 | 16/7 | NR | NR | NR | NR |
| Takao (1997) | Japan | Asia | Cross-sectional | NR | RT-PCR | NR | 111 | 20 | NR | NR | NR | NR |
| Bonacini (1998) | USA | North America | Cross-sectional | NR | RT-PCR | NR | 157 | 22/3 | NR | NR | 36±6 | NR |
| Iba´n˜ez (1998) | Spain | Europe | Cross-sectional | 1993-1997 | RT-PCR | NR | 168 | 18 | NR | NR | NR | NR |
| Quiros (1998) | Spain | Europe | Cross-sectional | NR | RT-PCR | NR | 30 | 36/3 | NR | NR | NR | NR |
| Nerurkar (1998) | USA | North America | Cross-sectional | 1994-1997 | RT-PCR | NR | 209 | 17/7 | 19/85 | 13/70 | NR | NR |
| de Martino (1998) | Italy | Europe | Cross-sectional | NR | RT-PCR, ELISA | Yes | 58 | 20/7 (RT-PCR), 34/5 (ELISA) | 0/00 | 20/70 | 29.5 (23-35) | NR |
| Clevenberg (1998) | France | Europe | Cross-sectional | 1997 | RT-PCR, ELISA | Yes | 100 | 4 (RT-PCR), 31 (ELISA) | 3/66 | 5/56 | NR | NR |
| Woolley (1998) | USA | North America | Cross-sectional | NR | RT-PCR | NR | 192 | 23 | 21/82 | 29/63 | 37/70 | NR |
| Toyoda (1998) | Japan | Asia | Cross-sectional | NR | RT-PCR | NR | 41 | 27/5 | 27/50 | 0/00 | 13/00 | NR |
| Oubin˜a (1999) | Argentina | South America | Cross-sectional | NR | RT-PCR | NR | 70 | 30 | NR | NR | NR | G1=21, G2a =11, G2b=4, G3 =5 |
| Lefre`re (1999) | France | Europe | Cohort | 1985–1991 | RT-PCR | Yes | 95 | 24/2 | 24/32 | 23/81 | 29.75±5.9 | NR |
| Bourlet (1999) | France | Europe | Cross-sectional | 1993-1997 | RT-PCR, ELISA | NR | 80 | 31 (RT-PCR), 40 (ELISA) | NR | NR | NR | NR |
| Lau (1999) | USA | North America | Clinical trial | 1988-1991 | RT-PCR | Yes | 180 | 36/6 | 37/43 | 22/22 | 32 (22–52) | NR |
| Lefihre (1999) | Farance | Europe | Cross-sectional | NR | RT-PCR, ELISA | NR | 105 | 24/7 (RT-PCR),16/2 (ELISA) | NR | NR | NR | NR |
| Goubau (1999) | Belgium | Europe | Cross-sectional | NR | RT-PCR, ELISA | NR | 138 | 21/7 (RT-PCR), 14/5 (ELISA) | NR | NR | NR | NR |
| Rey (1999) | France | Europe | Cross-sectional | 1996-1997 | RT-PCR | NR | 379 | 37/7 | NR | NR | NR | NR |
| Dámaso (2000) | Spain | Europe | cross-sectional | 1998-1999 | RT-PCR, ELISA | Yes | 19 | 10/5 (RT-PCR), 79 (ELISA) | NR | NR | NR | NR |
| Puing-Basagoiti (2000) | Spain | Europe | Cross-sectional | 1993-1997 | RT-PCR | NR | 160 | 17/5 | NR | NR | NR | NR |
| E.T. Yeo (2000) | USA | North America | Cross-sectional | 1982-1996 | RT-PCR, ELISA | Yes | 131 | 14/5 (RT-PCR),29 (ELISA) | NR | NR | NR | NR |
| XIANG (2001) | USA | North America | Cross-sectional | 1988-1999 | RT-PCR | Yes | 362 | 39/7 | 39/81 | 39/53 | 34/50 | NR |
| Tillmann (2001) | Germany | Europe | Cross-sectional | 1993-1994 | RT-PCR, ELISA | Yes | 197 | 16/7 (RT-PCR), 56/8 (ELISA) | 13/07 | 29/55 | 34.0±8.8 | NR |

| Autor/ Publish Year | Country | Continent | Study Design | Study Period | Diagnostic Method | ART status | Sample Size | HGV Prevalence (%) | | | Age of Patients with HIV/ HGV (year) | HGV Genotypes (N) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Total | Male | Female | | |
| Pontali (2001) | Italy | Europe | Cohort | 1998 -1999 | RT-PCR | NR | 49 | 0 | 0/00 | 0/00 | NR | NR |
| Markus (2002) | Sweden | Europe | Cohort | 1995 | RT-PCR | Yes | 157 | 23 | 20/69 | 29/27 | NR | NR |
| Voirin (2002) | France | Europe | Cohort | 1995-1999 | RT-PCR | Yes | 105 | 32/4 | 32/22 | 40/00 | 31.88±2.62 | NR |
| Rodriguez (2003) | USA | North America | Cohort | 1995-1997 | RT-PCR | No | 146 | 21/2 (RT-PCR), 22/6 (ELISA) | 20/47 | 26/32 | 39.32 ± 8.3 | NR |
| López Calvo (2003) | Italy | Europe | Cross-sectional | NR | RT-PCR | Yes | 222 | 28/8 | 34/38 | 31/25 | 33.2±6.7 | NR |
| Nunnari (2003) | Italy | Europe | Cross-sectional | 1989 | RT-PCR | Yes | 80 | 21 | 21/15 | 21/43 | 24.2± 1.6 | NR |
| Chakraborty (2003) | Kenya | Africa | Cross-sectional | 2000 | RT-PCR | Yes | 60 | 5 | NR | NR | NR | NR |
| Shieh (2003) | Taiwan | Asia | Cross-sectional | 1996–2000 | RT-PCR | No | 62 | 11/7 | 11/76 | 0/00 | 42/00 | NR |
| Weintrob (2004) | Tanzania | Africa | Cohort | 1992–1993 | RT-PCR, ELISA | No | 186 | 24 (RT-PCR), 298 (ELISA) | 0/00 | 24/00 | NR | NR |
| Ramia (2004) | Lebanon | Asia | Cross-sectional | NR | RT-PCR | Yes | 118 | 10/1 (RT-PCR), 30/5 (ELISA) | NR | NR | NR | NR |
| Barqasho (2004) | Sweden | Europe | Cohort | 1987-1994 | RT-PCR, ELISA | Yes | 52 | 5/7 (RT-PCR), 27 (ELISA) | 0/00 | 5/77 | NR | NR |
| Sathar (2004) | South Africa | Africa | Cross-sectional | 1995-1998 | RT-PCR | No | 75 | 36 | 0/00 | 36/00 | NR | NR |
| Williams (2004) | USA | North America | Cohort | 1984-1990 | RT-PCR | Yes | 271 | 39/4 | 39/48 | 0/00 | NR | NR |
| Antonucci (2005) | Italy | Europe | Cohort | 2003 | RT-PCR | No | 400 | 29/3 | 29/81 | 27/27 | 35 (31–41) | NR |
| Van der Bij (2005) | Netherlands; | Europe | Cohort | 1986-1996 | RT-PCR, ELISA | Yes | 326 | 42 (RT-PCR), 41 (ELISA) | NR | NR | NR | NR |
| Smith (2005) | USA | North America | Cross-sectional | 2004 | RT-PCR, ELISA | Yes | 351 | 23/2 (RT-PCR), 32/3 (ELISA) | 25/00 | 20/69 | 44.7 ± 7.9 | NR |
| Kaye (2005) | Gambia | Africa | Cohort | NR | RT-PCR | NR | 250 | 19/2 | 0/00 | 19/20 | NR | NR |
| Aster (2005) | Czech | Europe | Cross-sectional | 2002-2003 | RT-PCR, ELISA | NR | 273 | 33/3 (RT-PCR), 38/5 (ELISA) | NR | NR | NR | NR |
| Gime´nez-Barcons (2005) | Spain | Europe | Cross-sectional | 1994 -1996 | RT-PCR, ELISA | Yes | 161 | 27 (RT-PCR), 21 (ELISA) | 28/79 | 20/69 | 35.8±11 | NR |
| Schuval (2005) | USA | North America | Cross-sectional | 2002-2003 | RT-PCR | Yes | 353 | 5/7 (RT-PCR), 3/4 (ELISA) | 4/65 | 6/63 | 12.95±1.11 | NR |
| Sall (2006) | Cambodia | Asia | Cross-sectional | 2003 | RT-PCR | NR | 213 | 16/4 | 17/78 | 14/10 | NR | NR |

| Autor/Publish Year | Country | Continent | Study Design | Study Period | Diagnostic Method | ART status | Sample Size | HGV Prevalence (%) | | | Age of Patients with HIV/ HGV (year) | HGV Genotypes (N) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Total | Male | Female | | |
| Li (2006) | Ghana | Africa | Cross-sectional | NR | RT-PCR, ELISA | NR | 515 | 24 (RT-PCR), 6/9 (ELISA) | NR | NR | NR | NR |
| Descamps (2006) | France | Europe | Cohort | NR | RT-PCR, ELISA | Yes | 183 | 23/5 (RT-PCR), 24 (ELISA) | 23/08 | 24/24 | 36.6 (29.0-42.2) | NR |
| Souza (2006) | Brazil | South America | Cohort | 1995-1997 | RT-PCR | No | 175 | 24 | 28/46 | 13/46 | 33.23±8.11 | NR |
| Ryt-Hansen (2006) | Denmark | Europe | Cohort | 1984-1987 | RT-PCR | Yes | 112 | 24 | 24/00 | 0/00 | NR | NR |
| Mosam (2007) | Africa | Africa | Cohort | NR | RT-PCR | No | 38 | 23/7 | 25/00 | 22/22 | 32.25±3.19 | NR |
| Denks (2007) | Estonia | Europe | Cross-sectional | 2005-2006 | RT-PCR | Yes | 95 | 35 | 40 | 23 | NR | NR |
| Handelsman (2007) | USA | North America | Cross-sectional | 1990-2002 | RT-PCR, ELISA | Yes | 397 | 10/8 (RT-PCR), 35/5 (ELISA) | 0/00 | 10/83 | NR | NR |
| Sheng (2007) | Taiwan | Asia | Cross-sectional | 1999-2004 | RT-PCR | Yes | 385 | 12/2 | NR | NR | 39.97±10.93 | NR |
| Hattori (2007) | Japan | Asia | Cross-sectional | NR | RT-PCR | No | 182 | 20/3 | 22/52 | 8/11 | NR | G1=3, G2=12, G3=22 |
| Haji Molla Hoseini (2007) | Iran | Asia | Cross-sectional | NR | RT-PCR | No | 103 | 15/5 | 16/05 | 13/64 | NR | NR |
| Hekmat (2008) | Iran | Asia | Cross-sectional | NR | RT-PCR | Yes | 106 | 11/3 | 13/92 | 3/70 | 39.3±11.1 | G2a=11 |
| Zhu (2008) | China | Asia | Cross-sectional | NR | RT-PCR | No | 203 | 25/6 | 29/91 | 19/77 | 41.62±9.13 | NR |
| Ramezani (2009) | Iran | Asia | Cross-sectional | NR | RT-PCR | NR | 82 | 10/97 | NR | NR | NR | NR |
| Compston (2009) | Ghana | Africa | Cohort | NR | RT-PCR | NR | 288 | 16/3 (RT-PCR),13/1 (ELISA) | NR | NR | NR | NR |
| Rabbad (2010) | Yemen | Asia | Cross-sectional | 2007-2008 | ELISA | Yes | 60 | 6/7 | NR | NR | NR | NR |
| Keyvani (2010) | Iran | Asia | Cross-sectional | NR | RT-PCR | NR | 80 | 18/7 | 20/63 | 11/76 | NR | NR |
| Alcalde (2010) | Brazil | South America | Cross-sectional | 2003-2004 | RT-PCR | Yes | 210 | 30 | 33/57 | 22/39 | 37.5 (34–43) | G1=5, G2a=20, G2b=21, G3=3 |
| Schwarze-Zander (2010) | Germany | Europe | Cross-sectional | NR | RT-PCR | Yes | 128 | 30 | 34/69 | 13/33 | NR | NR |
| Giret (2011) | Brazil | South America | Cross-sectional | NR | RT-PCR | No | 233 | 23/2 | 23/58 | 22/22 | 27.61 (24–35) | G1=5, G2a=13, G2b=27 |
| Feng (2011) | China | Asia | Cross-sectional | 2005–2008 | RT-PCR | NR | 70 | 44/3 | NR | NR | NR | NR |
| Campos (2011) | Brazil | South America | Cohort | 1996-1999 | RT-PCR, ELISA | Yes | 248 | 23 (RT-PCR), 23/3 (ELISA) | 0/00 | 23/00 | 31.8 (27.9–38.4) | NR |
| Supapol (2011) | Thailand | Asia | Cross-sectional | 1992–1994, 1996–1998, 1999–2004 | RT-PCR, ELISA | Yes | 1387 | 19/7 (RT-PCR),13 (ELISA) | 0/00 | 19/70 | 25 (22-28) | G2a=107, G2b=5, G3=95, G4=43 |
| Maaref (2011) | Tunis | Africa | Cross-sectional | 2006 | RT-PCR | NR | 125 | 36/8 | 35/53 | 38/78 | NR | NR |

| Autor/Publish Year | Country | Continent | Study Design | Study Period | Diagnostic Method | ART status | Sample Size | HGV Prevalence (%) | | | Age of Patients with HIV/HGV (year) | HGV Genotypes (N) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Total | Male | Female | | |
| Alves-Sousa (2012) | Brazil | South America | Cross-sectional | 2006-2007 | RT-PCR, ELISA | Yes | 102 | 21/5 (RT-PCR), 26/4 (ELISA) | NR | NR | 40.8 | NR |
| Maaref (2011) | Tunis | Africa | Cross-sectional | 2006 | RT-PCR | NR | 125 | 36/8 | 35/53 | 38/78 | NR | NR |
| Alves-Sousa (2012) | Brazil | South America | Cross-sectional | 2006-2007 | RT-PCR, ELISA | Yes | 102 | 21/5 (RT-PCR), 26/4 (ELISA) | NR | NR | 40.8 | NR |
| Wu (2012) | China | Asia | Cross-sectional | 2009-2010 | RT-PCR | Yes | 156 | 36/5 | NR | NR | NR | G2=2, G3=8 |
| Stapleton (2012) | USA | North America | Cohort | NR | RT-PCR | No | 59 | 41 | NR | NR | NR | NR |
| Silva (2012) | Brazil | South America | Cohort | 1997-1999 | RT-PCR, ELISA | NR | 253 | 22/5 (RT-PCR), 25/3 (ELISA) | NR | NR | NR | NR |
| Tenckhoff (2012) | USA | North America | Cohort | 1989-990 | RT PCR | Yes | 191 | 24/6 | NR | NR | NR | NR |
| Rydze (2012) | USA | North America | Cross-sectional | NR | RT-PCR | Yes | 49 | 53 | 53/85 | 50/00 | 46/50 | NR |
| Keyvani (2012) | Iran | Asia | Cross-sectional | NR | RT-PCR | No | 71 | 19/7 | 20/97 | 11/11 | NR | NR |
| Liu (2012) | China | Asia | Cross-sectional | 2007-2010 | RT-PCR | NR | 99 | 27/2 | 27/27 | 0/00 | 26.25±2.05 | G2=4, G3=30 |
| Vahidnia (2012) | USA | North America | Cohort | 1995-1999 | RT-PCR, ELISA | Yes | 489 | 11/4 (RT-PCR), 35/4 (ELISA) | NR | NR | NR | NR |
| Ernst (2013) | Germany | Europe | Cohort | 1993-2012 | RT-PCR, ELISA | No | 156 | 16/6 (RT-PCR), 27/6 (ELISA) | 11/48 | 35/29 | 30.3(28.5–35.3) | NR |
| Anggorowati (2013) | Indonesia | Asia | Cross-sectional | 2010 | RT-PCR | Yes | 125 | 88/8 | NR | NR | NR | G2a=56, G3= 12, G6= 27 |
| Sahni (2014) | Canada | North America | Cohort | NR | RT-PCR | Yes | 288 | 21/5 | 21/83 | 0/00 | 48/00 | NR |
| Rodríguez (2014) | Venezuela | South America | Cross-sectional | NR | RT-PCR | No | 255 | 27 | NR | NR | 29.4±5.6 | G1=2, G2=27, G3=15 |
| Yirrell (2015) | Uganda | Africa | Cohort | 2002-2007 | RT-PCR | NR | 167 | 41/9 | NR | NR | NR | G1=4, G5=15 |
| Mohamed Hassanein (2015) | Egypt | Africa | Cross-sectional | 2012-2015 | RT-PCR | No | 6 | 50 | NR | NR | NR | NR |
| Tien Ng (2015) | Malaysia | Asia | Cross-sectional | 2009-2010 | RT-PCR | NR | 215 | 20/9 | NR | NR | NR | G2=35, G3=8, G4=2 |
| Afkari (2016) | Iran | Asia | Cohort | 2007-2011 | RT-PCR, ELISA | No | 150 | 26/6 (RT-PCR), 28 (ELISA) | NR | NR | NR | NR |
| Dias Da Mota (2016) | Brazil | South America | Cross-sectional | 2011-2012 | RT-PCR | Yes | 347 | 34 | 35/39 | 32/54 | NR | NR |
| Jõgeda (2016) | Estonia | Europe | Cross-sectional | 2011 | RT-PCR | NR | 172 | 41/2 | NR | NR | 30 (25-34) | NR |
| Barbosa de Miranda (2017) | Brazil | South America | Cross-sectional | 2015 | RT-PCR | Yes | 313 | 17 | 18/45 | 14/02 | NR | NR |
| Miao (2017) | China | Asia | Cross-sectional | 2011-2015 | RT-PCR | NR | 1062 | 23/4 | 23/71 | 23/50 | 37.31±9.7 | G2=7, G3=74, G4=10, G7=118 |
| Santos (2017) | Brazil | South America | Cross-sectional | 2010-2015 | RT-PCR | Yes | 63 | 25 | NR | NR | NR | NR |
| Horemheb-Rubio (2017) | Mexico | North America | Cohort | 2009, 2010, 2015 | RT-PCR | No | 1406 | 33/6 | NR | NR | NR | NR |

| Autor/Publish Year | Country | Continent | Study Design | Study Period | Diagnostic Method | ART status | Sample Size | HGV Prevalence (%) | | | Age of Patients with HIV/HGV (year) | HGV Genotypes (N) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Total | Male | Female | | |
| Guessan (2017) | Botswana | Africa | Cross-sectional | 2005 | RT-PCR | No | 133 | 30/8 | 43/48 | 28/18 | 34.5±3.46 | G1=5, G5=22 |
| Gonçalves Pereira (2020) | Brazil | South America | Cross-sectional | 2016 | RT-PCR | Yes | 60 | 28/3 | 21/62 | 39/13 | NR | G2a=6, G2b=10 |
| Silva (2020) | Brazil | South America | Cross-sectional | 2017-2018 | RT-PCR | No | 120 | 26/6 | NR | NR | NR | NR |
| Isabel Inês M. de PinaAraujo (2021) | Cabo Verde | Africa | Cross-sectional | 2010-2011 | RT-PCR | Yes | 102 | 19/6 | 21/43 | 19/44 | NR | G1=5, G2a=11, G2b=2 |
| Köksal (2023) | Türkiye | Europe | Cross-sectional | 2020 | RT-PCR | Yes | 351 | 27/3 | 29 | 13 | 41.9 | G1=1, G2a=89, G2b=6 |

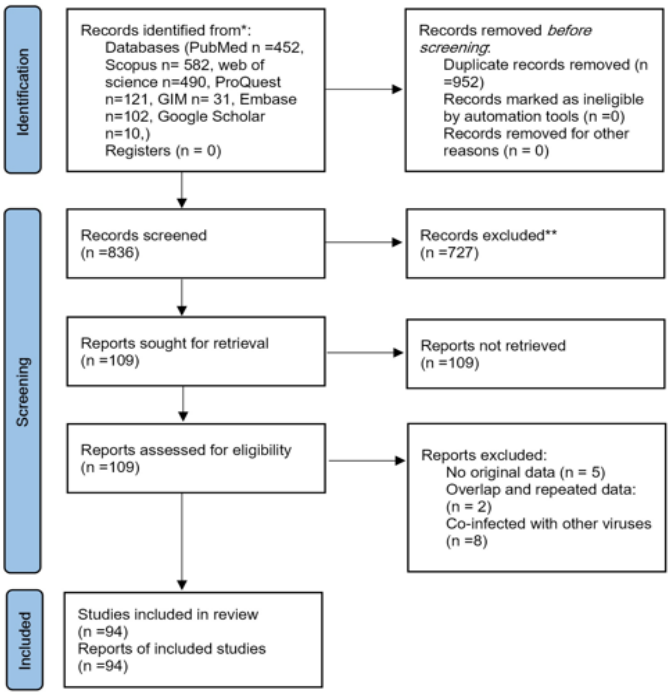


**Figure 1)** PRISMA flow diagram illustrating the study selection process for the systematic review and meta analysis of HPgV 1/HIV co infection

had a significant effect on the results of the meta-analyses ($p$ < .0001).

**Impact of HPgV-1 on HIV viral load:** A meta-analysis was performed on six eligible studies. The results showed that the overall mean of HIV viral load was not significantly different between HPgV-1 positive and HPgV-1 negative patients ($p$= .25) (Figure 3).

**Impact of HPgV-1 on CD4 cell count:** A meta-analysis was performed on nine eligible studies. The results showed that the overall mean of CD4 cell count was significantly different between HPgV-1-positive and HPgV-1-negative patients ($p$= .004) (Figure 4).

**Publication bias and sensitivity tests:** The Begg's test results indicated no statistically significant publication bias in the current meta-analysis (Tau = -0.077, $p$= .292). In addition, the Egger's test results indicated no strong evidence of publication bias (intercept = 1.30, $p$= .11). The funnel plot displayed a roughly symmetrical distribution of studies around the pooled effect estimate, with no clear visual evidence of publication bias (Figure 5). Most data points fell within the expected inverted funnel shape, suggesting minimal small-study effects. While minor asymmetry was observed in the lower

**Table 2)** Subgroup analysis of sources of heterogeneity in the pooled prevalence of hepatitis G virus in HIV-positive individuals

| Subgroup | | No. of Studies | Effect Size (95% CI) | Heterogeneity ($I^2$, P-Value) |
|---|---|---|---|---|
| Continent | Africa | 10 | 0.265 (0.214 – 0.323) | $I^2$=84.3%, $p$< .001 |
| | Asia | 20 | 0.219 (0.180 – 0.263) | $I^2$=90.3%, $p$< .001 |
| | Europe | 27 | 0.267 (0.238 – 0.298) | $I^2$=80.4%, $p$< .001 |
| | North America | 16 | 0.246 (0.196 – 0.304) | $I^2$=93.9%, $p$< .001 |
| | South America | 13 | 0.251 (0.223 – 0.283) | $I^2$=63.7%, $p$= .001 |
| | Overall | 86 | 0.253 (0.236 – 0.270) | $I^2$=88.9%, $p$< .001 |
| | Test for subgroup differences: Q=3.572, df=4, $p$= .467 | | | |
| Type of study | Clinical trial | 1 | 0.248 (0.174 – 0.339) | - |
| | Cohort | 24 | 0.251 (0.215 – 0.290) | $I^2$=88.9%, $p$< .001 |
| | Crosssectional | 61 | 0.248 (0.225 – 0.272) | $I^2$=89.2%, $p$< .001 |
| | Overall | 86 | 0.249 (0.230 – 0.269) | $I^2$=88.9%, $p$< .001 |
| | Test for subgroup differences: Q=0.019, df=2, $p$= .991 | | | |
| Year of publication | Pre 2000 | 16 | 0.252 (0.214 – 0.294) | $I^2$=78.5%, $p$< .001 |
| | 2000–2010 | 38 | 0.221 (0.193 – 0.252) | $I^2$=89.7%, $p$< .001 |
| | Post 2010 | 32 | 0.281 (0.249 – 0.316) | $I^2$=90.5%, $p$< .001 |
| | Overall | 86 | 0.251 (0.232 – 0.271) | $I^2$=88.9%, $p$< .001 |
| | Test for subgroup differences: Q=6.910, df=2, $p$= .032 | | | |
| Antiviral Drug | YES | 40 | 0.248 (0.215 – 0.284) | $I^2$=92.2%, $p$< .001 |
| | NO | 18 | 0.256(0.228 – 0.287) | $I^2$=73.7%, $p$< .001 |
| | Not Reported | 28 | 0.248 (0.219 – 0.280) | $I^2$=84.9%, $p$< .001 |
| | Overall | 86 | 0.251 (0.234 – 0.270) | $I^2$=88.9%, $p$< .001 |
| | Test for subgroup differences: Q=0.201, df=2, P= .904 | | | |
| Risk factor | Drug user | 35 | 0.293 (0.240 – 0.353) | $I^2$=88.2%, $p$< .001 |
| | Hemophilia | 9 | 0.242 (0.166 – 0.339) | $I^2$=45.6%, $p$< .001 |
| | Heterosexual | 30 | 0.180 (0.147 – 0.220) | $I^2$=80.1%, $p$< .001 |
| | Homosexual | 27 | 0.239 (0.203 – 0.280) | $I^2$=74.4%, $p$< .001 |
| | Transfusion | 11 | 0.161 (0.091 – 0.267) | $I^2$=74.6%, $p$< .001 |
| | Overall | 112 | 0.229 (0.207 – 0.253) | $I^2$=83.8%, $p$< .001 |
| | Test for subgroup differences: Q=13.548, df=4, $p$= .009 | | | |

**Table 3)** Results of the meta-regression analyses

| Covariate | Coefficient | Standard Error | 95% Lower | 95% Upper | Z-value | 2-sided p-value |
|---|---|---|---|---|---|---|
| Sample size | -0.0002 | 0.0002 | -0.0006 | 0.0003 | -0.62 | 0.4096 |
| Publication year | -0.0005 | 0.0000 | -0.0006 | -0.0005 | -13.81 | 0.0000 |

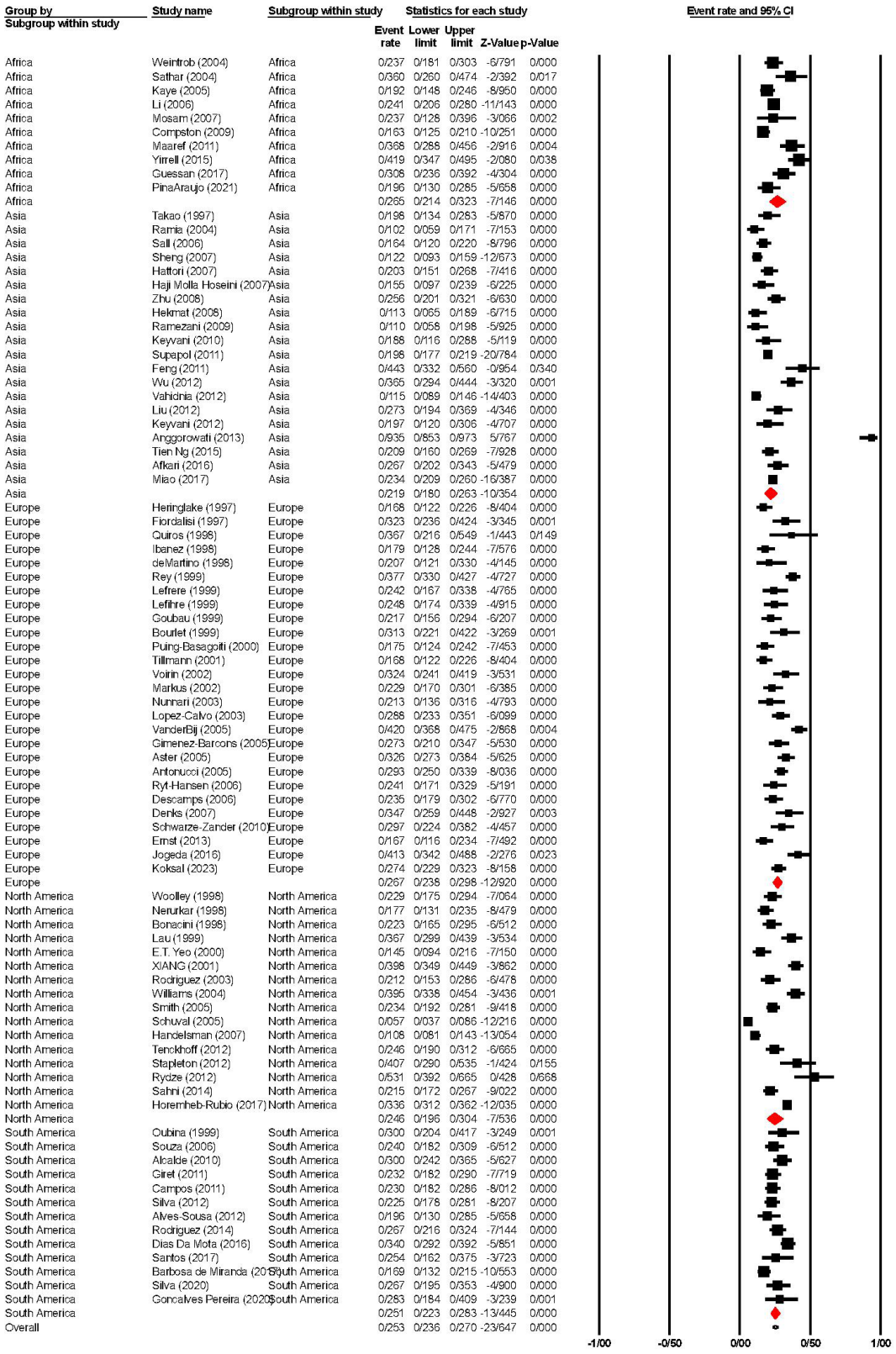


**Figure 2)** Forest plot of the overall prevalence of HPgV 1 among HIV positive patients. The pooled prevalence (25.3%; 95% CI 23.6–27%) was calculated using a random effects model.

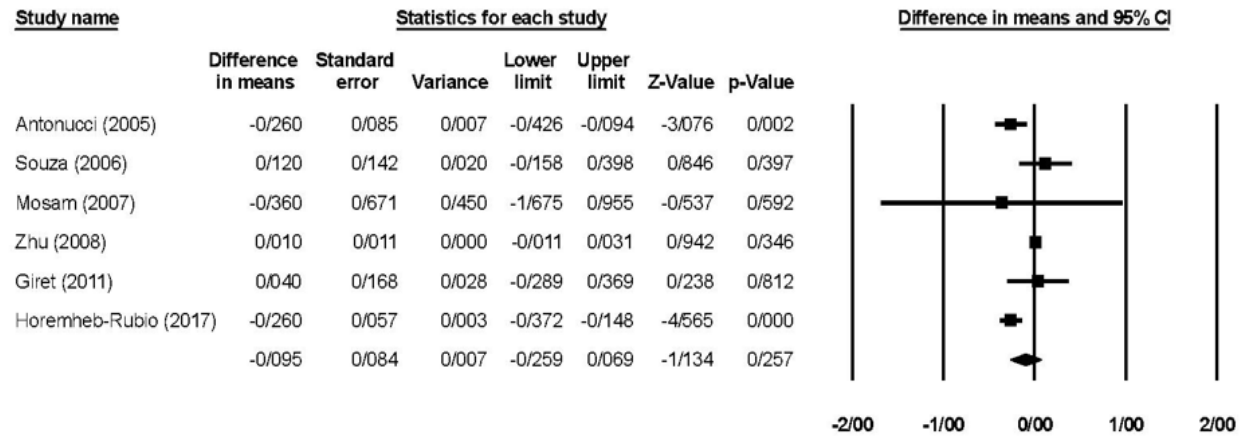


**Figure 3)** Forest plot comparing HIV viral load ($log_{10}$ copies/mL) between HPgV 1 positive and HPgV 1 negative HIV patients. The overall mean difference was not statistically significant (p = .25).

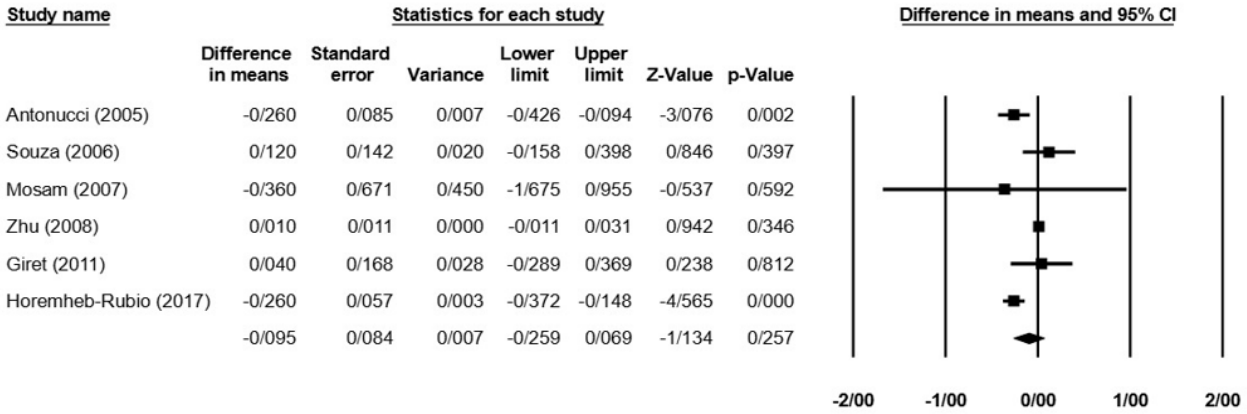


**Figure 4)** Forest plot comparing $CD4^+$ T cell count (cells/μL) between HPgV 1 positive and HPgV 1 negative HIV patients. Co infected patients showed significantly higher CD4 counts (p = .004).

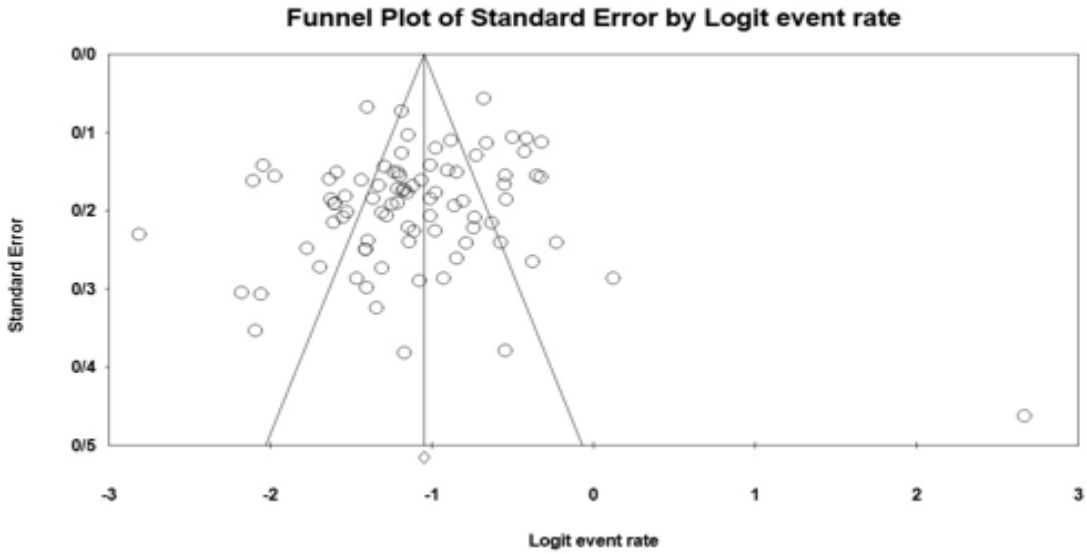


**Figure 5)** Funnel plot assessing publication bias for the meta analysis of HPgV 1 prevalence. The symmetric distribution of studies suggests no strong evidence of small study effects (Egger's test p = .11).

precision range (higher standard error), the overall pattern supported the robustness of the meta-analysis findings. This aligns with the non-significant Egger's test results ($p$= .11), further reinforcing confidence in the validity of the pooled estimates. Sensitivity analysis confirmed the robustness of the meta-analysis results, with stable pooled effect estimates (0.26, 95% CI: 0.25–0.27) across all studies excluded.

## Discussion

Since the discovery of hepatitis G virus in 1995, numerous studies have been conducted to estimate the prevalence rate of HPgV-1 in different populations, such as HIV-positive patients [16]. HPgV-1 infection is prevalent among HIV-positive people due to similar transmission modes. According to the current study, the frequency of HPgV-1/HIV co-infection varies from 5.7 to 93% in different regions. The present meta-analysis estimated the overall prevalence of HPgV-1 among HIV-positive patients to be 25.3% (95% CI 23.6–27%). The prevalence of HIV/HPgV-1 coinfection in different continents was as follows: 26.5% in Africa, 21.9% in Asia, 26.7% in Europe, 24.6% in North America, and 25.1% in South America. These variations in frequency could be explained by the various socioeconomic situation of the countries. Previous studies have reported that HPgV-1 prevalence is higher in developing regions than in developed regions [17]. For instance, the prevalence of HPgV-1 among healthy blood donors varies from 0.8–46.6% in developing countries of Asia and Africa and from 1.1–6% in developed countries of North America and Europe [6]. However, in the present meta-analysis, the prevalence of HPgV-1 among HIV-positive patients was lower in Asia than in other continents. This difference in prevalence could be attributed to different transmission routes or genetic susceptibility present within each population. This aligns with the findings of Yang and colleagues (2020) [7], who noted lower HPgV-1 seroprevalence in Asian blood donors. HPgV-1 is known as a blood-borne virus that could be transmitted through blood transfusion, sexual contact, and mother-to-fetus transmission, like HIV [18].

In 1998, early studies found that HIV-infected persons who were also infected with HPgV-1 had lower mortality rates and HIV viral loads [19, 20]. Subsequent to these reports, several investigations were conducted which verified the correlation between HPgV-1 infection and improved clinical outcomes or survival in HIV-positive populations. To summarize the data on the impact of HPgV-1 co-infection on the survival of HIV-positive patients, Zhang et al. (2006) performed a meta-analysis of 11 studies before the advent of HAART (highly active antiretroviral therapy) [21]. They found no clear evidence linking HPgV-1 infection to better survival of HIV-positive patients during two years of HIV seroconversion. However, when HPgV-1 infection persisted for more than two years following HIV seroconversion, they found a decrease in mortality (HR= 0.41, 95% CI=0.23). Although viral load and CD4 count are factors related to survival of HIV patients, no data on the effect of HPgV-1 infection on these two factors were reported in their study.

The findings of this meta-analysis demonstrated a significant association between HPgV-1/HIV coinfection and higher CD4 cell counts in HIV-positive patients ($p$= .004), while no such effect was observed on HIV viral load ($p$= .25). These results are consistent with those of previous observational studies, such as Xiang et al. (2001) [8] and Yeo et al. (2000) [9], which hypothesized a possible beneficial role of HPgV-1 in slowing HIV progression; however, these studies, like the present meta-analysis, could not establish causality. However, the results contrast with those of the meta-analysis by Zhang

et al. (2006) [21], who found no clear survival benefit during the first two years of HIV seroconversion but noted improved outcomes in long-term HPgV-1 coinfection. This inconsistency may be attributed to differences in HPgV-1 genotype distributions or host immune responses, though remaining confounding factors could not be excluded. The observed association between HPgV-1 coinfection and higher CD4 count is compatible with the hypothesis that HPgV-1 may modulate immune activation, as proposed by Horemheb-Rubio et al. (2017) [10], but because this analysis was based on cross-sectional comparisons, and the included studies did not uniformly adjust for potential confounders such as HIV disease stage and duration of infection, no causal protective effect could be inferred. Therefore, the clinical significance remains uncertain pending long-term prospective data.

The lack of impact on HIV viral load in this study contradicts some earlier reports, such as Heringlake et al. (1998) [19], who proposed that HPgV-1 might suppress HIV replication. This inconsistency could reflect variations in HPgV-1 genotypes or host immune responses across different regions. For instance, HPgV-1-2, the most prevalent genotype in this analysis (48.65%), may interact differently with HIV compared to other genotypes. Additionally, the small number of studies included in the viral load analysis (n=6) limits the power to detect subtle effects, highlighting the need for larger, longitudinal studies.

## Conclusion

HPgV-1 coinfection was not significantly associated with HIV viral load ($p$= .25) but was associated with higher CD4 cell counts in co-infected patients ($p$= .004). However, this cross-sectional association does not establish a causal protective effect, as the included studies did not uniformly adjust for important confounders such as HIV disease stage and duration of infection. These findings highlight the need for rigorous longitudinal studies that could control for such factors to determine whether and how HPgV-1 infection modulates HIV disease progression.

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**Authors' contributions:** Shayestehpour M: study design and conceptualization, investigation, writhing, reviewing, preparation of the original draft. Piroozmand A: reviewing, editing, and supervision

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