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Review

Sexual transmission of hepatitis C virus among gay and bisexual men: a systematic review

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Abstract. A systematic review was performed on the evidence of sexual transmission of hepatitis C virus (HCV) in gay and bisexual men (GBM). Studies conducted in industrialised countries and published in English from 2000 to 2015 with data on HCV in GBM were included. Pooled estimates of prevalence and incidence of HCV infection were stratified by study settings and participants' HIV status using random effect models. Case-series reports were summarised descriptively. Of the 38 cross-sectional studies, the pooled HCV prevalence was substantially higher in HIV-positive men (8.3%, 95% CI: 6.7–9.9) than in HIV-negative men (1.5%, 95% CI 0.8–2.1), and higher in those who reported injecting drug use (34.8%, 95% CI 26.9–42.7) than in those who did not (3.5%, 95% CI 2.4–4.5). Of the 16 longitudinal studies, the pooled HCV incidence was markedly higher in clinic-based (7.0 per 1000 person-years, 95% CI 4.6–9.5) than in community-based (1.4 per 1000 person-years, 95% CI 0.7–2.1) studies, and in HIV-positive men (6.4 per 1000 person-years, 95% CI 4.6–8.1) than in HIV-negative men (0.4 per 1000 person-years, 95% CI 0–0.9). Since the early 2000s, 15 case-series reports increasingly pointed to the importance of sexual transmission of HCV in mainly HIV-positive men. Injecting drug use remained the major transmission route of HCV in GBM. Receptive condomless intercourse and concurrent ulcerative sexually transmissible infections are likely drivers that facilitated HCV sexual transmission in HIV-positive men. HCV incidence remains very low in HIV-negative GBM.

Additional keywords: homosexuality, male, injecting drug use, pooled estimate, risk factors.

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Introduction

Hepatitis C is a liver disease caused by hepatitis C virus (HCV) infection. Among 18–34% of those with acute HCV infection will spontaneously clear the virus within 6 months;[1] however, the remaining will become chronic and approximately 25% of chronic HCV infection will lead to liver cirrhosis. Approximately 25% of those with liver cirrhosis will eventually develop end-stage liver disease or liver cancer.[1,2] Globally, ~130–150 million people are living with chronic HCV infection.[3] In Australia, this figure was estimated to be 230 470 in 2014.[4]

Parenteral exposures have long been established as the main transmission mode for HCV, primarily among injecting drug users (IDU) and in blood transfusion recipients. The latter has been largely eradicated in industrialised countries since the advent and the implementation of routine HCV serological testing among blood donors in the early 1990s.[5]

The extent to which this virus can be transmitted through sexual contact has been controversial.[6] Early studies focusing on sexual partners of HCV-infected haemophiliac index cases reported low HCV prevalence, but slightly higher prevalence was observed in partners in which the index cases were HCV/HIV co-infected.[7] Other studies among sexual partners of non-haemophiliac index cases and sexual health clinic attendees showed slight excesses of HCV prevalence in those high-risk populations.[7] Nevertheless, the risk of parenteral transmission could not be ruled out in these studies, as IDU may be over-represented in the study populations.

Gay and bisexual men (GBM) are particularly vulnerable to sexually transmissible infections (STI) due to higher exposure to new sexual partners.[8] HIV-positive GBM men have an even higher burden of STIs.[9] The prevalence of HCV infection is also markedly higher in those living with HIV compared with their HIV-negative counterparts.[10] Up until recently, the higher incidence of HCV infection appears to be limited to HIV-positive GBM.[10] Why higher incidence is limited to HIV-positive men is unclear, but potential reasons included behavioural factors, such as higher rates of condomless anal intercourse (CAI) mainly with other HIV-positive partners,[11] and biological factors, such as higher HCV viral load in semen in addition to lower immunity,[12] in HIV-positive than in HIV-negative GBM. However, this may change because of the implementation of pre-exposure prophylaxis (PrEP) aimed at high-risk HIV-negative GBM as a means of biomedical HIV

prevention.[13,14] There are concerns that high-risk HIV-negative GBM might be at increased risk of HCV infection via sexual contact by having CAI with HCV/HIV co-infected partners.[15,16]

This systematic review set out to summarise evidence published since the year 2000 on the risk of HCV infection in GBM.

Methods

Eligibility criteria

This review considered full text articles published from 2000 to 2015 with data on HCV prevalence or incidence or risk factors in GBM. Eligible articles included original research and letters to the editor. Only studies conducted in industrialised countries and written in English were considered. Articles used self-reported HCV status only, with no laboratory confirmation of infection were excluded. For studies concerning HIV-positive individuals, only those that presented data separately for HIV-positive GBM were included.

Information sources

A literature review was conducted on PubMed for the period between 1 January 2000 and 31 December 2015. Articles in the reference list of published papers that were published in the same period were also searched.

Search

The following keywords were used to search articles: (gay OR homosexuality, male) AND (HCV OR hepatitis C) AND (prevalence OR incidence OR risk factors).

Study selection

Article titles and abstracts were reviewed for inclusion, based on the inclusion and exclusion criteria. Studies were included in the review if they had quantitative measures of the variables of interest and were undertaken for populations that included HIV-positive or HIV-negative men who were described as homosexual, bisexual or men who have sex with men.

Data collection process

A data abstraction table was used to record data from articles, as recommended by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[17]

Data items

Data were recorded separately for studies of various designs, including case-series reports, cross-sectional and longitudinal studies. First author, the year of publication, and the city and country of the origin of the study were recorded for all studies. For case-series reports, extra data points included: number of cases, number of cases who were HIV-positive, proportion of cases who were non-IDU and potential risk factors that contributed to HCV acquisition. For cross-sectional studies, extra data points included: study settings (community- or clinic-based), methods of HCV testing, overall sample size and HCV prevalence, then stratified by IDU and HIV status, and other risk factors identified. HCV prevalence was not recorded for case-control studies. For longitudinal studies, extra data points included: study settings, methods of HCV testing, overall sample size, person-years and HCV incidence, then stratified by IDU and HIV status, and other risk factors identified.

Summary measures

In cross-sectional studies, HCV prevalence was presented as a percentage of participants who tested HCV positive among the total number tested. In longitudinal studies, HCV incidence was presented as the number of participants who had acquired HCV infection divided by per 1000 person-years of follow up.

Statistical analysis

The prevalence and incidence of HCV infection were summarised in forest plots. Pooled prevalence and incidence and their 95% confidence intervals (CI) were estimated using random effect models because of the high heterogeneities expected arising from a diversity of study populations and HCV testing methods. Pooled estimates were stratified by study settings and participants' HIV status. For cross-sectional studies, there was an additional stratification by drug injecting status. The stratification by drug injecting status was not summarised in a forest plot for longitudinal studies because only a few studies ($n = 3$) had this information available.

Results

A total of 260 studies were identified, of which 65 studies fit the selection criteria and 60 were included in the systematic review after the removal of five repeat studies (Fig. 1). Seven studies were included in both cross-sectional and longitudinal reviews, and two were included in both cross-sectional and case-series reports.

Cross-sectional studies

Thirty-eight studies were included, including five case-control studies (Table 1).

HCV prevalence stratified by study setting

Among 14 studies that recruited GBM from community-based settings, HCV prevalence ranged between 0 and 11.6%, with a pooled prevalence of 3.4% (95% CI: 2.1–4.7; Fig. 2). The study that reported a zero prevalence involved non-injecting partners of IDUs in Scotland and included only 12 GBM.[18] The study that reported the highest HCV prevalence at 11.6% tested serum collected as part of the US National HIV Behavioural Surveillance system (NHBS) in 2004 and 2008 from HIV-positive GBM in San Francisco.[19] Apart from this study, HCV prevalence in community-recruited GBM was generally under 5%. In 18 studies that recruited participants from clinic-based settings, HCV prevalence ranged between 0.7 and 14.2%, with a pooled prevalence of 5.7% (95% CI: 4.2–7.2, Fig. 2).

HCV prevalence stratified by HIV status

In HIV-negative men, HCV prevalence ranged between 0.6% and 5.6% among 14 studies. The majority (10 studies) reported a prevalence of under 1.5%, and the pooled prevalence was 1.5% (95% CI: 0.8–2.1, Fig. 3). The pooled HCV prevalence in

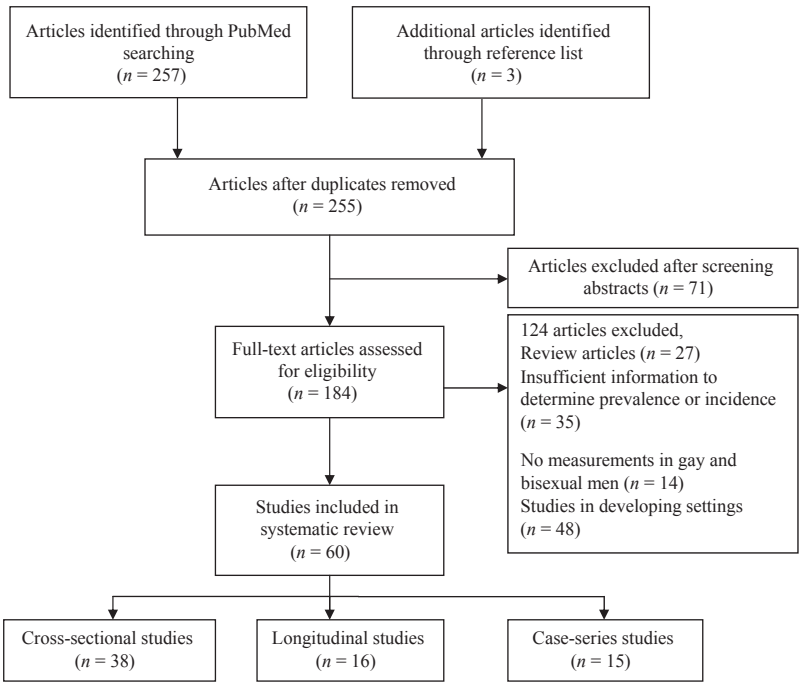

Fig. 1. Articles identified and screened for eligibility.

HIV-positive GBM among 26 studies was markedly higher at 8.3% (95% CI: 6.7–9.9, Fig. 3). Close to half of the studies (11 studies) reported a prevalence of above 10% in HIV-positive men.

HCV prevalence stratified by drug injecting status

There was a striking difference in pooled HCV prevalence between GBM who did and who did not report a history of injecting drug use. A total of 17 studies included participants who were non-IDUs, and HCV prevalence was reported to be under 5% in 12 studies, with a pooled prevalence of 3.5% (95% CI: 2.5–4.5, Fig. 4*a*). In contrast, the majority (14 studies) of the 18 studies that had data in IDUs reported a prevalence of above 20%, with a pooled prevalence of 34.8% (95% CI: 26.9–42.7, Fig. 4*a*). Among those who did not report injecting drug use, the pooled prevalence in HIV-negative men was 0.9% (95% CI: 0.0–1.8) and 7.1% (95% CI: 5.1–9.0) in HIV-positive men (Fig. 4*b*). In IDUs, the pooled prevalence was similar between HIV-negative (23.5%, 95% CI: 7.9–39.2) and HIV-positive men (35.7%, 95% CI: 26.4–45.1, Fig. 4*c*).

Other risk factors in addition to injecting drug use

A history of STIs was associated with HCV prevalence in seven studies.[20–26] Receptive CAI was reported as a risk factor for prevalent HCV infection in five studies,[21,24–27] and in two of them, the association was independent of injecting drug use.[25,26] Traumatic sexual practices, such as fisting, were also identified as a risk factor in five studies.[20,21,25,28,29]

Longitudinal studies

Sixteen studies were included (Table 2).

HCV incidence stratified by study setting

In four longitudinal studies that recruited GBM from the community, the HCV incidence ranged between 0.4 and 2.1 per 1000 person-years, with a pooled incidence of 1.4 per 1000 person-years (95% CI: 0.7–2.1, Fig. 5). This is markedly lower than the pooled incidence of 7.0 per 1000 person-years (95% CI: 4.6–9.5) arising from nine cohort studies conducted in clinics (Fig. 5). The highest incidence was reported in 379 HIV-positive men attending a community healthcare centre in Boston, US. There was more than 1480 person-years of follow up in this study; 23 men seroconverted, hence an incidence rate of 16.3 per 1000 person-years.[30]

HCV incidence stratified by HIV status

In five longitudinal studies that recruited HIV-negative GBM, the pooled HCV incidence was 0.4 per 1000 person-years (95% CI: 0–0.9, Fig. 6). One Dutch study followed 1565 initially HIV-negative men for a total of 10 888 person-years between 1982 and 2012, and reported no seroconversions to HCV. In 13 studies that recruited HIV-positive men, the pooled HCV incidence was substantially higher at 6.4 per 1000 person-years (95% CI: 4.6–8.1, Fig. 6). In five studies, the reported HCV incidence was above 9 per 1000 person-years.

Injecting drug use as a risk factor

Injecting drug use was examined as a risk factor for incident HCV infection in three studies. Among 1182 HIV-positive men, a clinic-based study in Japan reported an HCV incidence of 7.9 and 40.0 per 1000 person-years in non-IDUs and IDUs, respectively.[31] The large-scale Multicenter AIDS Cohort in the US reported an HCV incidence of 1.6 per 1000 person-years

Table 1. Cross-sectional studies on hepatitis C virus (HCV) infection in gay and bisexual men between 2000 and 2015
EIA, enzyme immunoassay; ALT, alanine aminotransferase; PCR, polymerase chain reaction

Reference	Year	City/Country	Setting	HCV testing
Hammer *et al.*[69]	2003	San Francisco, US	Clinic-based	EIA
Diamond *et al.*[70]	2003	Seattle, US	Community-based	EIA
Roy *et al.*[18]	2004	Glasgow, UK	Community-based	EIA
Amin *et al.*[71]	2004	Multinational	Clinic-based	Serology and PCR
Rauch *et al.*[39]	2005	Switzerland	Clinic-based	EIA
Alary *et al.*[59]	2005	Montreal, Canada	Community-based	EIA
Cohen *et al.*[72]	2006	Boston, US	Clinic-based	EIA
van de Laar *et al.*[50]	2007	Amsterdam, Netherlands	Community-based	EIA
Buffington *et al.*[74]	2007	US	Clinic-based	EIA
Danta *et al.*[21]	2007	England, UK	Clinic-based[A]	Serology or (PCR and ALT)
Dougan *et al.*[75]	2007	UK	Clinic-based	Serology or PCR
Bollepalli *et al.*[73]	2008	Phoenix, US	Clinic-based[A]	Serology
Kim *et al.*[76]	2008	New York, US	Clinic-based	EIA
Larsen *et al.*[77]	2008	France	Clinic-based	Serology or PCR
Urbanus *et al.*[29]	2009	Amsterdam, Netherlands	Clinic-based	EIA
Palacios *et al.*[78]	2009	Spain	Clinic-based	EIA
Myers *et al.*[53]	2009	Canada	Community-based	EIA
Scott *et al.*[79]	2010	England UK	Clinic-based	Serology
Jin *et al.*[22]	2010	Sydney, Australia	Community-based	Serology
CDC[27]	2011	New York, US	Clinic-based[A]	Serology
Schmidt *et al.*[28]	2011	Germany	Clinic-based[A]	Medical record
Raymond *et al.*[19]	2011	San Francisco, US	Community-based	Serology
Raymond *et al.*[54]	2012	San Francisco, US	Community-based	Serology
Marongiu *et al.*[80]	2012	UK	Community-based	EIA
Mayer *et al.*[81]	2012	Rhode Island, US	Community-based	Serology
Price *et al.*[24]	2013	London, UK	Community-based	EIA
Matser *et al.*[23]	2013	Amsterdam, Netherlands	Clinic-based	EIA
Garg *et al.*[30]	2013	Boston, US	Clinic-based	Serology
Seaberg *et al.*[56]	2014	US	Community-based	EIA
Kouyos *et al.*[82]	2014	Switzerland	Clinic-based	EIA or PCR
Urbanus *et al.*[25]	2014	Netherland	Clinic-based	EIA
Oster *et al.*[83]	2014	US	Community-based	Serology
Schmidt *et al.*[84]	2014	Zurich, Switzerland	Community-based	Serology
Nishijima *et al.*[31]	2014	Tokyo, Japan	Clinic-based	Serology
Marcellin *et al.*[85]	2015	France	Clinic-based	Medical record
Wong *et al.*[26]	2015	Vancouver, Canada	Community-based	Dried blood spot, EIA
Apers *et al.*[20]	2015	Belgium	Clinic-based[A]	Serology
Jansen *et al.*[38]	2015	Germany	Clinic-based	Serology

[A]Case-control studies.

in non-IDUs, and this was significantly higher in previous and current IDUs (6.9 and 25.8 per 1000 person-years, respectively).[32] In Melbourne, Australia, 620 HIV-positive men were followed up for a total of 4359 person-years, and the incidence of HCV was significantly higher in IDUs (hazard ratio = 8.7, 95% CI 4.6–16.6).[33]

Other risk factors in addition to injecting drug use

An increase in HCV incidence over time, starting from the late 1990s and early 2000s, was highlighted in five studies, all of which were conducted in Europe.[34–38] Similar to cross-sectional studies, CAI[32,37,39] and a history of syphilis[32,37] were also reported as risk factors for incident HCV infection. However, the association with CAI was only significant in non-IDU in one Swiss clinic-based study of 1543 HIV-positive men.[39]

Case-series reports

Since the early 2000s, case-series reports of acute HCV infection among mainly HIV-positive GBM began to surface. A total of 15 reports were included in this review (Table 3). The first report emerged in France, with five men presenting with infectious syphilis who also acquired acute HCV infection.[40] All denied a history of injecting drug use, but CAI was identified as an important risk factor. Shortly afterwards, similar findings were echoed in other studies from major cities in Europe,[41–50] Australia,[51,52] and the US.[16,27] The number of cases in these reports ranged from 2 to 74 men. Until recently,[16,47] nearly all men in these cases were HIV positive. In 11 of these studies, over 90% of the men were non-IDUs,[27,41–50] and co-diagnoses with ulcerative STIs were common, including syphilis,[40,42–44,50] lymphogranuloma venereum (LGV)[42,46,50] and genital herpes.[44,50] Traumatic sexual practices were also frequently reported, particularly fisting.[44,46,50] In 2015, McFaul *et al.* summarised

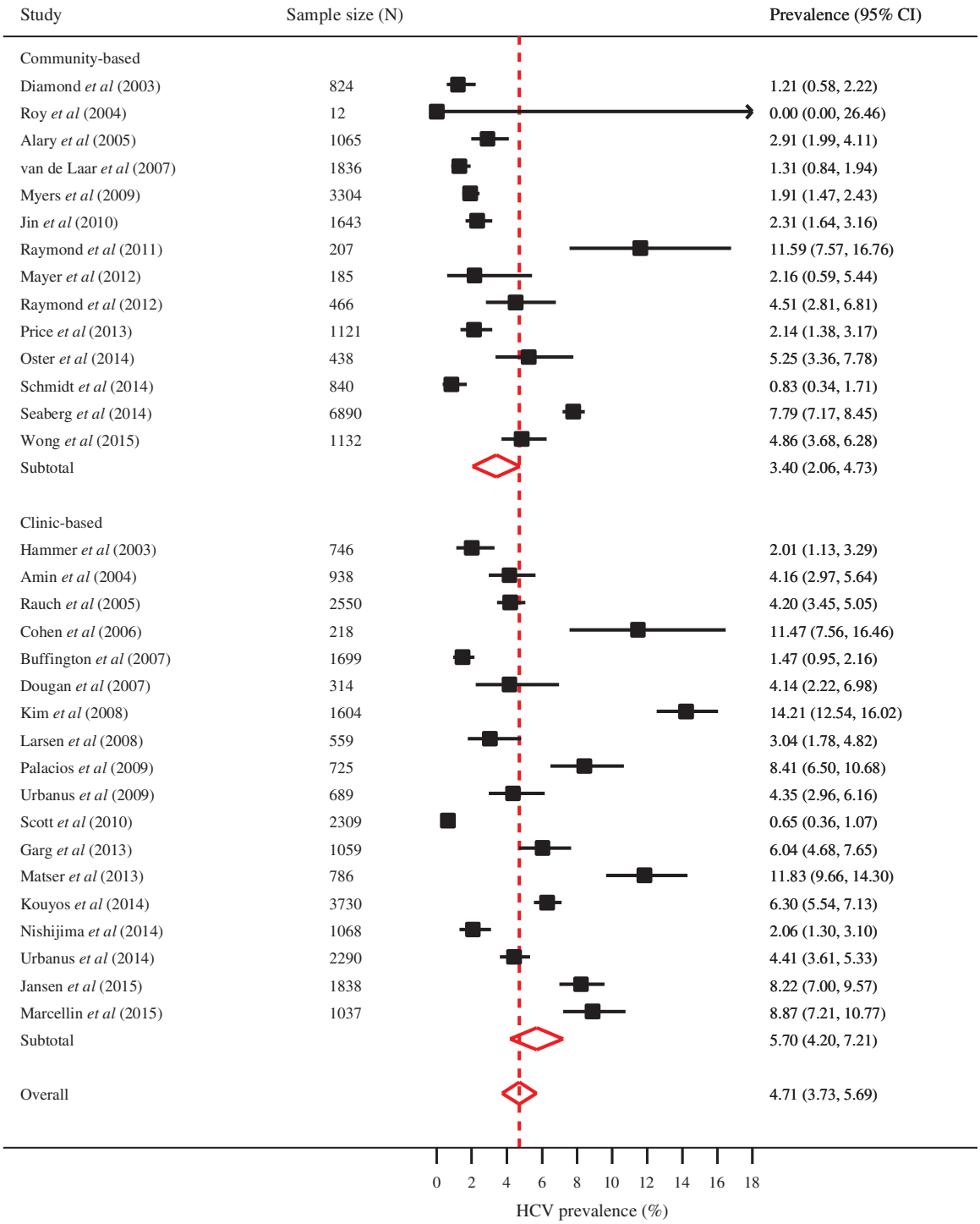

Fig. 2. Hepatitis C virus prevalence in cross-sectional studies involving gay and bisexual men stratified by community- versus clinic-based recruitment.

42 cases of acute HCV infection diagnosed in Europe between 2010 and 2014 who were HIV-negative GBM,[47] with nearly 80% being non-IDU. Almost all (93%) reported CAI, and one-quarter reported fisting. A co-existing rectal or ulcerative STI was diagnosed in nearly one-third (29.5%). This report also noted that eight men received post-exposure HIV prophylaxis within 6 months before acute HCV infection, and two men were participants of a PrEP trial in the UK. In the same year, Volk *et al.* also reported two cases of acute HCV infection among participants of a PrEP trial in the US who also denied a history of injecting drug use;[16] both were diagnosed with syphilis and rectal STIs on numerous occasions during the study.

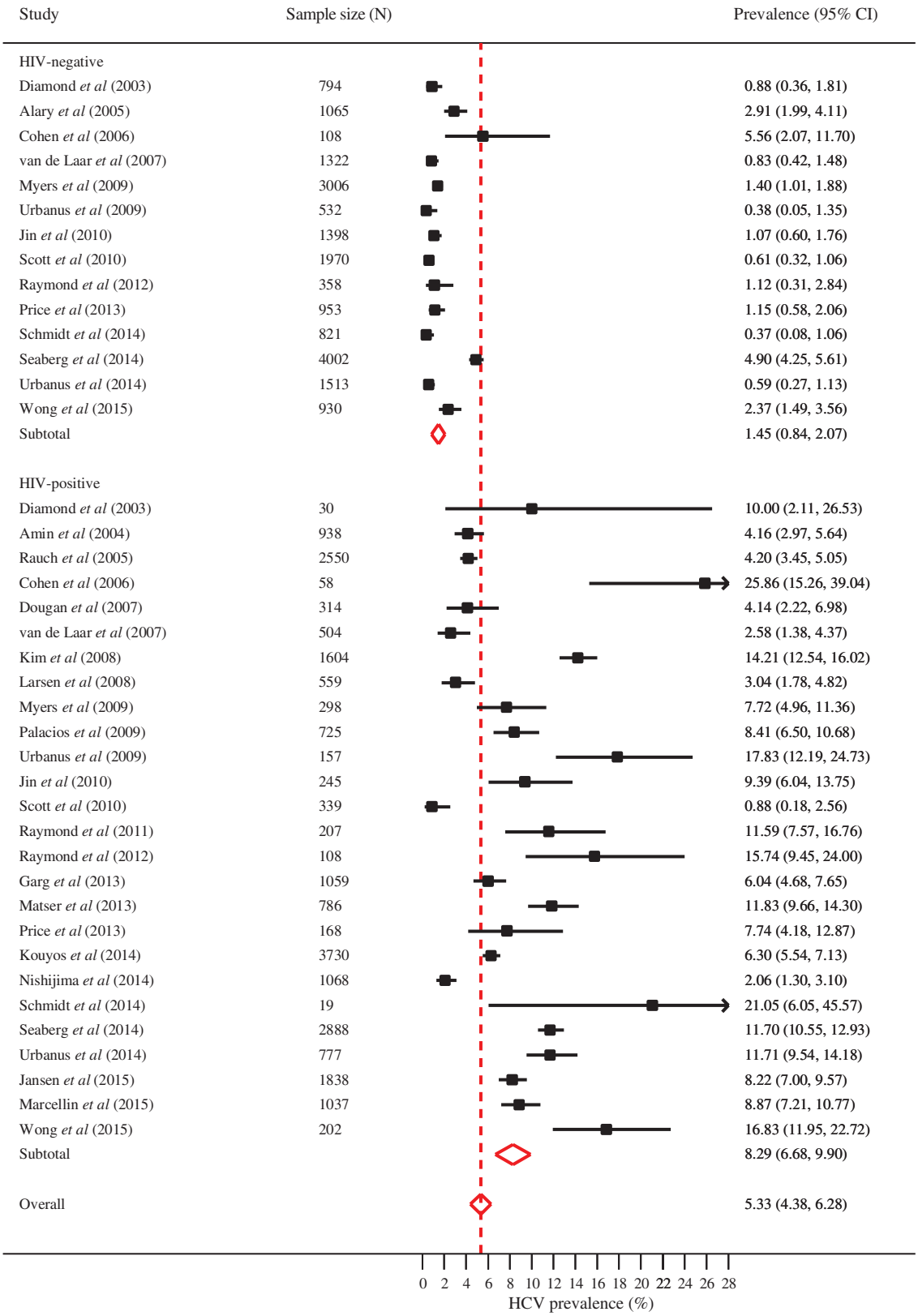

Fig. 3. Hepatitis C virus prevalence in cross-sectional studies involving gay and bisexual men stratified by HIV status.

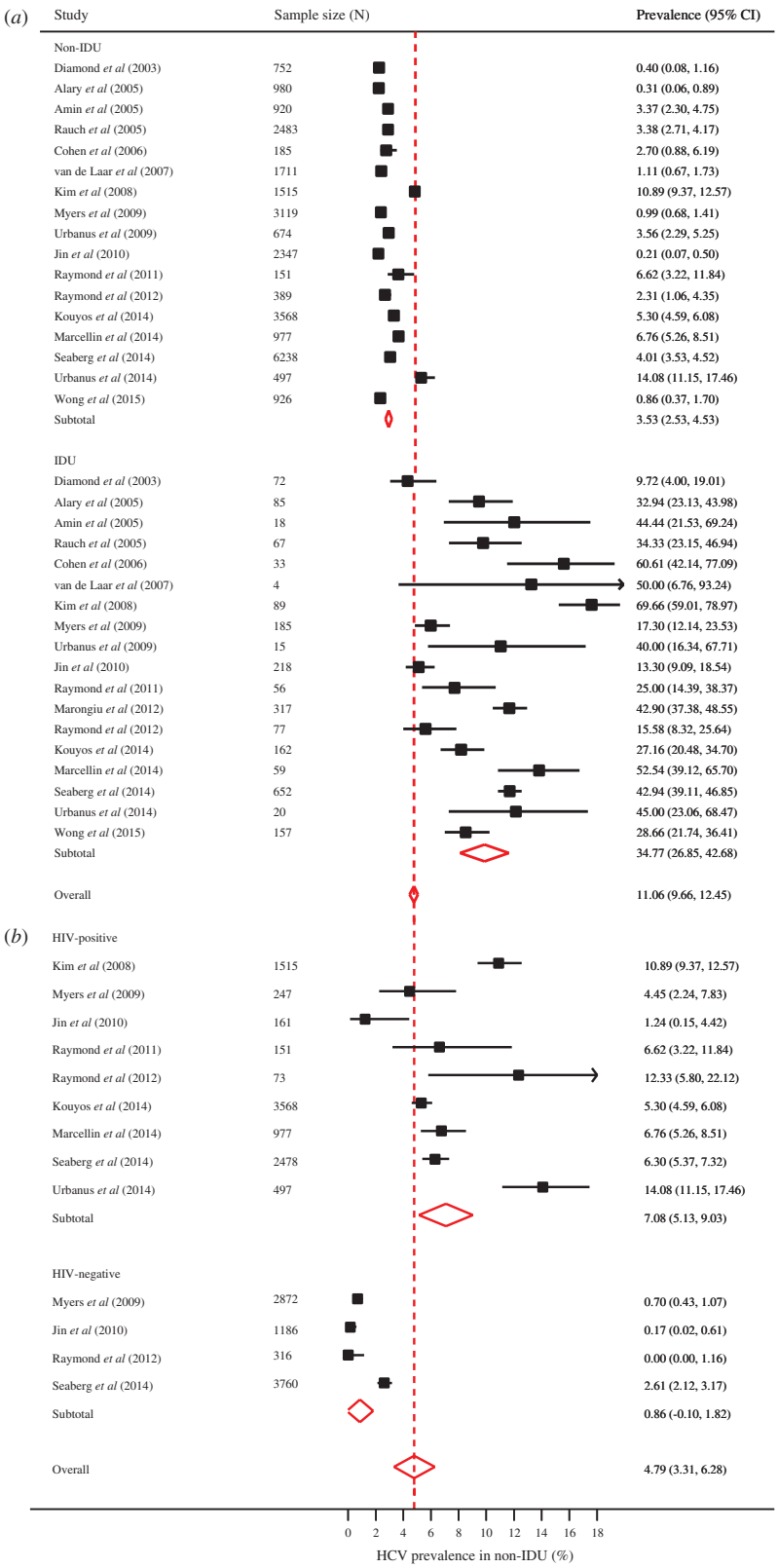

Fig. 4. (*a*) Hepatitis C virus prevalence in cross-sectional studies involving gay and bisexual men stratified by injecting drug use status. (*b*) HCV prevalence in cross-sectional studies involving non-injecting drug users stratified by HIV status. (*c*) HCV prevalence in cross-sectional studies involving injecting drug users stratified by HIV status.

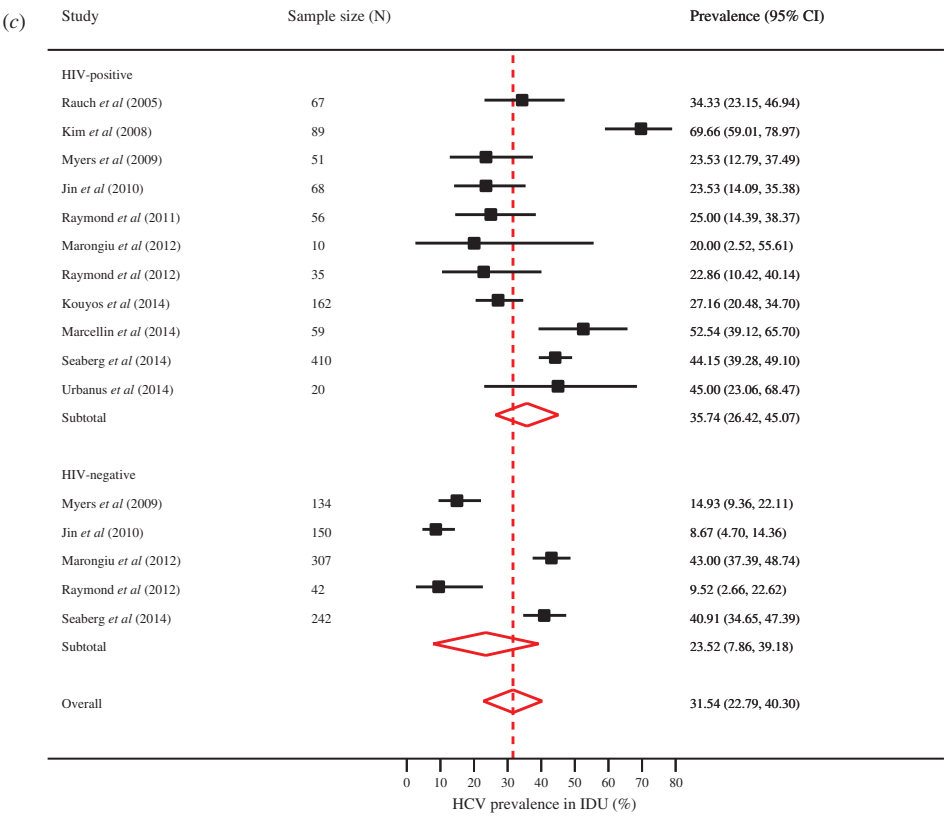

Fig. 4. (*continued*).

Table 2. Longitudinal studies on hepatitis C virus (HCV) infection in gay and bisexual men between 2002 and 2015
EIA, enzyme immunoassay; PCR, polymerase chain reaction

First author	Year	City/Country	Setting	HCV testing
Hammer *et al.*[69]	2003	San Francisco, US	Clinic	EIA
Rauch *et al.*[39]	2005	Switzerland	Clinic	EIA
Alary *et al.*[59]	2005	Montreal, Canada	Community	EIA
Turner *et al.*[86]	2006	London, UK	Clinic	Serology or PCR
Richardson *et al.*[35]	2008	Brighton, UK	Clinic	Serology
Giraudon *et al.*[34]	2008	Brighton and London, UK	Clinic	Serology or PCR
Palacios *et al.*[78]	2009	Spain	Clinic	EIA
Jin *et al.*[22]	2010	Sydney, Australia	Community	EIA
van der Helm *et al.*[63]	2011	Europe and Canada	Clinic	Serology and/or PCR
Gamage *et al.*[33]	2011	Melbourne, Australia	Clinic	EIA
Wandeler *et al.*[37]	2012	Switzerland	Clinic	EIA
Witt *et al.*[32]	2013	US	Community	EIA
Garg *et al.*[30]	2013	Boston, US	Clinic	Serology
Kouyos *et al.*[87]	2014	Switzerland	Clinic	Serology or PCR
Vanhommerig *et al.*[36]	2014	Amsterdam, Netherlands	Community	EIA
Nishijima *et al.*[31]	2014	Tokyo, Japan	Clinic	Serology

Discussion

Hepatitis C virus prevalence varied markedly in studies concerning GBM. Overall, the pooled prevalence in studies conducted in community-based settings (3.4%, 95% CI 2.1–4.7) was similar to that of clinic-based settings (5.7%, 95% CI 4.2–7.2). In contrast, there was a substantial difference in pooled prevalence between HIV-negative (1.5%, 95% CI 0.8–2.1) and HIV-positive (8.3%, 95% CI 6.7–9.9) men. Injecting drug use remained the major transmission route for HCV infection in the gay communities, as demonstrated by a 10-fold increase in pooled prevalence between non-IDUs (3.5%, 95% CI 2.5–4.5) and IDUs (34.8%, 95% CI 26.9–42.7). Approximately one-third of GBM who had a history of injecting drug use were HCV-positive regardless of their HIV status. In longitudinal studies, the pooled annual incidence was

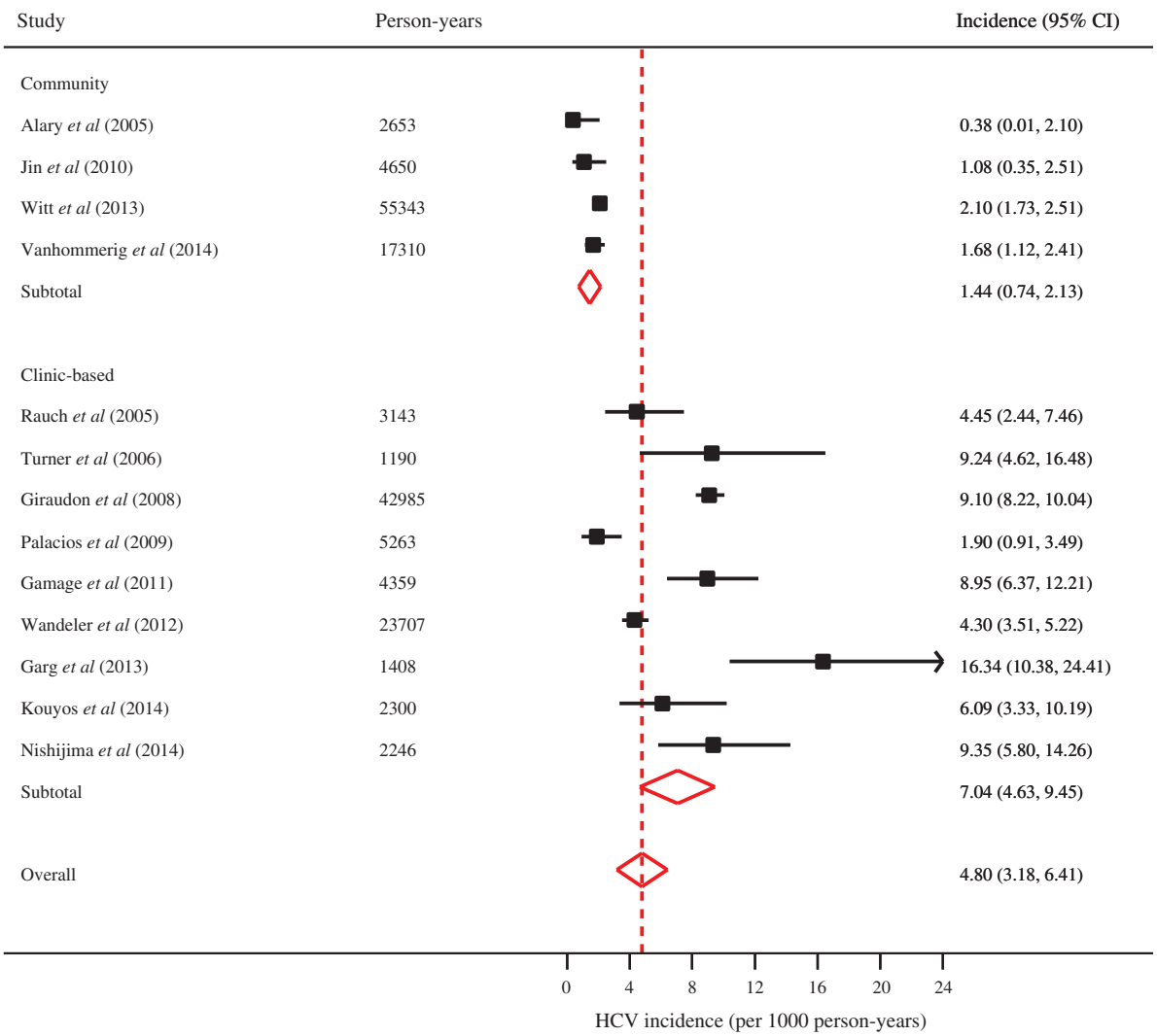

Fig. 5. Hepatitis C virus incidence in longitudinal studies involving gay and bisexual men stratified community- versus clinic-based recruitment.

just under 5 per 1000 person-years in GBM (4.8 per 1000 person-years, 95% CI 3.2–6.4), and HIV-positive men had a pooled incidence more than 15-fold higher than HIV-negative men (6.4 vs 0.4 per 1000 person-years). In case-series reports, since 2000, there has been a trend of increasing number of reports of clusters of mainly HIV-positive men who were likely to have acquired acute HCV infection through sexual contact. It was also likely that the acquisition could be further facilitated by the presence of ulcerative STIs, including syphilis and LGV. There have been a few recent reports of HIV-negative men who acquired acute HCV infection through sexual means, including four cases who were PrEP trial participants.

In HIV-negative GBM who do not report a history of injecting drug use, the prevalence of HCV is generally below 1%,[22,53,54] which is similar to or slight lower than that of the general population in corresponding industrialised countries.[55] Nevertheless, the large-scale Multicenter AIDS Cohort (MAC) study reported a HCV prevalence of 2.6% in its HIV-negative non-IDU participants after testing stored sera.[56] The majority of the participants of the MAC study were recruited in the mid-1980s, an era before the advent of HCV serological testing. It is likely that some HCV infections in HIV-negative participants could be attributable to blood transfusion.

In individuals who do not have immune-compromising conditions, the probability of acquiring HCV infection thorough sexual contact is generally low. In heterosexual couples, an Italian study followed 776 spouses of HCV-infected index cases for a mean of 10 years and reported three cases of incident HCV infection;[57] however, genotyping and phylogenetic tests indicated that none were transmitted from the index partners. One more recent cohort study in the US followed 500 HCV serodiscordant heterosexual couples for a total of 8377 person-years and recorded three within-couple HCV transmissions as confirmed by a phylogenetic test.[58] Although there are no cohort studies on HCV sexual transmission among male GBM couples, incident HCV infection rates reported by longitudinal studies of HIV-negative GBM have also been consistently low, with a pooled incidence of 0.4 per 1000 person-years.[22,32,35,36,59]

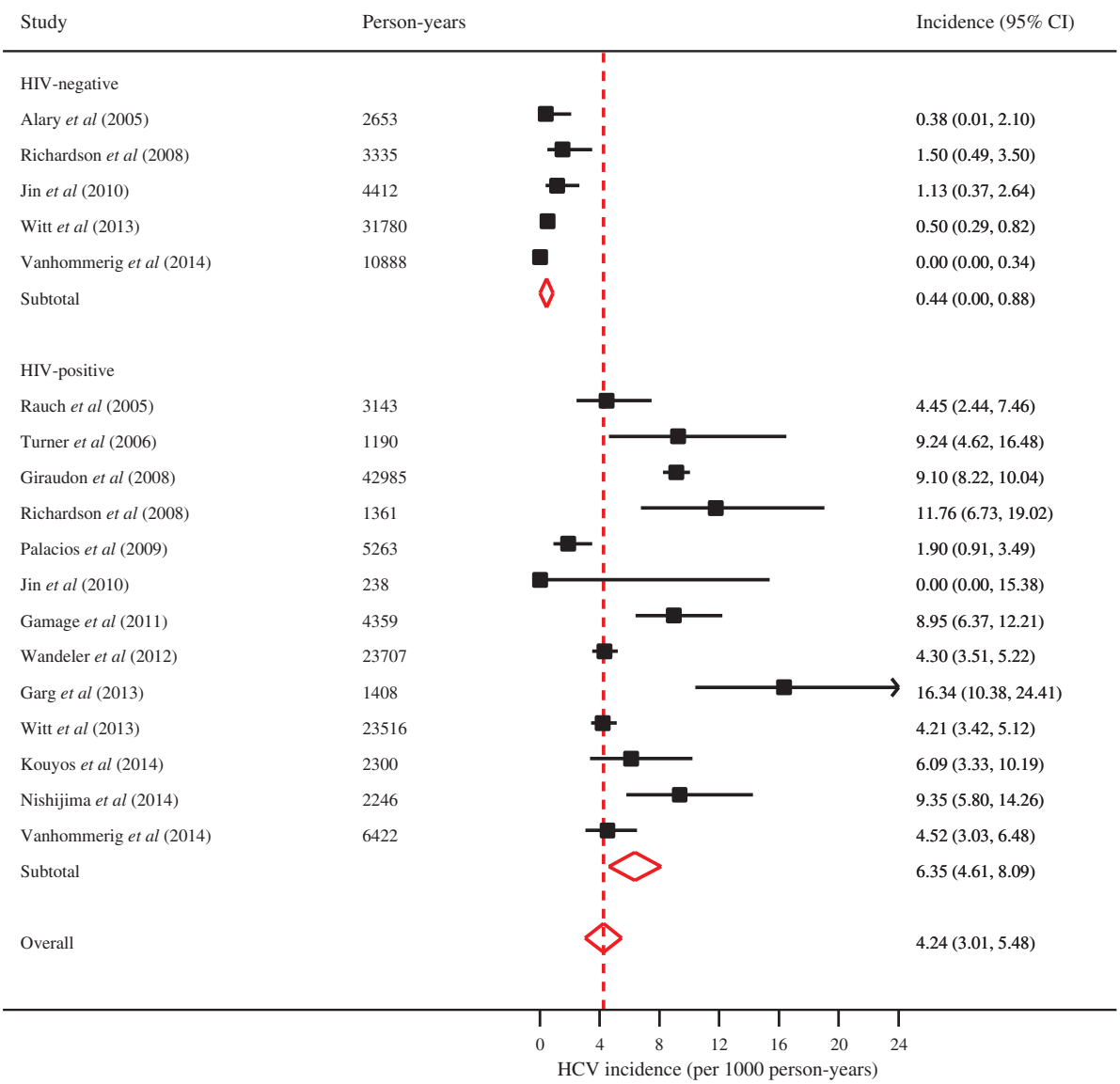

Fig. 6. Hepatitis C virus incidence in longitudinal studies involving gay and bisexual men stratified by HIV status.

The sexual transmission of HCV is complicated by HIV infection, either in the index or in the susceptible partner. In early cross-sectional studies of heterosexual couples in which the index partners were haemophiliac, HCV prevalence was higher in sexual partners whereby the index partner was also HIV-infected.[7] However, a more recent longitudinal study involving 171 heterosexual couples in which the index partner was HCV/HIV co-infected reported no cases of HCV acquisition.[60] This was despite 529 person-years of follow up and over 5800 episodes of condomless vaginal and anal intercourse. The role of partners' HIV status in relation to HCV acquisition in HIV-negative GBM is less clear. Although it was not statistically significant, four of five HCV seroconverters arising from a cohort of HIV-negative men in Sydney, Australia, reported sex with HIV-positive partners before HCV infection (hazard ratio 8.2, 95% CI 0.9–74.3).[22] Nevertheless, only one reported CAI with the HIV-positive partner. Biological evidence showed a higher seminal HCV viral load in HCV/HIV co-infected than in HCV mono-infected men and a lower HCV clearance rate in the co-infected,[12] and thus a higher likelihood of transmitting the virus to the susceptible partner. Two recent studies that investigated the role of HCV in semen demonstrated a high correlation between blood and semen HCV viral load and frequent seminal HCV viral shedding in HIV-positive GBM.[61,62]

Some longitudinal studies in Europe demonstrated an increase in HCV incidence in HIV-positive GBM since the late 1990s.[34,35,63,64] Most of those who acquired acute HCV infection did not report a history of injecting drug use,[27,41–50] with sexual contact being the mostly likely transmission route in these individuals. Behaviourally, this has coincided with the increasing practice of CAI and esoteric sexual practices, such as fisting, between partners of the same HIV status in the form of serosorting with the aim to minimise the HIV risk.[65,66]

Table 3. Case-series reports of acute hepatitis C virus (HCV) infection in gay and bisexual men between 2000 and 2015

First author	Year	City/Country	No. of acute HCV cases (*n*)	No. of HIV positive cases (*n*)	Non-injecting drug users (%)
Browne *et al.*[43]	2004	London, UK	26	25	92
Ghosn *et al.*[40]	2004	Paris, France	5	5	100
Gambotti *et al.*[44]	2005	Paris, France	29	29	100
Gotz *et al.*[46]	2005	Rotterdam, Netherlands	7	6	100
Matthews *et al.*[52]	2007	Australia	26	26	54
van de Laar *et al.*[50]	2007	Amsterdam, Netherlands	34	33	100[A]
Ghosn *et al.*[45]	2008	Paris, France	2	2	100
Bottieau *et al.*[42]	2010	Antwerp, Belgium	67	67	97
Barfod *et al.*[41]	2011	Copenhagen, Denmark	14	14	92.8
Montoya-Ferrer *et al.*[48]	2011	Madrid, Spain	4	4	100
CDC[27]	2011	New York, US	74	74	100
Sanchez *et al.*[49]	2013	Madrid, Spain	19	19	100
Mahony *et al.*[51]	2013	Melbourne, Australia	31	31	61
McFaul *et al.*[47]	2015	Europe	44	0	79.5
Volk *et al.*[16]	2015	San Francisco, US	2	0	100

[A]Among the 27 cases reviewed.

Biologically, this has also co-existed with a resurgence of ulcerative STIs, including syphilis and LGV, in HIV-positive communities in the same region.[67]

We have yet to see an increase in HCV incidence in HIV-negative GBM, but there have been a few recent case-series reports of acute HCV infection in this population, particularly in participants of PrEP trials.[16,47] Individuals receiving PrEP may decide not to use condoms with a male partner of the opposite HIV status. As an untoward consequence, rates of STIs could rise in HIV-negative men due to increases in anal intercourse between the HIV-negative and -positive men; that is, they are not protected by condom use.[15] Given that sexual transmission of HCV can occur in HIV-negative GBM men, this is of concern. In the context of PrEP, it is likely to be facilitated by increased sexual contact with HIV-positive partners in the form of CAI and in the presence of ulcerative STIs. While more evidence is needed to determine to what extent this might occur, surveillance should be in place to monitor HCV status in PrEP recipients. In its PrEP guidelines, the US Centres for Disease Control and Prevention acknowledges HCV risk in GBM and recommends HCV testing before PrEP is prescribed to document HCV status,[13] but no regular follow-up HCV tests are recommended once PrEP is initiated. In its most recent revision, PrEP guidelines for the Australian state of New South Wales recommend screening at baseline and during follow up in men who report potential risk factors for transmission.[68]

This review has focused on HCV risk in GBM in industrialised countries, so the results might be of limited generalisability in other settings. Even with this restriction on the scope of the review, substantial heterogeneities were observed in studies of HCV prevalence and incidence in GBM. In addition to injecting drug use, HIV status and study settings that have been examined in the analyses; other sources might have also contributed to the diversity of the rates observed between the studies, such as the geographic region and the age composition of the study population. Although injecting drug use remains the major transmission route for HCV infection, information on drug injecting was not always presented. A history of injecting drug use was generally self-reported by the study participants. As injecting drug use is probably associated with more stigma than CAI in the gay community, it is likely that the levels of drug injecting were underreported. This might have led to an underestimation of injecting drug use-associated HCV risk. Although all cases of HCV infection were confirmed by laboratory testing or medical records, various tests involved usually carry different performance characteristics. In older studies, HCV testing might be less sensitive than the ones used in more recent studies. It is also a possibility that studies that did not employ supplementary, more sensitive PCR testing might have missed cases of early HCV infection. In relation to ulcerative STIs as risk factors for HCV acquisition, particularly for the association with LGV, the conclusion relies on case-series reports. There have been no cross-sectional or longitudinal studies conducted in GBM to explicitly examine this association.

The risk for HCV infection in GBM who do not report a history of injecting drug use is generally low. Sexual transmission of HCV cannot be ruled out in this population, particularly in those with HIV infection in whom levels of CAI and rates of ulcerative STI are usually higher. In HIV-negative GBM, HCV sexual transmission can occur, and this could be further facilitated by the implementation of PrEP as a biomedical means for HIV prevention. For individual and public health considerations, HCV status should be determined before and monitored during PrEP use.

Conflicts of interest

None declared.

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