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Prevalence of hepatitis C virus infection among HIV+ men who have sex with men: a systematic review and meta-analysis

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Abstract

Since 2000, an increase in hepatitis C virus infection among HIV-infected (HIV+) men who have sex with men has been observed. Evidence points to blood exposure during sex as the medium of hepatitis C virus transmission. Hepatitis C virus prevalence among HIV+MSM overall and in relation to injection drug use is poorly characterized. In this study, a systematic review and meta-analysis examining global hepatitis C virus antibody prevalence and estimating active hepatitis C virus prevalence among HIV+MSM were conducted; 42 reports provided anti-hepatitis C virus prevalence data among HIV+MSM. Pooled prevalence produced an overall anti-hepatitis C virus prevalence among HIV+MSM of 8.1%; active HCV prevalence estimate was 5.3%–7.3%. Anti-hepatitis C virus prevalence among injection drug use and non-injection drug use HIV+MSM was 40.0% and 6.7%, respectively. Among HIV+MSM, hepatitis C virus prevalence increased significantly over time among the overall and non-injection drug use groups, and decreased significantly among injection drug use HIV+MSM. We identified a moderate prevalence of hepatitis C virus among all HIV+MSM and among non-injection drug use HIV+MSM; for both, prevalence was observed to be increasing slightly. Pooled prevalence of hepatitis C virus among HIV+MSM was higher than that observed in the 1945–1965 US birth cohort. The modest but rising hepatitis C virus prevalence among HIV+MSM suggests an opportunity to control HCV among HIV+MSM; this combined with data demonstrating a rising hepatitis C virus incidence highlights the temporal urgency to do so.

Keywords

Men who have sex with men; systematic review; meta-analysis; hepatitis C virus infection; HIV/AIDS; HIV/HCV co-infection

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Introduction

Men who have sex with men (MSM) are at substantial risk for HIV infection worldwide.^{1,2} In high-income countries, HIV epidemic trends are in decline, except among MSM.² There are significant HIV epidemics among MSM in many low- and middle-income countries; global HIV prevalence among MSM is estimated at 3–26%.²

One-third of HIV-infected persons is co-infected with hepatitis C virus (HCV) largely, because injection drug use (IDU) is both an HCV and HIV risk.³ HCV has become the leading cause of death among HIV-infected persons in high-income countries.⁴ Since 2000, an increase in the incidence of HCV infection among HIV+MSM has been observed.⁵ A recent systematic review and meta-analysis (SR/MA) estimated that the rate of sexually transmitted HCV in HIV+MSM was 0.53/100 person-years (PYs), and identified a small but statistically significant increase in incidence rates.⁵

Several factors may contribute to the excess incidence and prevalence of HCV among HIV +MSM, including biologic interactions of the two viruses, co-existent sexually transmitted infections (STIs), behavioral risks such as blood exposure during sex and sero-sorting in relation to HIV status, and overlaps between sexual and drug injection networks.^{6,7} HCV is readily transmitted by non-sterile IDU, but sexual transmission among heterosexuals has been shown to be very low.⁸ Research points to the presence of blood during high-risk sex as the primary means of HCV transmission among HIV+MSM.^{9,10} HCV viral loads are higher in semen and blood in the presence of HIV, and this may increase the likelihood of transmission.¹¹ Immunosuppressant effects of HIV may lead to a decreased likelihood of clearing HCV if exposed, and hence to higher rates of persistent infection.^{12,13} Mucosally ulcerative STIs, which are clearly associated with HIV transmission, might similarly facilitate HCV acquisition; associations between STIs and incidence of HCV in HIV+MSM have been observed.^{2,14,15}

HCV transmission among HIV+MSM has been associated both with unprotected sexual behaviors that may be both mucosally traumatic and likely to be associated with blood exposure.^{14,15} Sexual network characteristics may confer increased HCV transmission risk; since sero-concordance is generally sought on the basis of HIV rather than HCV status, this may facilitate HCV transmission.^{16,17} Further, HCV prevalence is usually high among people who inject drugs (PWID), and sexual networks of MSM may include MSM who also inject drugs, increasing the likelihood of HCV exposure.^{6,7,18,19} Non-IDU may also be associated with increased HCV acquisition risk through prolonged sexual activity or increased risk taking.¹⁵

The objective of this SR/MA was to synthesize the data on HCV prevalence among HIV +MSM worldwide, both overall and specifically among those who have or have not also injected drugs. The goal of conducting this analysis was to estimate the pool of HCV-infected HIV+MSM who may transmit HCV to uninfected HIV+MSM in sexual and/or drug using contexts, to estimate the proportions of HIV+MSM who may benefit from HCV treatment, and to inform modeling, HCV screening and preventive interventions, and public health control efforts.

Methods

Details of the methods for this SR/MA have been published elsewhere (International Register of Prospective Systematic Reviews, registration number CRD42013006462);²⁰ hence, a summary is provided here.^{21,22}

Search strategy

A comprehensive automated search for published literature using the electronic databases of PubMed, EMBASE, and BIOSIS was conducted; the search string comprised terms related to HCV, HIV/AIDS, MSM, and epidemiology. Reference lists and gray literature were manually reviewed for inclusion.

Inclusion criteria

To be eligible, all reports must have been written in English, been published from 1/1990–2/2015, and provided data on HCV prevalence among HIV+MSM based on HCV antibody (anti-HCV), recombinant immunoblot assays (RIBA), or tests for HCV RNA to confirm HCV infection. The overall pooled estimate included all reports, even those that did not distinguish between injection drug using (IDU) HIV+MSM and non-IDU HIV+MSM. Some reports specifically gave prevalence estimates for non-IDU HIV+MSM while other reports gave prevalence estimates for IDU HIV+MSM; each were also examined separately. Thus, there were three sets of data examined: (1) HIV+MSM regardless of known or unknown injection history; (2) non-IDU HIV+MSM; and (3) IDU HIV+MSM. These three groups allowed examination of the prevalence of HCV among HIV+MSM not directly exposed via IDU, the prevalence among MSM who had also injected, as well as the prevalence among all HIV+MSM who may be connected via sexual networks regardless of individual drug injection status.

Screening and coding

Review of potentially eligible reports was a multi-stage process as depicted in the PRISMA flow diagram (Figure 1). Geographic location, years of data collection, study design elements, study sample characteristics, and HCV prevalence were abstracted from each report. The project director (PD) and graduate level research assistant (RA) screened, reviewed, and coded reports; the principal investigator, PD, co-authors, and RA collectively resolved any issues that emerged during screening and coding.

Quality ratings

Included studies were assigned quality ratings using a standardized instrument adapted from the Newcastle-Ottawa Scale (NOS).²³ Emphasis was placed on the risk of selection bias due to sampling method; reports were classified as being of higher quality if they employed consecutive or systematic sampling or of lower quality if they utilized convenience sampling.

Estimates provided in reports for individuals “currently”, “recently”, or “ever” having injected drugs were considered as a group; only reports that explicitly stated that participants had never injected drugs were included in the group of non-IDU HIV+MSM. Reports

providing estimates that either did not state IDU histories explicitly, provided histories of drug use without specifying the route of drug use, or did not clearly distinguish IDU from non-IDU were only included in the overall pooled estimate.

Data analysis

The first stage of data analysis was to determine an overall pooled HCV prevalence estimate based on all included studies, for non-IDU HIV+MSM, and for IDU HIV+MSM, respectively. Analyses were also conducted specifically of reports from the US to allow comparisons with recent data on the prevalence of HCV among those in the US cohort born between 1945 and 1965. Pooled prevalence estimates were calculated using a random effects model; 95% confidence intervals (CIs) were calculated around these estimates assuming a normal distribution. To determine whether observed heterogeneity across estimates was compatible with chance, Cochrane's Q statistic was calculated; I^2 was calculated to quantify inconsistency in prevalence measures. Analyses were done in R (CRAN R Project 2015) and SPSS (SPSS, Inc., Chicago IL).

Data on potential covariates were extracted; this was limited by low reporting of covariates and lack of consistency in covariate reporting across studies, including overlap and variability in recruitment location characteristics. However, calendar time, geographic region, and selection bias were available and examined as study-level moderators.

When reports presented HCV prevalence in relation to participants enrolled over a span of two to four years, we assigned the temporal midpoint of the data collection period to the prevalence rate. This allowed us to include these estimates, along with estimates reported in individual years, in moderator analyses; the analysis of time as a moderator of prevalence included 19 estimates of non-IDU HIV+MSM and 9 estimates of IDU HIV+MSM.

Estimating prevalence of active HCV infection

We estimated the prevalence of active HCV infection among the three groups (HIV+MSM overall, non-IDU HIV+MSM, and IDU HIV+MSM) using rates of HCV clearance among HIV+MSM and among PWID without HIV infection calculated in another SR/MA from this project.¹³ Prevalence estimates of chronic infection were calculated by multiplying the pooled prevalence rates by the clearance rate. Three estimates of the prevalence of active HCV infection were made for each group (Table 2).

1. A range of prevalence of active HCV infection using the range of the pooled prevalence for each group, and the upper and lower bounds of the relevant HCV clearance rate.
2. A minimum prevalence of active HCV infection by using the lower bound of the 95% CI of the estimated anti-HCV prevalence and the upper bound of the relevant HCV clearance rate.
3. A maximum prevalence of active HCV infection by using the upper bound of the 95% CI of the estimated anti-HCV prevalence and the lower bound of the relevant HCV clearance rate.

In a recent SR/MA, HCV clearance rates identified among HIV-PWID (25.7% [95% CI 16.4–35.0%]) were higher than those identified among either HIV + PWID (16.1% [95% CI 12.5–19.6%]) or among HIV+MSM (15.4% [95% CI 11.5–19.3%]).¹³ The relevant HCV clearance rates used in our estimates of active infection rates were as follows:

- a. For non-IDU HIV+MSM, since the incidence of sexually transmitted HCV among HIV–MSM is generally quite low, we assumed that HIV infection preceded HCV infection.²⁴ We therefore used the rates of HCV clearance among HIV+MSM identified in the recent SR/MA (11.5%–19.3%).
- b. For IDU HIV+MSM, and for the overall group of HIV+MSM (including IDU HIV+MSM, non-IDU HIV+MSM, and those of unknown IDU status) it cannot be assumed that HIV infection preceded HCV infection. While HCV infection may generally precede HIV infection among PWID, among IDU HIV+MSM whose individual risk behavior histories are not clearly known, and in groups reflecting variable and uncertain proportions of IDU and non-IDU HIV+MSM, it also cannot be assumed that HCV infection preceded HIV infection. Therefore, for both the overall group of HIV+MSM and IDU HIV+MSM, we estimated the minimum prevalence of active HCV by applying the upper bound of the 95% CI of the highest clearance rate identified (i.e. the 35% rate of HCV clearance identified among HIV-negative PWID), and estimated the maximum prevalence of active HIV by applying the lower bound of the 95% CI of the lowest rate of clearance identified (i.e. the 11.5% rate of HCV clearance identified among HIV +MSM).

Results

General

The literature search generated a total of 1049 potentially eligible abstracts; after duplicates and ineligible abstracts were removed, 188 were reviewed as full-text articles. Reasons for ineligibility can be found in Figure 1. A final set of 42 reports presenting HCV prevalence among HIV+MSM yielded 43 HCV prevalence estimates.^{10,14,25–62} Eleven reports provided 12 estimates for IDU HIV+MSM^{10,25,33,35,37,40,42,51,52,55,63} and 26 reports provided 27 estimates for non-IDU HIV+MSM^{10,14,25–29,33,35,37,39,40,42,45,47,49–52,54,56,57,59,63} (Table 1(a) to (c)).

Reports represented moderate geographic variation; half of reports were from Europe, 23.4% from North America (n=0, 6 from the US), and 16.7%, 4.8%, and 9.5% from Asia, South America, and Australia, respectively. All reports were from high-income countries except for five from upper middle-income countries. Only three of 42 reports used both anti-HCV and viral load to ascertain HCV infection (Table 1(a)).

Prevalence of HCV infection

Using a random effects model, pooled HCV prevalence among all 42 included reports (which included known IDU, known non-IDU, and those of unknown IDU status) was 8.2% (95% CI 6.6–9.7%). (Table 2, Figure 2). Significant heterogeneity was present in the overall

pooled prevalence estimates ($I^2=97.3\%$; Q -statistic=778.4, $p < .0001$). Among non-IDU HIV +MSM, pooled HCV prevalence was 6.7% (95% CI 5.3–8.1) (Table 2, Figure 3); heterogeneity was shown by a Q statistic of 308.5 ($p < .0001$) and an I^2 value of 94.1%. Among IDU HIV+MSM, pooled HCV prevalence was 40.0% (95% CI 28.0–51.0%) with a range from 17.9% to 80% (Table 2, Figure 4); heterogeneity was present ($Q=50.4$ [$p < .0001$] and $I^2 = 91.2\%$).

The overall pooled HCV prevalence for HIV+MSM among US-based reports was 12% (95% CI 8.5–15.4%); the range was 6.0–17.9%. The pooled HCV prevalence among US-based reports for non-IDU HIV+MSM and IDU HIV+MSM was 7.51% (95% CI 5.2–9.9%) and 35.6% (95% CI 21.1–50.1%), respectively.

Moderators of prevalence of HCV infection

Among all HIV+MSM, HCV prevalence in the earliest year of data collection (1988) was 2.9%;⁴⁹ in 2011, the latest year of data collection, the prevalence of HCV was 15.7%.⁵² Meta-regression identified a statistically significant increase in HCV prevalence among all HIV+MSM, increasing 0.334% per year ($p < .000$). A statistically significant increase in HCV prevalence was also observed among non-IDU HIV+MSM among whom prevalence increased by 0.367% per year ($p < .000$). However, HCV prevalence was shown to decrease among IDU HIV+MSM (−1.44% per year, $p < .000$).

Geographic variation was not a significant predictor of HCV prevalence among all HIV +MSM ($P = 0.390$) or any of the subgroups of interest.

Study quality was a statistically significant predictor of HCV prevalence, with consecutive or systematic sampling (vs. convenience sampling) being associated with a higher HCV prevalence ($p < .000$).

Prevalence of active HCV infection

The estimated range of prevalence of active HCV infection among all HIV+MSM was 5.3%–7.3%. The estimated range of prevalence of active HCV infection among non-IDU HIV+MSM was 4.9%–5.9%. Among IDU HIV+MSM, the estimated range of prevalence of active HCV infection was 26%–35.4%. Minimum and maximum estimated prevalence ranges are presented in Table 2.

Discussion

This SR included 38,986 unique HIV+MSM enrolled in 42 studies. We identified a moderate prevalence of HCV among all HIV+MSM and among non-IDU HIV+MSM; for both groups, the prevalence rate was observed to be increasing slightly. Among IDU HIV +MSM, we observed a high, yet slightly decreasing prevalence of HCV.

The prevalence rates identified among non-IDU HIV+MSM and HIV+MSM were substantially lower than among PWID or IDU HIV+MSM. However, even the lower bound of the pooled prevalence among HIV+MSM in the U.S (8.5%) is higher than that among US

residents in the 1945–1965 birth cohort, for whom an identified 3.25% (95% CI 2.80–3.80%) prevalence has led to recommendations for focused screening.⁶⁴

Recent evidence suggests a small but statistically significant increase in incidence of HCV among non-IDU HIV+MSM.⁶ While our data are based on reports of anti-HCV, our finding of a rising prevalence is consistent with the fact that for an infection that remains chronic in the majority of untreated persons, even a stable incidence has the potential to result in a rising prevalence (unless losses [e.g. deaths] outpace new infections).

Our finding of a 40% pooled prevalence of HCV among IDU HIV+MSM was consistent with prevalence rates identified in cohort studies and meta-analyses among PWID generally (43–85%).^{65,66} The observation that the pooled HCV prevalence was higher among HIV+MSM who have also injected than among those who have not is consistent with the efficiency of HCV transmission by non-sterile injection. It also highlights that MSM who inject may serve as a key bridge population between high-prevalence PWID and HIV+MSM who do not inject but do engage in blood-involved sexual behaviors.

IDU MSM have been recognized as an important bridge population in the trajectory of the HIV epidemic, through the combination of two highly efficient HIV transmission behaviors, and through complex social networks that include non-IDU MSM.⁶⁷ IDU MSM have constituted a decreasing proportion of new HIV diagnoses in the US (from 8% in 1990 to 3% in 2013), this decrease has occurred in temporal conjunction with a very significant decrease in the prevalence of HIV among PWID during that same time frame.^{67,68} In contrast, during the same period, there has been only a modest decrease in HCV prevalence among PWID in some settings; our finding of a modest decrease in HCV among IDU HIV+MSM is consistent with these trends.⁶⁸ Nonetheless, the prevalence of HCV among PWID has remained high during this same time period in many recent estimates.^{18,65} The 2011 US National Health and Behavior Survey found that 5% of MSM had a history of drug injection.⁶⁹ Thus, while the overlap between injecting and non-injecting MSM may be small, the high prevalence of HCV among PWID makes this a critical bridge population in the epidemiology of HCV among HIV+MSM, highlighting the need for effective combined prevention addressing both drug use and sexual risks.^{18,19}

The pooled prevalence, the apparent rise in prevalence over time, and the estimated rates of active HCV infection support existing recommendations to screen HIV+MSM for HCV. A recent SR/MA showed that 22% of incident HCV infections in HIV+MSM were attributable to unprotected anal intercourse and 36% were related to non-IDU associated with sex.⁵ Similarly, several papers identified in the current SR/MA reported on sexual risk behaviors among HIV+MSM, but either did not do so with comparably defined variables or did not present data among HCV prevalent cases both precluding further formal meta-analyses.^{10,14,41,59}

While active HCV infection is generally associated with high blood HCV viral loads, HCV has been detected intermittently in the semen of a minority (but up to 42%) of HCV/HIV co-infected men, albeit with HCV viral loads significantly lower than those found in blood.^{11,70–74} Unprotected receptive anal intercourse with ejaculation has been associated

with new HCV infection in a case-control study with a high odds ratio although without mention of whether there was associated blood exposure.⁷⁴ The extent to which transmission is attributable to HCV virus in semen or to blood exposure is not entirely clear and requires further study. Consequently, for all HIV+MSM, enhanced sexual risk reduction should include both condom use and avoiding blood exposure during sex as important components of combined HCV prevention.^{15,75} For HIV+MSM who inject drugs, ensuring access to sterile syringes and to evidence-based drug treatment including methadone and buprenorphine are likely to be important components of efforts to prevent HCV acquisition and transmission including those for HIV+MSM.^{76,77} Effective strategies to reduce or make safer the use of non-injection drugs associated with high-risk sex are also needed.

Interventions to improve the retention of HCV-infected MSM in the HCV care continuum, including increased engagement in HCV treatment through to sustained viral responses, will be key to improving individual outcomes and reducing community viral load to facilitate control of the epidemic.⁷⁸ However, a meta-analysis has found pooled re-infection rates following successful HCV treatment to be 11.4 per 100 PYs, suggesting that additional post-treatment interventions to prevent re-infection may be needed.⁵

Limitations

There are several important limitations to this study. The generalizability and validity of our pooled estimates are dependent on the quality of study-level measurements of the relationship between exposures and prevalent HCV infection. Of note, however, higher quality reports were associated with a higher HCV prevalence. There was some potential for misclassification as to MSM and/or IDU status, and, again, we were limited by the precision in MSM and/or IDU ascertainment in the original studies. As several reports were not explicit with respect to IDU status, we specifically examined prevalence among those reports that clearly distinguished IDU from non-IDU as separate groups. The overall pooled prevalence group includes persons of both known and unknown injection histories and thus this prevalence estimate must be understood as reflecting an undefined proportion of IDU HIV+MSM.

Moderator analysis of calendar time relied on interpolation for some of the estimates, which may have overestimated the precision of observed trends in HCV prevalence over time. However, the observation that HCV prevalence among HIV+MSM has increased over time is congruent with studies reporting an increase in HCV incidence among HIV+MSM.²² Data were not available for many geographic settings, particularly lower middle- or low-income countries, limiting generalizability. Whether the observed lack of apparent geographic variation is due to similarities in prevalent risk behaviors, to bridging between international populations leading to similar rates, or to an artifact due to sampling or lack of representativeness of the countries studied in particular to the paucity of data from low- and middle-income countries is not clear and requires further study.

Our estimates of rates of active infection from anti-HCV data are only as reliable as the estimates of rates of HCV clearance; nonetheless, the approach we took to such estimates used the best available data from a current SR/MA and provides conservative estimates.

Conclusion

While combined prevention has successfully reduced the incidence and prevalence of HIV among PWID in many settings, there has been a lesser degree and less consistent impact of combined prevention on HCV incidence and prevalence among PWID.^{77,79} It is in this setting that rising incidence and prevalence rates of HCV among HIV+MSM have been observed. Both modeling and experience with public health efforts in various cities and countries to reverse and end high-prevalence HIV and HCV epidemics demonstrate that the higher the baseline prevalence, the greater the public health effort and longer time required to achieve epidemic control, let alone infection elimination.^{80,81} The finding of a still modest, but relevant and rising, HCV prevalence among HIV+MSM suggests that there is still the opportunity to control HCV among HIV+MSM; this combined with data demonstrating a rising HCV incidence highlights the temporal urgency to do so.^{5,82}

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Figure 1.
PRISMA flow of information from identification to inclusion of studies.

Figure 2.
Forest plot of HCV Prevalence among HIV+MSM overall.

Figure 3.
Forest plot of HCV Prevalence among non-IDU HIV+MSM.

Figure 4.
Forest plot of HCV Prevalence among IDU HIV+MSM.

Table 1
Prevalence of hepatitis C virus (HCV) infection among HIV + men who have sex with men: (a) Overall and (b) Non-IDU (c) IDU.

First author, publication year	Location	Study years	Recruitment method	Recruitment setting	HCV test (anti-HCV, RIBA/RNA)	No. HCV positive	Sample size	Prevalence (%)	95% CI (LL)	95% CI (UL)
(a). Overall										
Amin et al. ²⁵	Multiple	1995–1996	Systematic	n/s	Anti-HCV	18	951	1.9%	1.1%	3.0%
Barford et al. ²⁶	Denmark	2006–2009	Consecutive	HIV clinic	Anti-HCV	41	871	4.7%	3.4%	6.3%
D'Oliveira et al. ²⁷	Greece	1986–1997	Consecutive	HIV clinic	Anti-HCV	81	1431	5.7%	4.5%	7.0%
Dimitrakopoulos et al. ²⁸	France	1992–2002	Consecutive	Hospital	Anti-HCV	10	123	8.1%	4.0%	14.4%
Edelenyi-Pinto et al. ²⁹	Brazil	1990–1991	Convenience	Research study	Anti-HCV	9	113	8.0%	3.7%	14.6%
Fainboim et al. ³⁰	Argentina	1994–1995	Convenience	Hospital	Anti-HCV	14	99	14.1%	8.0%	22.6%
Filippini et al. ³¹	Italy	1996–2001	Convenience	HIV clinic	Anti-HCV	3	29	10.3%	2.2%	27.4%
Gamage et al. ³²	Australia	2002–2010	Consecutive	HIV clinic	Anti-HCV	109	869	12.5%	10.4%	14.9%
Garg et al. ³³	U.S.	1997–2009	Convenience	CBO	Anti-HCV	64	1059	6.0%	4.7%	7.7%
Heiligenberg et al. ³⁴	Netherlands	2007–2008	Convenience	HIV clinic	Anti-HCV	27	649	4.2%	2.8%	6.0%
Jin et al. ³⁵	Australia	2005–2007	Convenience	HIV clinic; hospital	Anti-HCV	2	161	1.2%	0.2%	4.4%
Johns and Gill ³⁶	Canada	1985–1997	Consecutive	HIV clinic	Anti-HCV	10	167	6.0%	2.9%	10.7%
Kim et al. ³⁷	U.S.	1999–2007	Consecutive	HIV clinic	Anti-HCV	225	1652	13.6%	12.0%	15.4%
Larsen et al. ³⁸	France	2004	Convenience	Hospital	Anti-HCV	17	558	3.0%	1.8%	4.8%
Lee et al. ³⁹	Taiwan	2000–2005	Consecutive	Hospital	Anti-HCV	6	113	5.3%	2.0%	11.2%
Lincoln et al. ⁴⁰	Australia	1999–2002	Consecutive	STI, HIV clinics	Anti-HCV	158	1338	11.8%	10.1%	13.7%
Matser et al. ⁴¹	Netherlands	2008–2009	Not specified	STI clinic	Anti-HCV	108	850	12.7%	10.5%	15.1%
Matser et al. ⁴	Netherlands	2008–2009	Convenience	STI, HIV clinics	Anti-HCV	91	784	11.6%	9.5%	14.1%
Mohsen et al. ⁴²	U.K.	2000–2002	Convenience	HIV clinic	Anti-HCV	13	375	3.5%	1.9%	5.9%
Nishijima et al. ⁴³	Japan	2005–2010	Convenience	HIV clinic	Anti-HCV	22	1068	2.1%	1.3%	3.1%
Onsetti et al. ⁴⁴	Italy	1989–2011	Consecutive	HIV clinic	Anti-HCV	5	80	6.3%	2.1%	14.0%
Palacios et al. ⁴⁵	Spain	2006	Consecutive	Hospital	Anti-HCV	61	725	8.4%	6.5%	10.7%
Price et al. ⁴⁶	U.K.	2008	Convenience	MSM social venues	Anti-HCV	13	168	7.7%	4.2%	12.9%
Puig-Basagoti et al. ⁴⁷	Spain	1993–1997	Not specified	n/s	Anti-HCV	6	39	15.4%	5.9%	30.5%
Quan et al. ⁴⁸	Canada	1990–1992	Convenience	Health clinic	Anti-HCV/RIBA/RNA	8	186	4.3%	1.9%	8.3%

First author, publication year	Location	Study years	Recruitment method	Recruitment setting	HCV test (anti-HCV, RIBA/RNA)	No. HCV positive	Sample size	Prevalence (%)	95% CI (LL)	95% CI (UL)
Quaranta et al. ⁴⁹	France	1988–1991	Convenience	HIV and hospital clinics; CBOs	Anti-HCV	2	68	2.9%	0.4%	10.2%
Rauch et al. ⁵⁰	Switzerland	2000–2004	Consecutive	Public health center; HIV and other clinics	Anti-HCV	108	2550	4.2%	3.5%	5.1%
Raymond et al. ⁵¹	U.S.	2004	Systematic	MSM CBO and social settings	Anti-HCV	14	92	15.2%	8.6%	24.2%
Raymond et al. ⁵¹	U.S.	2008	Systematic	MSM CBO and social settings	Anti-HCV	10	117	8.5%	4.2%	15.2%
Raymond ⁵²	U.S.	2011	Systematic	MSM CBO and social settings	Anti-HCV	17	108	15.7%	9.5%	24.0%
Rivas-Estilla et al. ⁵³	Mexico	2003–2004	Convenience	Health clinic; CBO	Anti-HCV, RIBA/RNA	II	61	18.0%	9.4%	30.0%
Roca et al. ⁵⁴	Spain	1997–2002	Convenience	HIV clinic	Anti-HCV	59	509	11.6%	8.9%	14.7%
Sulkowski et al. ⁵⁵	U.S.	1995–2001	Convenience	HIV clinic	Anti-HCV	100	560	17.9%	14.8%	21.3%
Sun et al. ⁵⁶	Taiwan	1994–2010	Not specified	Health clinic	Anti-HCV	102	1576	6.5%	5.3%	7.8%
Tseng et al. ⁵⁷	Taiwan	2009–2010	Convenience	Hospital	Anti-HCV	24	434	5.5%	5.4%	8.1%
Turner et al. ⁵⁸	U.K.	1996–2007	Consecutive	STI clinic; hospital	Anti-HCV	863	12059	7.2%	6.7%	7.6%
Urbanus et al. ¹⁰	Netherlands	2007–2008	Convenience	STI clinic	Anti-HCV	28	157	17.8%	12.2%	24.7%
Van de Laar et al. ⁵⁹	Netherlands	1984–2003	Convenience	STI clinic; hospital; social venues for MSM	Anti-HCV, RIBA/RNA	13	504	2.6%	1.4%	4.4%
Vogt et al. ⁶³	U.S.	1989–1993	Systematic	HIV clinic	Anti-HCV	45	486	9.3%	6.8%	12.2%
Wandeler et al. ¹⁴	Switzerland	1998–2001	Convenience	HIV clinic	Anti-HCV	147	4629	3.2%	2.7%	3.7%
Zhang et al. ⁶²	China	2005–2006	Convenience	Health clinic	Anti-HCV	3	16	18.8%	4.0%	45.7%
Zhao et al. ⁶¹	China	2000–2010	Convenience	HIV and hospital clinics; CBOs	Anti-HCV	56	230	24.3%	19.0%	30.4%
Zhou et al. ⁶⁰	Asia, Australia	2003–2005	Convenience	HIV and hospital clinics; CBOs	Anti-HCV	23	372	6.2%	4.0%	9.1%
(b). Non IDU										
Amin et al. ²⁵	Multiple	1995–1996	Systematic	n/s	Anti-HCV	31	920	3.4%	2.3%	4.8%
Barfod et al. ²⁶	Denmark	2006–2009	Consecutive	HIV clinic	Anti-HCV	41	871	4.7%	3.4%	6.3%
D'Oliveira et al. ²⁷	Greece	1986–1997	Consecutive	HIV clinic	Anti-HCV	81	1431	5.7%	4.5%	7.0%
Dimitrakopoulos et al. ²⁸	France	1992–2002	Consecutive	Hospital	Anti-HCV	10	123	8.1%	4.0%	14.4%
Edelenyi-Pinto et al. ²⁹	Brazil	1990–1991	Convenience	Research study	Anti-HCV	9	113	8.0%	3.7%	12.6%
Garg et al. ³³	U.S.	1997–2009	Convenience	CBO	Anti-HCV	49	965	5.1%	3.8%	6.7%
Jin et al. ³⁵	Australia	2005–2007	Convenience	HIV clinic; hospital	Anti-HCV	2	161	1.2%	0.2%	442.0%
Kim et al. ³⁷	U.S.	1999–2007	Consecutive	HIV clinic	Anti-HCV	162	1556	10.4%	8.9%	12.0%
Lee et al. ³⁹	Taiwan	2000–2005	Consecutive	Hospital	Anti-HCV	6	113	5.3%	2.0%	11.2%
Lincoln et al. ⁴⁰	Australia	1999–2002	Consecutive	STI, HIV clinics	Anti-HCV	107	1236	8.7%	7.1%	10.4%

First author, publication year	Location	Study years	Recruitment method	Recruitment setting	HCV test (anti-HCV, RIBA/RNA)	No. HCV positive	Sample size	Prevalence (%)	95% CI (LL)	95% CI (UL)
Matser et al. ⁴¹	Netherlands	2008–2009	Not specified	STI clinic	Anti-HCV	108	850	12.7%	10.5%	15.1%
Matser et al. ⁴¹	Netherlands	2008–2009	Convenience	STI, HIV clinics	Anti-HCV	91	784	11.6%	9.5%	14.1%
Mohsen et al. ⁴²	U.K.	2000–2002	Convenience	HIV clinic	Anti-HCV	13	375	3.5%	1.9%	5.9%
Palacios et al. ⁴⁵	Spain	2006	Consecutive	Hospital	Anti-HCV	61	725	8.4%	6.5%	10.7%
Puig-Basagoiti et al. ⁴⁷	Spain	1993–1997	Not specified	n/s	Anti-HCV	6	39	15.4%	5.9%	30.5%
Quaranta et al. ⁴⁹	France	1988–1991	Convenience	HIV and hospital clinics; CBOs	Anti-HCV	2	68	2.9%	0.4%	10.2%
Rauch et al. ⁵⁰	Switzerland	2000–2004	Consecutive	Public health center; HIV/other clinics	Anti-HCV	108	2550	4.2%	3.5%	5.1%
Raymond et al. ⁵¹	U.S.	2004	Systematic	MSM CBO and social settings	Anti-HCV	6	69	8.7%	3.3%	18.0%
Raymond et al. ⁵¹	U.S.	2008	Systematic	MSM CBO and social settings	Anti-HCV	4	82	4.9%	1.3%	12.0%
Raymond et al. ⁵²	U.S.	2011	Systematic	MSM CBO community and social settings	Anti-HCV	9	73	12.3%	5.8%	22.1%
Roca et al. ⁵⁴	Spain	1997–2002	Convenience	HIV clinic	Anti-HCV	59	509	11.6%	8.9%	14.7%
Sun et al. ⁵⁶	Taiwan	1994–2010	Not specified	Health clinic	Anti-HCV	102	1576	6.5%	5.3%	7.8%
Tseng et al. ⁵⁷	Taiwan	2009–2010	Convenience	Hospital	Anti-HCV	24	434	5.5%	3.6%	8.1%
Urbanus et al. ¹⁰	Netherlands	2007–2008	Convenience	STI clinic	Anti-HCV	23	143	16.1%	10.5%	23.2%
Van de Laar et al. ⁵⁹	Netherlands	1984–2003	Convenience	STI clinic; hospital; MSM social venues	Anti-HCV, RIBA/RNA	13	504	2.6%	1.4%	4.4%
Vogt et al. ⁶³	U.S.	1989–1993	Systematic	HIV clinic	Anti-HCV	29	442	6.6%	4.4%	9.3%
Wandeler et al. ¹⁴	Switzerland	1998–2001	Convenience	HIV clinic	Anti-HCV	147	4629	3.2%	2.7%	3.7%
Amin et al. ²⁵	Multiple	1995–1996	Systematic	n/s	Anti-HCV	8	10	80.0%	44.4%	97.5%
Garg et al. ³³	U.S.	1997–2009	Convenience	CBO	Anti-HCV	12	21	57.1%	34.0%	78.2%
Jin et al. ³⁵	Australia	2005–2007	Convenience	HIV clinic; hospital	Anti-HCV	16	68	23.5%	14.9%	35.4%
Kim et al. ³⁷	U.S.	1999–2007	Consecutive	HIV clinic	Anti-HCV	63	96	65.6%	55.2%	75.0%
Lincoln et al. ⁴⁰	Australia	1999–2002	Consecutive	STI and HIV clinics	Anti-HCV	51	102	50.0%	39.9%	60.1%
Mohsen et al. ⁴²	U.K.	2000–2002	Convenience	HIV clinic	Anti-HCV	5	9	55.6%	21.2%	86.3%
Raymond et al. ⁵¹	U.S.	2004	Systematic	MSM CBOs and social settings	Anti-HCV	8	23	34.8%	16.4%	57.3%
Raymond et al. ⁵¹	U.S.	2008	Systematic	MSM CBOs and social settings	Anti-HCV	6	33	18.2%	7.0%	35.5%
Raymond et al. ⁵²	U.S.	2011	Systematic	MSM CBOs and social settings	Anti-HCV	8	35	22.9%	10.4%	40.1%
Sulkowski et al. ⁵⁵	U.S.	1995–2001	Convenience	HIV clinic	Anti-HCV	100	560	17.9%	14.8%	21.3%
Urbanus et al. ¹⁰	Netherlands	2007–2008	Convenience	STI clinic	Anti-HCV	5	14	35.7%	12.8%	64.9%
Vogt et al. ⁶³	U.S.	1989–1993	Systematic	HIV clinic	Anti-HCV	16	44	36.4%	22.4%	52.2%

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HCV: hepatitis C virus; IDU: injection drug use; MSM: men who have sex with men; CI: confidence interval; CBO: Community-based organization; STI: Sexually transmitted infection.

Table 2

Estimated prevalence of active HCV infection among HIV + MSM.

	Pooled HCV antibody prevalence (%)	95% confidence intervals for HCV antibody prevalence	Estimated prevalence of active HCV infection (%) ^a	Minimum estimated prevalence of active HCV infection (%) ^b	Maximum estimated prevalence of active HCV infection (%) ^c
HIV+MSM, <i>overall</i>	8.2	Lower bound:6.6 Upper bound:9.7	5.3 ^d -7.3 ^e	4.3 ^d	8.6 ^e
HIV+MSM, non-IDU	6.7	Lower bound:5.3 Upper bound:8.1	4.9 ^f -5.9 ^e	4.3 ^e	7.2 ^e
HIV+MSM, IDU	40.0	Lower bound:28.0 Upper bound:51.0	26.0 ^d -35.4 ^e	18.2 ^d	45.0 ^e

HCV: hepatitis C virus; IDU: injection drug use; MSM: men who have sex with men.

^aLower end of the prevalence range of active HCV infection = (estimated prevalence of anti-HCV) × (1 – lower bound of estimated HCV clearance rate) and upper end of the prevalence range of active HCV infection = (estimated prevalence of anti-HCV) × (1 – upper bound of estimated HCV clearance rate).^bMinimum estimated prevalence of HCV infection = (lower bound of the 95% CI of estimated anti-HCV prevalence) × (1 – upper bound of 95% CI of estimated clearance rate).^cMaximum estimated prevalence of HCV infection = (upper bound of the 95% CI of estimated HCV antibody prevalence of anti-HCV) × (1 – lower bound of 95% CI of estimated clearance rate).^dUsing an estimated clearance proportion of 35.0%.^eUsing an estimated clearance proportion of 11.5%.^fUsing an estimated clearance proportion of 19.3%.