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A meta-analysis of the hepatitis C virus distribution in diverse racial/ethnic drug injector groups

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Abstract

Hepatitis C virus (HCV) is mostly transmitted through blood-to-blood contact during injection drug use via shared contaminated syringes/needles or injection paraphernalia. This paper used meta-analytic methods to assess whether HCV prevalence and incidence varied across different racial/ethnic groups of injection drug users (IDUs) sampled internationally. The 29 prevalence and 11 incidence studies identified as part of the HCV Synthesis Project were categorized into subgroups based on similar racial/ethnic comparisons. The effect estimate used was the odds or risk ratio comparing HCV prevalence or incidence rates in racial/ethnic minority groups versus those of majority status. For prevalence studies, the clearest disparity in HCV status was observed in the Canadian and Australian Aboriginal versus White comparison, followed by the US non-White versus White categories. Overall, Hispanic IDUs had greater HCV prevalence, and HCV prevalence in African-Americans was not significantly greater than that of Whites in the US. Aboriginal groups showed higher HCV seroconversion rates when compared to others, and African-Americans had lower seroconversion rates compared to other IDUs in the US. The findings suggest that certain minority groups have elevated HCV rates in comparison to other IDUs, which may be a consequence of stigma, discrimination, different risk behaviors or decreased access to health care, services and preventive education. Future research should seek to explicitly explore and explain racial/ethnic variations in HCV prevalence and incidence, and define the groups more precisely to allow for more accurate detection of possible racial/ethnic differences in HCV rates.

Keywords

Hepatitis C virus (HCV); Injection drug use (IDU); Race/ethnicity; Meta-analysis; Health disparities; Review

Hepatitis C virus (HCV) is hyperendemic in injection drug users (IDUs) (Hagan, 1998) and its prevalence in this population can reach levels of over 90% (McCarthy & Flynn, 2001). Chronic HCV can lead to cirrhosis, hepatocellular carcinoma, end-stage liver disease and death (Sulkowski & Thomas, 2005). HCV is transmitted most frequently through blood-to-blood contact during injection drug use (Alter & Moyer, 1998; Estrada, 2002) via sharing of contaminated syringes/needles (Hagan et al., 2001; Hahn, Page-Shafer, Lum, Ochoa, & Moss, 2001; Smyth, Barry, & Keenan, 2005; Thorpe, Bailey, Huo, Monterroso, & Ouellet, 2001; Thorpe et al., 2002) or injection paraphernalia (Cox, Lawless, Cassin, & Geoghegan, 2000; Hagan et al., 2001; Huo, Bailey, Garfein, & Ouellet, 2005; Maher et al., 2001).

In this paper, we ask whether HCV rates vary across racially or ethnically distinct IDU groups globally. As discussed below, we raise this question because (1) pronounced racial/ethnic differences exist in the prevalence of another injection-related infection – human immunodeficiency virus (HIV) – among IDUs (Friedman et al., 1999), which may also be reflected in HCV infection rates; and (2) systematically documenting the existence of racial/ethnic differences in HCV prevalence may strengthen scientific knowledge related to HCV etiology, and efforts to prevent its transmission. Defining “race” and “ethnicity” is challenging, particularly in a global context. We preface this discussion by defining these concepts.

Race and ethnicity are complex social constructs, therefore it is difficult to find a common definition that cuts across multiple societies. We consulted the guidelines of the “World Conference against Racism, Racial Discrimination, Xenophobia and Related Intolerance” (WCAR) (Durban, South Africa, 2001, <http://www.un.org/WCAR/>) to define these concepts in a global context. The guidelines identified racial minorities, migrants and indigenous peoples as individuals who experience social exclusion, persistent intolerance and discrimination due to their descent, color, and national or ethnic background.

Strides in the research of the HIV epidemic have revealed frameworks that could also be applied to understanding the etiology of HCV in IDUs. A number of studies have shown that HIV is distributed unevenly across racial/ethnic IDU groups in the US, with Blacks and Hispanics having disproportionately higher rates than Whites (Friedman et al., 1999; Haverkos, Turner, Moolchan, & Cadet, 1999; Kottiri, Friedman, Neaigus, Curtis, & Des Jarlais, 2002). Although trends of HIV infection in New York City showed declines in all groups between 1991 and 1996, Hispanics and Blacks were still significantly more likely to be infected compared to Whites over the course of six years (Friedman et al., 1999). Evidence of racial/ethnic disparities in HIV prevalence has also been recorded internationally. In the UK, between 1994 and 2000, there have been increases in new HIV diagnoses in the Black population, while the reverse was seen for Whites (Barry et al., 2002).

Given that HIV and HCV share common parenteral transmission routes (Hagan, Thiede, & Des Jarlais, 2005; Haydon et al., 1998; Huckans, Blackwell, Harms, Indest, & Hauser, 2005; Nyamathi et al., 2005; Sulkowski & Thomas, 2005; Zocchetto et al., 2006), it is plausible that disparities in HCV distributions may also occur. Relatively few empirical studies, however, have examined the association between HCV infection and racial/ethnic membership among IDUs. For example, in Australia, (Maher, Li, Jalaludin, Chant, & Kaldor, 2007) although

ethnic minority new injectors reported similar risk behaviors to their Anglo-Australian counterparts, they had considerably higher HCV seroconversion rates. Further, in the US, minority IDUs disproportionately account for cases of HIV/AIDS, HCV, HBV and tuberculosis (Estrada, 2002).

Documenting possible disparities in HCV across IDU groups has the potential to lay the foundation for further research intended to strengthen our scientific understanding of HCV etiology in IDUs. A large body of research has addressed health disparities, yielding diverse definitions, causes, prevention and intervention strategies (Carter-Pokras & Baquet, 2002). The term health disparity implies an injustice due to “unequal access to resources such as education, health care, clean air and water or ... [living] in unhealthy conditions” (p. 429), resulting in uneven disease distribution. Underpinning such disparities may be societal vulnerability (Mann & Tarantola, 1996), a set of contextual factors that demarcate and limit personal and programmatic vulnerability. Vulnerability is a predisposition to risk due to individual, community or social conditions of disadvantage (including having a racial/ethnic minority status) (Ezard, 2001). For drug users, vulnerability encompasses both individual risks and reduced access to health care, services and prevention.

Increasingly, infectious disease transmission is being interpreted by use of social and structural level factors, which, although produced and reproduced at levels above the individual, have a profound impact on individual risk and/or protective behaviors (Miller & Neaigus, 2001; Rhodes, Singer, Bourgois, Friedman, & Strathdee, 2005). Individualistic frameworks used to explain the epidemiology and build the prevention of blood-borne viruses like HCV and HIV may not reduce risk behavior to a sufficient degree to achieve significant drops in infection rates (Rhodes et al., 2005). IDUs' risk environments need to be considered, as they assume multiple level influences from interpersonal relationships to national or global policies. Rhodes et al. (2005) note that besides their independent impact on HIV transmission, structural factors like unequal racial/ethnic, social, economic, cultural and/or gender positions often converge to act concomitantly towards the reproduction of the HIV epidemic, placing some groups at a disadvantage. Poverty, which is often concentrated among specific racial/ethnic groups, has been documented to be a strong structural factor mediating HIV transmission and to be associated with higher density of IDUs and incident HIV infections (Rhodes et al., 2005). Impoverished communities tend to have reduced social cohesion and protective resources, but increased drug use and sexual risk.

Residential segregation, a purposeful strategy of spatial separation of groups to create dissimilar privilege, may also be linked to hypotheses of higher HCV rates in minority IDUs. Segregation has deep negative implications for minorities, including the creation of health disparities (Williams & Collins, 2001). Segregation is primarily a US phenomenon and a product of racism, which may create vulnerability to HCV. However, racism will manifest differently in different countries, and thus may produce country-specific vulnerabilities to HCV. Segregation in the US is an institutional act of racism that induces poverty, and reduces access to standard education, resources and employment opportunities for minority groups. Further, members of segregated communities (e.g., African-Americans and Latinos in the US) form distinct in-group networks, which often explain high rates of infectious disease.

Such risk and social networks are mechanisms for HIV and HCV transmission in IDUs (Friedman et al., 1997; Kottiri et al., 2002; Miller & Neaigus, 2001). Specifically, one's social affiliations, and injection and sexual risk practices expose them to blood-borne viruses due to their contact with high risk in-group members. The latter in turn have connections with other members, associations that assure the effective transmission of HIV and HCV within these networks. Socializing, including injection practices and drug transactions, tends

to take place through in-group connections and infrequently involves members of other groups (Friedman et al., 1999; Kottiri et al., 2002). Further, Miller and Neaigus (2001) state that large social networks with strong peer risk norms are good preservers of risk behaviors and are resistant to change, thus posing a challenge to interventions. This research has shed light on how transmission of blood-borne viruses is facilitated by one's direct and indirect connections with other members within their own risk networks. Therefore, increased prevalence and incidence of infection can be perpetuated within specific racial/ethnic groups of lesser privilege in the US and other countries.

As has happened with HIV, documenting variability in HCV rates across racial/ethnic groups has important implications for directing prevention efforts where they may be scant or missing all together (Friedman et al., 1999). Inequitable distribution of health services, prevention and treatment leads to vulnerability that varies geographically and is a structural determinant of epidemics among poor and subordinated IDUs. Higher rates of HCV conversion in ethnic minorities in Australia were attributed to increased social isolation and limited knowledge of blood-borne viruses (Maher et al., 2007). Similarly, the increased cases of HIV/AIDS, HCV, HBV and tuberculosis in US minorities were associated with poorer access to health care, later disease detection, and costly treatments among racially/ethnically marginalized populations (Estrada, 2002). Thus, documenting racial/ethnic disparities in HCV infection in IDUs carries public health importance due to the implications it has for tailoring prevention and care programming to the needs of diverse groups, and ensuring that services are accessible to those in most need.

In this paper, we examine the available empirical evidence from the HCV Synthesis Project (Hagan et al., 2007; Scheinmann et al., 2007) to determine whether certain racial/ethnic groups bear a disproportionate burden of disease (Rhodes et al., 2005). Further, underprivilege based on one's racial/ethnic identity is not a monolithic experience, and this paper seeks to systematically capture variations in HCV prevalence and incidence both across and within minority IDU groups inhabiting various national and global territories.

Methods

The HCV Synthesis Project overview

The data in this paper were collected during the NIDA-funded "Synthesis: A meta-analysis of HCV epidemiology and prevention in drug users" (the HCV Synthesis Project), which is a systematic review of international research examining the epidemiology and prevention of hepatitis C in drug-user populations. Published and unpublished studies from the US and other countries on the prevalence and incidence of HCV infection, the molecular epidemiology of HCV genotypes, and HCV co-infection with HIV, HBV and HAV in drug users were retrieved and coded. We retrieved reports released between January 1, 1989, when testing for HCV became available (Kuo et al., 1989), and December 31, 2006. Previous publications (Hagan et al., 2007; Scheinmann et al., 2007) describe the HCV Synthesis methods in further detail.

As noted in the introduction, we relied on the WCAR definition of race and ethnicity. Based upon this definition, the following groups of IDUs from our sample were examined: (1) racial/ethnic minorities: African-Americans or Hispanics in the US, the Roma in Bulgaria, Asians in Australia, non-Western Europeans in the Netherlands and non-Han groups in China; and (2) indigenous individuals: Aboriginals in Canada and Australia. In this paper we refer to those vulnerable and marginalized as "minority" groups and to their more advantaged counterparts as "majority" groups (even if the majority may be smaller in numbers than the minority).

We excluded from our statistical analyses subgroups or studies with vaguely described racial/ethnic categories (e.g., undifferentiated non-Aboriginal). In some cases, although the group definition was unclear, based on the PI's and co-investigators' knowledge of the study, we were able to estimate the composition of the group and included those cases in the analysis, such as 'White/other' (Hagan et al., 2001) or 'non-White' (Murrill et al., 2002). We also excluded subgroups that could not be classified with similar categories across the studies (e.g., American Indian/Pacific Islander). Some studies had overlapping data (e.g., originated from the same study sample), in which case only one study was chosen for analysis, based on the level of detail provided. Lastly, some studies reported data for distinct groups (e.g., White, Hispanic, African-American), while others grouped one or more racial/ethnic categories together (e.g., non-Western European including Turkish/Moroccan, Surinamese, Antillean and other IDU). For each metaanalysis, we selected groups that best fit our definitions of "minority" or "majority" status groups.

Three reports (Hagan, Vassilev, & Des Jarlais, 2006; van de Laar et al., 2005; Miller et al., 2006) contained more than one study because they provided data for different time periods or demographic characteristics. For example, van de Laar et al. (2005) provided data for the time periods of 1985–1989 and 2000–2004 for different samples (these appear in Table 1 as prevalence 1 and prevalence 2). Further, some reports had both a prevalence and incidence study and appear accordingly in our results and tables. Prevalence and incidence rates were abstracted from each study as given, or were calculated when a denominator and/or numerator of positive cases or percentage of positive cases were available. Self-reported HCV positive status was not considered reliable, therefore such studies were excluded all together from the HCV Synthesis sample. Only participants subjected to HCV antibody testing were included.

Quality measures

Guided by the MOOSE group's recommendations (Stroup et al., 2000), a quality rating scale was developed and applied to each study. Based on previous meta-analyses (Navas-Acien et al., 2005; Thakkinstian et al., 2005), we developed two separate scales for prevalence and incidence studies to assess their methodology and data reporting. Examples from the scales include: (1) Were the selection criteria for the sample well defined and explained? (2) Were details of recruitment methods given? A full description of our non-injection drug users (NIDUs) quality scale items can be found in Scheinmann et al. (2007), which is identical to the IDU scale used in the current paper except for a few elements characteristic to NIDUs. The quality coding and scoring was done for each report by research associates (CLW and RS), and inconsistencies were resolved at staff meetings.

We performed Cronbach's alpha reliability analyses to verify the scales' internal consistency by using all the studies from the HCV Synthesis Project. The alpha values were 0.73 for prevalence and 0.71 for incidence studies, both of which reflecting adequate scale coherence. The distributions of quality scores for both prevalence and incidence were bimodal, and were recoded dichotomously by splitting between the modes. For prevalence studies (19 items), the modes were 12 and 16, the split was <14 versus ≥14, and the median was 16. Of the 29 prevalence studies in this analysis, 22 scored ≥14 (coded as 1) and seven scored <14 (coded as 0). For incidence studies (9 items), the median was 5, the mode was 6, and the split was <5 versus ≥5. Of the 11 incidence studies in this analysis, 10 scored ≥5 (coded as 1) and one scored <5 (coded as 0). The higher the quality score, the more rigorous the methodology, and reporting of research design, setting, sample, analyses and findings.

Statistical analysis

Study effects were categorized preliminarily into subgroups based on similar racial/ethnic comparisons (e.g., Black–White comparisons in the US, or Aboriginal–White comparisons in Canada and Australia). The effect estimate of the racial/ethnic comparison was the odds or risk ratio (OR or RR) (for prevalence and incidence studies, respectively) comparing the rates of HCV in the racial/ethnic ‘minority’ (vulnerable) group to the ‘majority’ (more privileged) group.

Effects were considered homogeneous if they were similar across studies, and heterogeneous if they were not. Identification of homogeneous subgroups for statistical analysis was an iterative process of graphical displays and statistical testing using the Comprehensive Meta-Analysis software (Borenstein, Hedges, Higgins, & Rothstein, 2005). Homogeneity of effects was tested statistically using Cochran’s Q and the I^2 statistic (DerSimonian & Laird, 1986; Higgins & Thompson, 2002; Higgins, Thompson, Deeks, & Altman, 2003). For Q , homogeneity is confirmed by comparison with the chi-square distribution using standard significance criteria and is indicated by a non-significant result. I^2 is a continuous measure of inconsistency of effects, and ranges from 0% to 100%; values below 50% indicate homogeneity (Higgins & Thompson, 2002).

Two-tailed z tests for the pooled ORs or RRs were also calculated. To maximize the generalizability of the results, we assumed random effects. We also explored statistical power and study quality scores as effect moderators in order to account for heterogeneity in the comparisons (Cooper & Hedges, 1994).

One incidence study (Patrick et al., 2001) only provided follow-up time per person averaged across all the racial-ethnic groups. Therefore, the average follow-up time was assumed to be equal across racial-ethnic groups for incidence calculations.

Power calculations

Each subgroup comparison was evaluated to determine whether adequate statistical power existed to detect a meaningful difference. Given that most of the studies did not have race/ethnicity associations as a stated research objective, we set values of 70% or above to indicate sufficient power. To represent an important difference between racial/ethnic groups, power was calculated using a two-tailed 0.05 significance level, and an odds ratio of 2.0 for prevalence, and a 10% change for incidence. Calculations were performed using NCSS/PASS software (Hintze, 2006), based on procedures for logistic regression (Hsieh, Block, & Larsen, 1998), and case–control cohort data (Machin, Campbell, Fayers, & Pinol, 1997).

Results

Sample characteristics

The HCV Synthesis Project identified 29 prevalence and 11 incidence studies reporting HCV data by racial/ethnic categories (Table 1). The 40 studies represent a modest 6% of our entire HCV Synthesis sample. Furthermore, in only four reports (Bradshaw, Pierce, Tabrizi, Fairley, & Garland, 2005; Hagan et al., 2006; Miller et al., 2006; Thorpe, Ouellet, Levy, Williams, & Monterroso, 2000) was the examination of race/ethnicity in relation to HCV a stated objective. In the majority of studies, the participants were recruited from street locations, drug treatment programs and medical settings, via street outreach or self-referral.

Studies demographics

Sample sizes in the studies varied between 61 and 1345 for prevalence studies and from 29 to 484 for incidence studies. The majority of participants were males. Nine prevalence

studies included young injectors only (30 years old and under) (Table 1). The majority of studies reporting age characteristics had samples with a mean/median age of 23–29 years old. Mean/median duration of injection was 3 years in three studies, 4–8 years in five studies and 11–17 years in three studies. Of the incidence studies, two reported median age of their sample and four studies included only young injectors (under the age of 30). One study reported the median duration of injection to be 7 years.

Prevalence studies

A stream of studies from the US shows higher HCV rates in non-Whites (Table 2). In Denver, non-White IDUs had a 79% HCV prevalence compared to 48% in White IDUs (Kwiatkowski, Corsi, & Booth, 2002). Non-Whites in San Francisco had higher HCV prevalence than Whites (87% versus 60%) (Murrill et al., 2002). One study reported 80% HCV prevalence for African-Americans and Hispanics, and 70% for Whites in Los Angeles (Nyamathi et al., 2002). In Oregon, White IDUs had lower HCV prevalence (51%) compared to African-Americans (76%) (Thomas, 2006). In Chicago, Thorpe et al. (2000) found the HCV prevalence distribution in young Hispanic, Black and White IDUs to be: 41%, 29% and 21%, respectively. Lower HCV rates in White IDUs (69%) than in Hispanics and African-Americans were found in Sacramento California (83% and 77%, respectively) (Zeldis et al., 1992). Exceptions to the above rates are two US studies showing no differences in HCV rates between Whites and non-Whites (Des Jarlais et al., 2005; McCarthy & Flynn, 2001). Further, four US studies presented higher HCV rates in White IDUs. In New Mexico, Hispanic/White, White, American Indian/Pacific Islander, and African-American IDUs had HCV prevalence rates of 86%, 77%, 68% and 53%, respectively (Samuel, Doherty, Bulterys, & Jenison, 2001). In San Francisco, Black IDUs had the lowest HCV rates compared to Whites, Hispanics and others: 33%, 45%, 42% and 48%, respectively (Hahn et al., 2001). Finally, African-American IDUs in Baltimore had considerably lower HCV prevalence compared to Whites and Whites/Others (Garfein et al., 1998; Ompad, Fuller, Vlahov, Thomas, & Strathdee, 2002).

In both Australia and Canada, Aboriginals consistently had the highest HCV prevalence rates (Bradshaw et al., 2005; Miller et al., 2006; Patrick et al., 2001; Wylie, Shah, & Jolly, 2006). Asians in Australia had increased HCV rates compared to non-Asians (Maher, Chant, Jalaludin, & Sargent, 2004). Roma IDUs in Bulgaria had higher HCV prevalence than White Bulgarians (Hagan et al., 2006), as did non-Han IDUs in China compared to the Han majority IDUs (Ruan et al., 2004).

Imprisoned Aboriginal IDUs had slightly lower rates compared to non-Aboriginals in Australia, although the referent group may have been a heterogeneous sample of low and higher risk individuals, including immigrants (Butler et al., 1999). In the Netherlands, van de Laar et al. (2005) found higher HCV prevalence in 1985–1989 and in 2000–2004 for Western European IDUs compared to that of non-Western Europeans (consisting of Turkish, Moroccan, Surinamese, Antilleans and other IDUs). In Brazil, Zoccratto et al. (2006) found that non-White IDUs had lower HCV rates compared to White IDUs.

Prevalence studies meta-analyses

Aboriginals versus Whites in Canada and Australia—Three studies compared Aboriginal groups to Whites (Table 3). The summary OR for HCV prevalence among Aboriginals as compared to Whites was 2.04 (95% CI = 1.48, 2.82). The I^2 of 12% and $Q = 2.3$, NS, indicate homogeneity in the comparison.

Ethnic minorities versus majority groups—This group had eight potential comparisons drawn from six studies that contrasted HCV in ethnic minorities to various

local majority groups (Bradshaw et al., 2005; Hagan et al., 2006; van de Laar et al., 2005; Maher et al., 2004; Ruan et al., 2004; Zocchetto et al., 2006). When all the groups were included in the analysis, the heterogeneity in the data was high ($I^2 = 82\%$) and the difference was not significant (data not shown). When we limited the meta-analysis to comparisons for which the composition of the contrasted groups was not ambiguous (see Methods section for details) (Hagan et al., 2006; van de Laar et al., 2005; Maher et al., 2004; Ruan et al., 2004), the effect size indicated higher HCV prevalence in minorities (OR = 1.97, 95% CI = 1.20, 3.24), $I^2 = 58\%$ and $Q = 9.5$, $p = 0.05$.

Blacks versus Whites in the US—Seven studies reported HCV prevalence in Black or African-American in contrast to White IDUs. The summary OR was 1.34 (95% CI = 0.89, 2.02), indicating that Blacks had marginally higher HCV prevalence, but the difference was not significant. Heterogeneity ($I^2 = 45\%$, $Q = 10.9$, $p = 0.09$) was reduced ($I^2 = 0\%$) when we selected only those studies meeting our quality criteria, but neither the pooled effect size nor statistical significance changed substantially (data not shown).

Hispanics versus Whites in the US—Six studies reported HCV prevalence data that allowed a Hispanic–White comparison, however, the associations were heterogeneous ($I^2 = 68\%$ and $Q = 15.5$, $p = 0.15$) and the summary OR was 1.44 (95% CI = 0.87, 2.38). When studies with quality scores below 14 were removed, heterogeneity was reduced ($I^2 = 34\%$) and there was excess HCV prevalence among Hispanics (OR = 2.14, 95% CI = 1.39, 3.29) (data not shown).

Non-Whites versus Whites in the US—Kwiatkowski et al. (2002) and Murrill et al. (2002) compared non-Whites versus Whites, the former including primarily Blacks and Hispanics. There was a strong significant effect size indicating higher HCV prevalence among non-White groups (OR = 4.32, 95% CI = 2.72, 6.84), and the data were homogeneous ($I^2 = 0\%$ and $Q = 0.1$, NS). Overall statistical power was adequate (70%) for each pooled prevalence comparison.

Incidence studies

Of the 11 studies that reported HCV incidence in relation to race/ethnicity, only seven were included in our analyses (three had overlapping data and one did not report data relevant to our analyses). Four studies reported elevated HCV incidence rates in vulnerable groups (Table 4). In Australia, the incidence in Aborigines was 24 per 100 Person Years (PY) versus 16.8 per 100 PY for non-Aborigines, although the sample size was small (Butler, Kariminia, Levy, & Kaldor, 2004). In another Australian study, HCV incidence in ethnic minorities was 55.9 per 100 PY compared to 22 per 100 PY for non-ethnic minorities (Maher et al., 2006). In Canada, Aborigines had an incidence of 58.4 per 100 PY compared to 42.9 per 100 PY for Whites (Patrick et al., 2001), and in Chicago, HCV incidence was 1.51 times higher in Hispanics versus Whites (95% CI = 0.63, 3.61) (Thorpe et al., 2002).

Two US studies showed lower seroconversion rates in non-White IDUs. Specifically, HCV incidence in African-Americans and Whites/Others in Seattle were 5.4 per 100 PY and 19.2 per 100 PY, respectively (Hagan et al., 2001). In San Francisco, the HR for non-Whites versus Whites was 0.7 (95% CI = 0.4, 1.5) (Hahn et al., 2002). Two studies found no differences (Dolan et al., 2005; Thorpe et al., 2002).

Incidence studies meta-analyses

Two meta-analyses seemed conceptually feasible for incidence data. The first one consisted of Aboriginal IDUs versus others in Australia and Canada (Butler et al., 2004; Dolan et al., 2005; Patrick et al., 2001). Aboriginal groups had higher HCV seroconversion rates,

although the difference was not statistically significant ($RR = 1.31$, 95% $CI = 0.87, 1.99$); there was no heterogeneity in the data ($I^2 = 0\%$ and $Q = 1.1$, NS). In the second meta-analysis, we examined seroconversion rates in African-Americans versus other IDUs in the US (Hagan et al., 2001; Thorpe et al., 2002). There was no evidence of heterogeneity ($I^2 = 18\%$ and $Q = 1.2$, NS), and African-Americans had lower seroincidence rates than other IDUs in the US, although the pooled effect lacked statistical significance ($RR = 0.45$, 95% $CI = 0.20, 1.08$). Overall statistical power for the pooled incidence comparisons was adequate (70%) for Aboriginals versus other IDUs in Australia and Canada, but not adequate for African-Americans versus other IDUs in the US.

Discussion and conclusions

By use of systematic reviews and meta-analytic methods, this paper examined whether HCV rates in IDUs varied across racial/ethnic groups, specifically asking if marginalized or vulnerable groups may present higher rates of HCV infection. It is extremely challenging to unpack the concept of race/ethnicity, as it encapsulates a conglomerate of social, economic, political and cultural forces. Given the international sample of studies included in our analyses, the particulars of what determines one's position of disadvantage varied based on geography and unique histories of each minority group for which data were available. Our findings need to be interpreted with the awareness that although several groups may be classified as belonging to a minority group, their underlying characteristics and conditions may not yield homogeneous distributions of HCV infection.

From the HCV Synthesis Project database, we identified 29 prevalence and 11 incidence studies which presented HCV infection rates in diverse racial/ethnic IDUs groups; this represents only 6% of the studies on drug users and HCV in the database. Further, in only four reports was race/ethnicity a stated objective of the paper, indicating that little attention has been paid to studying HCV variation in relation to IDUs' racial/ethnic membership. Given that only four out of the 40 studies intended to study race/ethnicity, publication bias may be ruled out. Further, when the four studies (Bradshaw et al., 2005; Hagan et al., 2006; Miller et al., 2006; Thorpe et al., 2000) were removed from analyses, the results did not change.

Several of our comparisons revealed marginal or significant heterogeneity, with some subsets of those yielding homogeneous and significant differences indicating excess HCV in minority IDUs. Given that we combined in the same meta-analyses groups from different countries and settings, with unique histories and racial/ethnic attributes, it could be argued that looking at these data in a meta-analytic manner would inevitably uncover heterogeneity. While a systematic review of these studies may have constituted an alternative to our meta-analyses, it seemed important to take on the challenging task of identifying groups who, due to a shared fundamental ideology of vulnerability and marginalization, are more susceptible to contracting HCV than their more privileged counterparts. As expected, heterogeneity was present, however, we were still able to identify homogeneous subsets, as well as prevalence and incidence suggestive of higher rates of HCV in minority groups. Further, it was often the case that analyses in which we only included studies that met our study quality criteria had reduced heterogeneity and revealed excess HCV rates in minority groups.

For prevalence studies, the clearest disparity in HCV status was observed in the Aboriginal versus White comparison, followed by the US non-White versus White comparison. Aboriginal groups in Canada and Australia also showed higher HCV seroconversion rates when compared to others. We excluded groups from the ethnic minorities meta-analysis whose composition we could not, at the very least, estimate. Studies with the most clearly defined racial/ethnic categories yielded the most homogeneous comparisons and higher

HCV rates in minority groups. When studies with some degree of ambiguity were included, excess HCV prevalence among minorities was no longer observed and heterogeneity increased.

Overall, Hispanics in the US had elevated rates of HCV prevalence (Table 2), yet the difference in comparison to the White referent group was not significant (Table 3). However, the removal of studies with quality scores below 14 yielded a homogeneous analysis and showed higher HCV rates for Hispanics (Hahn et al., 2001; Thorpe et al., 2002; Zeldis et al., 1992). The fact that Hispanics in the US are a diverse group, depending on their country of origin, may explain the variations we observed. These results suggest that unless data become available to allow analyses for individual Hispanic subgroups, an overall Hispanic comparison may not generate reliable conclusions.

The US Black/African-American versus White/Others meta-analyses showed somewhat increased prevalence but decreased incidence in African-Americans, suggesting that longitudinal research is needed to elucidate the context and changing trends of HCV transmission in this US group. Cross-sectional prevalence studies do not control for differences in time at risk across racial/ethnic groups, which could generate confounding results. This may have occurred to some degree in our data. Kottiri et al. (2002) assessed HIV transmission in Black, Puerto Rican and White IDUs networks in New York City and found that HIV risk behavior was similar in Black and White IDUs. However, risk networks of Black, Puerto Rican and White IDUs were segregated, therefore implying different group risk behaviors and protective mechanisms, even when individual risk practices may be the same. Further, the background HIV prevalence was much higher for Black and Latino IDUs compared to Whites, therefore the same injection behaviors exposed the first two groups to more risk compared to White IDUs. The research targeting social networks has also explained similar differences in these groups' sexually transmitted infections rates (Laumann & Youm, 1999).

There were limitations to our meta-analyses. Given our conceptual interest in race/ethnicity, we examined smaller subgroups of each study's entire sample. Not all subgroup comparisons were sufficiently powered to enable the detection of possible racial/ethnic differences. The fact that HCV distribution in relation to race/ethnicity is studied on small scales and infrequently is in itself a finding, and we recommend that future studies undertake race/ethnicity as a principal focus. Further, 66% of the prevalence and incidence studies we examined comprised mostly of IDUs who belonged to majority groups. Significant differences between racial/ethnic groups may have been attenuated by disproportionate sample demographics. Our analyses were also limited by the lack of clear definitions of some racial/ethnic groups. We suspect that the inclusion of groups that may present different characteristics and risk behaviors could have either inflated or underestimated HCV rates. Lastly, given that the studies did not have elaborate conceptual models of race/ethnicity, our own conceptualization of this otherwise complex variable was to some extent restricted.

Aside from documenting unequal racial/ethnic distributions in HCV, it is also important that future research attempts to explain this variability to understand the multiple underlying determinants of HCV transmission, including social and structural differences among groups of injectors (Miller & Neaigus, 2001; Rhodes et al., 2005). For example, unequal health-related services are structural factors that need to be accounted for in epidemiology and are most likely strong predictors of higher disease rates (Diez-Roux, 1998) and unmet health care needs for minority groups (Wells, Klap, Koike, & Sherbourne, 2001). In the US, although a study looking at 94 metropolitan statistical areas (MSAs) found that the majority of the injecting population comprised of Whites, it was estimated that Blacks had an increased likelihood of injecting, while the Latino/White injection rates were similar, with

certain disparities throughout the MSAs (Cooper, Friedman, Tempalski, Friedman, & Keem, 2005). The authors suggest further investigation of correlations between increased risk for injection (and therefore blood-borne infectious diseases transmission) and residential segregation (which is in turn associated with increased poverty, unemployment and decreased education quality and access to health services). Future research should however be cautious when making inferences about observed risk behaviors and varying HCV rates studied cross-sectionally, given that HCV differences could have occurred earlier and may not have a direct link to current risk behaviors. Knowledge generated by these research agendas could nonetheless inform policy and design of prevention and intervention for minority IDUs in different countries. Thus, prevention becomes a political task, as it necessitates profound structural and large scale changes intended to address inequalities, human rights and welfare (Rhodes et al., 2005).

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Table 1

Study characteristics

Study (study name)	Location	Dates	Recruitment method	Recruitment setting	Sample characteristics
Prevalence studies					
Bradshaw et al. (2005)	Melbourne, Australia	1999–2002	Street outreach	Needle exchange; street locations	Mean age =26 years; 4 years= median duration IDU =4 years; 63% male; 71% White, 11% Aboriginal, 7% Southeast Asian, 11% Other
Butler et al. (1999)	New South Wales, Australia	March–September 1996	Systematic	Correctional	75.6% male; 29% Aboriginal, 71% non-Aboriginal
Des Jarlais et al. (2005)	New York, NY, USA	2000–2001	Systematic	Drug treatment center	27% White, 54% Hispanic, 18% Black, 1% Other
Garfein and Vlahov (1996) ^a (AIDS Link to Intravenous Experience)	Baltimore, MD, USA	1988–1992	Self-referral; peer driven	Drug treatment center; medical setting; STD clinic; HIV clinic; other community based organizations; correctional; street outreach; public housing	Mean age= 28.6 years; 72.5% male; 88% Black, 12% Other
Garfein et al. (1998) (Risk Evaluation Assessment of Community Health)	Baltimore, MD, USA	1994–1996	Self-referral; street outreach	Drug treatment center; public health clinic; medical setting; STD clinic; HIV clinic; other community based organizations; homeless shelters; street outreach	Median age= 23.5 years; median duration IDU= 2.5 years; 46% male; 79% African-American, 21%, non-African-American
Gunn et al. (2003)	San Diego, CA, USA	1999–2000	100% sample	STD clinic	71% are at least 30 years old; 8% African-American, 91% Other
Gynarmathy, Neaigus, Miller, Friedman and Des Jarlais (2002)	New York, NY, USA	1996–2001	Peer driven; street outreach	Street locations	71% male; 32% White, 45% Hispanic, 19% Black, 6% Other
Hagan et al. (2006) prevalence 1	Sofia, Bulgaria	2003–2004	Street outreach; unspecified	Drug treatment center; needle exchange	Mean age= 25.9 years; mean duration IDU =7.1 years; 80% male; 100% White
Hagan et al. (2006) prevalence 2	Sofia, Bulgaria	2003–2004	Street outreach; Unspecified	Drug treatment center; needle exchange	Mean age= 29.4 years; mean duration IDU =7.6 years; 76% male; 100% Roma
Hahn et al. (2001) UFO Study	San Francisco, CA, USA	1997–1999	Street outreach	Street locations; community based organizations	Median age =22 years; median duration IDU= 5 years; 69% male; 77% White, 23% non-White
Kwiatkowski et al. (2002)	Denver, CO, USA	1998–1999	Street outreach	Street locations	Mean age= 34.09 years; mean duration IDU= 13.43 years; 63%

Study (study name)	Location	Dates	Recruitment method	Recruitment setting	Sample characteristics
					male; 58% White, 42% non-White
Maier et al. (2004)	Sydney; Australia	1999–2002	Peer driven; street outreach	Drug treatment center; medical setting; needle exchange; street locations; youth health service	Mean age= 23.1 years; mean duration IDU= 4.3 years; 66% male; 66% White, 27% Asian, 7% Aboriginal
McCarthy and Flynn (2001)	Sacramento, CA, USA	Unspecified	100% sample	Drug treatment center	55% White, 17% Hispanic, 12% African-American, 16% Other
Miller et al. (2002) ^a VIDUS (Vancouver Injection Drug User Study)	Vancouver, Canada	1996–unspecified	Self-referral; street outreach	Street locations	Median age =21 years; age range =13–24; 50% male; 24% Aboriginals; 76% non-Aboriginals
Miller et al. (2006) prevalence 1 VIDUS	Vancouver, Canada	1996–2003	Self-referral; street outreach	Street locations	Median age =16 years; median duration IDU =5 years; 35% male; 100% Aboriginal
Miller et al. (2006) prevalence 2 VIDUS	Vancouver, Canada	1996–2003	Self-referral; street outreach	Street locations	Median age =17 years; median duration IDU= 3 years; 55% male; 100% non-Aboriginal
Murrill et al. (2002) (CDC National HIV Seroprevalence Studies)	San Francisco, CA, USA	1993–1994	100% sample	Drug treatment center	67% male; 67% White, 33% non-White
Nyamathi et al. (2002)	Los Angeles, CA, USA	1995–1999	Peer driven; street outreach; referral from agencies	Street locations; homeless shelters; other	67% male; 67% White, 33% non-White
Ompad et al. (2002) REACH II	Baltimore, MD, USA	1997–1999	Self-referral; peer driven; street outreach	Street locations	Mean age= 25.96 years; median duration IDU= 2.1 years; 33% male; 23% White; 77% African-American
Patrick et al. (2001) VIDUS	Vancouver, Canada	1996–1999	Street outreach	Street locations	Median age =34 years; median duration IDU= 11 years; 66% male; 60% White, 25% Aboriginal, and 15% Other
Ruan et al. (2004)	Sichuan Province, China	November 2002	Peer driven; street outreach	Street locations	83% male; 64% Han, 36% Other
Samuel et al. (2001)	Albuquerque, Clovis, Roswell, Hobbs, Carlsbad, and Las Cruces; New Mexico; USA	1995–1997	Self-referral; street outreach	Street locations	Median age= 37 years; median duration IDU =17 years; 73% male; 18% White, 73% Hispanic, 5% African-American; 4% American Indian
Thomas (2006)	Multnomah County, OR, USA	January–August 2000	Unspecified	Drug treatment center; STD clinic; HIV clinic; correctional	66% male; 76% White, 9% African-American, 6% Pacific Islander, 6% Aboriginal
Thorpe et al. (2000) CIDUS II (Collaborative Injection Drug Users Study II)	Chicago, IL, USA	1997–1999	Self-referral; peer driven; street outreach	Street locations	Median duration IDU= 2 years; 65% males; 58% White, 22% Hispanic, 17% Black, 3% Other

Study (study name)	Location	Dates	Recruitment method	Recruitment setting	Sample characteristics
van de Laar et al. (2005) prevalence 1 (Amsterdam Cohort Studies)	Amsterdam, The Netherlands	1985–1989	Peer driven; street outreach	Drug treatment center; street locations; STD clinic	44% male; 8% non-Western Europeans, 92% Western Europeans; 60% injected at least 4 years; 85% borrowed used needles
van de Laar et al. (2005) prevalence 2 (Amsterdam Cohort Studies)	Amsterdam, The Netherlands	2000–2004	Peer driven; street outreach	Drug treatment center; street locations	59% male; 18% non-Western Europeans, 82% Western Europeans; 50% injected at least 4 years; 44% borrowed used needles
Wood et al. (2005) (Scientific Evaluation of Supervised Injecting)	Vancouver, Canada	2003–2004	Systematic	Safer injection facility	71% male; 18% Aboriginal, 82% Other
Wylie et al. (2006)	Winnipeg, Manitoba, Canada	2003–2004	Self-referral; peer driven	Medical setting; street locations	57% male; 35% White, 65% Aboriginal
Zeldis et al. (1992)	Sacramento, CA, USA	1987–1989	Self-referral	Drug treatment center	Mean age =33 years; 59% male; 74% White, 17% Hispanic, 5% African-American, 2% Asian, 1% American Indian/Pacific Islander, 1% Other
Zoccatto et al. (2006) AJUDE	Sao Paulo; Sorocaba; Sao Jose do Rio Preto; Itajai; Porto Alegre; Brazil	June 1998	Targeted; street outreach	Needle exchange	Mean age =29.25 years; 83% male; 50% White, 50% Other
Incidence studies					
Butler et al. (2004)	New South Wales, Australia	1996	Systematic	Correctional	90% male; 35% Aboriginal, 65% Other
Des Jarlais et al. (2003) ^a CIDUS II	New York, NY, USA	1997–1999	Self-referral; peer driven; street outreach	Street locations	N/A
Dolan et al. (2005)	Sydney, New South Wales, Australia	1997–1998	100% sample	Correctional	100% male
Hagan et al. (1999) ^a RAVEN (Risk Activity Variables, Epidemiology, and Network Study)	Seattle, WA, USA	1994–1996	Systematic	Drug treatment center; other community based organizations; correctional; drug use assessment agencies	61% male; 77% White, 12% Black, 11% Other
Hagan et al. (2001) RAVEN	Seattle, WA, USA	1994–1997	Systematic	Drug treatment center; needle exchange; correctional; and street locations	60% male; 18% African-American, 82% White/Other
Hagan, Thiede and Des Jarlais (2004) ^a RAVEN	Seattle, WA, USA	1994–2001	Systematic	Drug treatment center; other community based organizations; correctional; drug use assessment agencies	62% male; 76% White, 14% African-American, 10% Other
Hahn et al. (2002)	San Francisco, CA, USA	2000–2001	Self-referral; peer driven; street outreach	Street locations	22 years= median age; 64% male; 80% White, 20% non-White
Maher et al. (2006)	New South Wales, Australia	1999–2002	Self-referral; street outreach	Drug treatment center; STD clinic; street	28% ethnic minorities, 72% non-minorities

Study (study name)	Location	Dates	Recruitment method	Recruitment setting	Sample characteristics
				locations	
Miller et al. (2002) ^a VIDUS	Vancouver, Canada	1996– unspecified	Self-referral; street outreach	Street locations	18% Canadian, 82% non-Aboriginal
Patrick et al. (2001) VIDUS	Vancouver, Canada	1996–1999	Street outreach	Street locations	Median age= 28 years; age range =15-55 years; median duration IDU= 7 years; 70% male; 54% White, 16% Aboriginal, and 30% Other
Thorpe et al. (2002) CIDUS II	Chicago, IL, USA	1997–1999	Self-referral; peer driven; street outreach	Street locations	64% male; 64% White, 12% Hispanic, 13% Black

^a Not included in statistical analyses due to overlapping or insufficient data.

Table 2

HCV prevalence across diverse racial/ethnic IDU groups

Study	Racial/ethnic category	Total IDU tested	Prevalence (%)
US studies			
Des Jarlais et al. (2005)	Black	76	59.0
	Hispanic	221	63.0
	White	111	65.0
Garfein et al. (1998)	African-American	180	34.0
	Other	49	51.0
Hahn et al. (2001)	Black	6	33.3
	Hispanic	12	41.7
	White	237	45.1
	Other	52	48.0
Kwiatkowski et al. (2002)	Non-White ^a	67	79.1
	White	91	48.4
McCarthy and Flynn (2001)	Hispanic/Mexican	70	94.0
	African-American	49	94.0
	White	227	96.0
	Other	67	96.0
Murrill et al. (2002)	Non-White	117	87.0
	White	238	60.0
Nyamathi et al. (2002)	African-American	83	79.5
	Hispanic	25	79.5
	White	43	70.0
	Other	2	100.0
Ompad et al. (2002)	White and Other	24	62.5
	African-American	82	37.8
Samuel et al. (2001)	American Indian/Pacific Islander	–	67.6
	Hispanic/White	–	86.3
	African-American	–	53.3
	White	–	76.7
Thomas (2006)	Mixed heritage	13	38.5
	Asian/Pacific Islander	2	0.0
	American Indian	27	66.6
	African-American	42	76.2
	White	349	50.7
	Other	16	62.5
	Other	16	62.5
Thorpe et al. (2000)	Black	118	28.8
	Hispanic	155	41.3
	White	404	21.3
	Other	21	28.6

Study	Racial/ethnic category	Total IDU tested	Prevalence (%)
Zeldis et al. (1992)	African-American	30	76.7
	Hispanic	100	83.0
	White	430	69.3
	Other ^b	25	68.0
Aboriginals in Canada and Australia			
Bradshaw et al. (2005)	Mostly Middle Eastern & European	35	71.0
	Southeast Asian	22	55.0
	Aboriginal	34	94.0
	White	223	73.0
Butler et al. (1999)	Aboriginal	96	65.6
	Non-Aboriginal	232	75.4
Miller et al. (2006)	Non-Aboriginal	211	37.9
	Aboriginal	80	66.3
Patrick et al. (2001)	Aboriginal	338	91.0
	Other	199	63.0
	White	808	82.0
Wood et al. (2005)	Aboriginal	123	90.2
	Other	568	87.0
Wylie et al. (2006)	Aboriginal Canadian	237	59.5
	White	128	44.5
Other groups			
Hagan et al. (2006)	White Bulgarians	599	70.0
	Roma	136	90.4
Maher et al. (2004)	Non-Asian ^c	270	31.5
	Asian	102	50.0
Ruan et al. (2004)	Other (not Han)	136	78.7
	Han	243	66.6
van de Laar et al. (2005)	Non-Western European ^d	16	87.5
	Western European	173	90.8
van de Laar et al. (2005)	Non-Western European ^d	11	36.4
	Western European	50	46.0
Zoccatto et al. (2006)	Non-White	137	46.7
	White	134	59.7

^aHispanic, African-Americans, Native Americans, and other ethnicity.

^bAsian Americans, Native Americans, and people whose ethnic origin could not be classified.

^cWhite and Aboriginals (mostly White).

^dTurkish/Moroccan, Surinamese, Antillean, and other.

Table 3

Meta-analyses of HCV prevalence and incidence in diverse racial/ethnic IDU groups

Prevalence Studies						
Study	Odds Ratio	Lower limit	Upper limit	Group Comparison	Quality ^a	Power ^b
Patrick et al., 2001	2.04	1.36	3.06		1	1
Bradshaw et al., 2005	5.89	1.37	25.33		1	0
Wylie et al., 2006	1.83	1.28	2.82		1	1
Random Effects	2.04	1.48	2.82			
$I^2=12\%$; $Q=2.3$, NS						
Hagan et al., 2006	4.06	2.24	7.39		1	1
Maher et al., 2004	2.18	1.37	3.47		1	1
Ruan et al., 2004	1.84	1.13	3.01		1	1
van de Laar et al., 2005.1	0.71	0.15	3.42		1	0
van de Laar et al., 2005.2	0.67	0.17	2.58		1	0
Random Effects	1.97	1.20	3.24			
$I^2=58\%$; $Q=9.5$, $p=0.05$						
Des Jarlais et al., 2005	0.79	0.43	1.43		0	0
Hahn et al., 2001	0.61	0.11	3.38		1	0
McCarthy & Flynn 2001	0.56	0.14	2.19		0	0
Nyamathi et al., 2002	1.68	0.73	3.90		0	0
Thomas, 2006	3.1	1.48	6.52		0	0
Thorpe et al., 2000	1.50	0.94	2.38		1	1
Zeldis et al., 1992	1.46	0.61	3.48		1	0
Random Effects	1.34	0.89	2.02			
$I^2=45\%$; $Q=10.9$, $p=0.09$						
Des Jarlais et al., 2005	0.92	0.57	1.48		0	1
Hahn et al., 2001	0.87	0.27	2.81		1	0
McCarthy & Flynn 2001	0.60	0.18	2.06		0	0
Nyamathi et al., 2002	1.73	0.53	5.62		0	0
Thorpe et al., 2000	2.60	1.75	3.87		1	1
Zeldis et al., 1992	2.16	1.23	3.79		1	1
Random Effects	1.44	0.87	2.38			
$I^2=68\%$; $Q=15.5$, $p=0.15$						
Kwiatkowski et al., 2002	4.04	1.97	8.29		1	0
Murrill et al., 2002	4.52	2.48	8.24		0	1
Random Effects	4.32	2.72	6.84			
$I^2=0\%$; $Q=0.1$, NS						
Incidence Studies						
Study	Rate Ratio	Lower limit	Upper limit	Group Comparison	Quality ^a	Power ^b

Prevalence Studies						
Study	Odds Ratio	Lower limit	Upper limit	Group Comparison	Quality ^a	Power ^b
Butler et al., 2004	1.43	0.50	4.12		0	0
Patrick et al., 2001	1.59	0.88	2.88		1	0
Dolan et al., 2005	0.98	0.49	1.96		1	-
Random Effects	1.31	0.87	1.99			
I ² =0%; Q=1.1, NS						
Thorpe et al., 2002	0.68	0.24	1.96		1	1
Hagan et al., 2001	0.28	0.09	0.90		1	1
Random Effects	0.45	0.19	1.08			
I ² =18%; Q=1.2, NS						

^aQuality scores: 1 = 14, 0 = < 14

^bPower scores: 1 = sufficiently powered comparison. 0 = underpowered comparison

Table 4

HCV incidence across diverse racial/ethnic IDU groups

Study	Racial/ethnic category	Total IDU tested	HCV positive	Person Years	Incidence/100 PY
Butler et al. (2004)	Aboriginal	10	6	25.0 ^a	24.0
	Non-Aboriginal	19	8	47.6 ^a	16.8
Dolan et al. (2005)	Other	–	28	130.0	22.0
	Aboriginal	–	11	53.0	21.0
Hagan et al. (2001)	White/Other	261	50	261.0	19.2
	African-American	56	3	56.0	5.4
Hahn et al. (2002)	Non-White	39	8	38.8 ^a	20.6
	White	156	40	142.4 ^a	28.1
Maher et al. (2006)	Non-ethnic minority	265	36	163.8	22.0
	Ethnic minority	103	32	57.2	55.9
Patrick et al. (2001)	Aboriginal	24	14	32.2 ^b	43.5 ^a
	Other	47	12	63.1 ^b	19.0 ^a
	White	84	36	112.7 ^b	31.9 ^a
Thorpe et al. (2002)	African-American	67	4	60.5 ^a	6.6 ^a
	Hispanic	60	8	54.2 ^a	14.8 ^a
	White	226	17	204.2 ^a	8.3 ^a

^aCalculated.^bEstimated.