HHS Public Access

Author manuscript

Hepatology. Author manuscript; available in PMC 2017 December 01.

Published in final edited form as:

Hepatology. 2016 December ; 64(6): 1969–1977. doi:10.1002/hep.28677.

Prevalence of chronic liver disease and cirrhosis by underlying cause in understudied ethnic groups: the Multiethnic Cohort

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Abstract

Chronic liver disease (CLD) and cirrhosis are major sources of morbidity and mortality in the United States. Little is known about the epidemiology of these two diseases in ethnic minority populations in the United States. We examined the prevalence of CLD and cirrhosis by underlying etiologies among African Americans, Native Hawaiians, Japanese Americans, Latinos and whites in the Multiethnic Cohort. CLD and cirrhosis cases were identified using Medicare claims between 1999 and 2012 among the fee-for-service participants (n=106,458). We used ICD-9 codes, body mass index, history of diabetes mellitus and alcohol consumption from questionnaires to identify underlying etiologies. A total of 5,783 CLD (3,575 CLD without cirrhosis and 2,208 cirrhosis) cases were identified. The prevalence of CLD ranged from 3.9% in African Americans and Native Hawaiians to 4.1% in whites, 6.7% in Latinos and 6.9% in Japanese. Nonalcoholic fatty liver disease (NAFLD) was the most common cause of CLD in all ethnic groups combined (52%), followed by alcoholic liver disease (ALD) (21%). NAFLD was the most common cause of cirrhosis in the entire cohort. By ethnicity, NALFD was the most common cause of cirrhosis in Japanese Americans, Native Hawaiians, and Latinos, accounting for 32% of cases. ALD was the most common cause of cirrhosis in whites (38.2%), while hepatitis C virus was the most common cause in African Americans (29.8%).

Conclusions—We showed racial/ethnic variations in the prevalence of CLD and cirrhosis by underlying etiology. NAFLD is the most common cause of CLD and cirrhosis in the entire cohort. The high prevalence of NAFLD among Japanese Americans and Native Hawaiians are novel findings and studies elucidating the causes are warranted.

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Conflicts of interest: the authors disclose no conflicts

Keywords

fatty liver; steatosis; Hepatitis C Virus; minority populations

Liver disease is a major health problem in the United States (US). According to the Centers for Disease Control and Prevention (CDC), chronic liver disease (CLD) and cirrhosis were the 12th leading cause of death in the US in 2013, accounting for more than 36,000 deaths (1). In a recent analysis, however, Mayo Clinic researchers showed that liver disease-related mortality in the US has been underestimated during the past two decades, and the figure was closer to 66,000 deaths annually (2).

Although chronic infection with hepatitis C virus (HCV) and alcoholic liver disease are major causes of CLD in the US, nonalcoholic fatty liver disease (NAFLD) has become the most common cause (3–6). Data from the National Health and Nutrition Examination Survey (NHANES) showed that while the prevalence of most major causes of CLD was stable, the prevalence of NAFLD increased steadily from 1988 to 2008 (4). A recent study from the United Network for Organ Sharing (UNOS) database showed that, between 2004 and 2013, the number of new waitlist liver transplant registrants with NAFLD increased by 170%, compared with 45% for ALD and 14% for HCV, making NALFD now the second most common cause of cirrhosis leading to liver transplant after HCV (7).

It has been shown that there are racial/ethnic differences in the prevalence of CLD and cirrhosis in the US (3, 8), however, the data are limited and restricted to a few ethnic/racial populations (3). A recent study suggested that some ethnic/racial groups that are not well studied could have a higher burden of liver disease; the researchers suggested conducting more detailed research in other ethnicities (9). Moreover, the prevalence of etiologies and common causes of CLD and cirrhosis within each ethnic/racial group have not been fully investigated. For instance, while it is known that the prevalence of HCV infection in African Americans is higher compared to other ethnic/racial groups (10), it is unknown if it is the most common cause of CLD and cirrhosis in this population.

In this study we examined the prevalence of both CLD and cirrhosis by underlying etiology in a large cohort of African Americans, Native Hawaiians, Japanese Americans, Latinos and Whites participating in the Multiethnic Cohort (MEC) study. Our data highlight the detailed etiologies of CLD and cirrhosis in each racial group and provide new information for understudied ethnic groups, such as Native Hawaiians and Japanese Americans.

Methods

Study population

The MEC is a prospective cohort of more than 215,000 men and women, aged 45–75 years, enrolled between 1993 and 1996. The MEC study design and baseline characteristics have been described in detail previously (11). Japanese Americans comprised the largest subgroup within each sex (28% of men and 25% of women), followed by whites (24% and 22%), Latinos (24% and 21%), African Americans (13% and 19%), and Native Hawaiians (6% and 7%). The baseline mailed questionnaire assessed diet, lifestyle, weight and height,

family and personal medical history and, for women, menstrual and reproductive history and hormone use. Since baseline, there have been four follow-up questionnaires, and between 1995 and 2006, blood and urine specimens were collected from ~70,000 participants for biomarker and genetic studies. Incident cancers in the cohort are identified through annual linkage to the Hawaii Tumor Registry, the Cancer Surveillance Program for Los Angeles County, and the California State Cancer Registry; these cancer registries are part of the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) program. Deaths are determined through annual linkage to state death certificate files in California and Hawaii, and periodic linkage to the National Death Index. The MEC participants older than 65 years were linked to Centers for Medicare Services (CMS) claims (1999–2012) using Social Security number, sex, and date of birth; 93% of these participants were successfully linked (12). For this analysis, we excluded participants who were not from the five major ethnic groups (N=12,008) or who were not fee-for-service (FFS) members (N=64,904). The characteristics of FFS vs. non FFS members are shown in Supplemental Table 1; the proportion of FFS participants was higher in Hawaii than in California, higher among whites, Japanese Americans and Hawaiians than among African Americans and Latinos, and higher among participants with a higher educational level. A total of 106,458 eligible participants were available for analysis. The average length of FFS enrollment was 6.7 years, ranging from 5.9 years for Latinos to 7.8 years for Japanese Americans. The Institutional Review Boards for the University of Southern California and the University of Hawaii approved this study.

Case ascertainment

We identified CLD and cirrhosis cases using 1 inpatient or ≥ 2 outpatient/carrier qualifying claims during a one-year period between 1999 and 2012 using International Classification of Diseases (ICD) 9th revision codes. CLD was identified using the ICD-9 codes: 571.0–571.9. Cirrhosis was identified using the following codes: 571.2 (cirrhosis with alcoholism); 571.5 (cirrhosis no mention of alcohol); 456.0, 456.1, 456.20, and 456.21 (esophageal varices); 567.23 (spontaneous bacterial peritonitis); 572.2 (hepatic encephalopathy); and 572.4 (hepatorenal syndrome). A total of 5,783 CLD cases (3,575 without cirrhosis and 2,208 with cirrhosis) were identified among the study population.

Etiology of CLD and cirrhosis

We followed published criteria (13) to define the underlying liver disease of CLD and cirrhosis cases in the MEC (Figure 1). We first identified cases with HCV and/or hepatitis B virus (HBV). These cases were categorized as of HCV, HBV, or HCV and HBV etiology regardless of any additional etiologies. Alcoholic liver disease (ALD) was identified as the cause in cases with alcohol use disorders (alcohol-related CLD, alcohol dependence, alcohol abuse, alcohol mental disorders, alcoholic polyneuropathy, alcoholic cardiomyopathy, and alcoholic pancreatitis). NAFLD was identified as etiologic for cases without any other cause (including HCV, HBV, hemochromatosis, primary biliary cirrhosis, primary sclerosing cholangitis, Wilson's disease, HIV, Alpha-1-Antitrypsin deficiency and autoimmune hepatitis) and with a baseline BMI ≥30 kg/m^2, diabetes mellitus or NAFLD ICD-9 code 571.8. Diabetes was identified via Medicare claims, as well as from baseline and follow up questionnaires. Using the American Association for the Study of Liver Diseases (AASLD)

guidelines (14), women with NAFLD who reported >14 drinks/week and men with NAFLD who reported >21 drinks/week on the alcohol baseline questionnaire were reclassified to ALD. If cases did not satisfy any of the above criteria, they were classified as cryptogenic.

Statistical analysis

We determined the number of cases with CLD or cirrhosis among MEC participants who were enrolled in the Medicare-FFS between 1999 and 2012. We examined the prevalence of CLD and cirrhosis by underlying etiologies across race/ethnicity by dividing the number of cases with the population size. We also examined whether the prevalence of CLD types differed between whites and the other groups using logistic regression adjusting for sex and duration of Medicare enrollment (in years). All analyses were conducted using SAS version 9.3 (SAS Institute, Inc., Cary, NC). All P values were two-sided.

Results

The characteristics of CLD cases in the MEC

We identified 5,783 cases of CLD with and without cirrhosis among the study population between 1999 and 2012 (Table 1). Among these cases, 2,990 (51.7%) had NAFLD; 1,195 (20.7%) had ALD; 498 (8.6%) had HCV; 180 (3.1%) had HBV; 168 (2.9%) had HCV and HBV; 376 (6.5%) were classified as cryptogenic; and the rest of the cases were due to other causes of liver disease. The average age at the first CLD claim was 72.9 years, with the oldest found among cryptogenic cases (74.4 years). The ethnicity breakdown was 39.5% Japanese Americans, 24.9% Latinos, 19.9% whites, 10.5% African Americans, and 5.2% Native Hawaiians. There were more women (56.6%) than men (43.3%). The average alcohol intake (ethanol g/day) was highest among ALD cases (36.5 g/day) and lowest among NAFLD cases (5.1 g/day). The average BMI was highest among NAFLD cases (28.7 kg/m^2) and lowest among cryptogenic cases (25.3 kg/m^2).

Prevalence of CLD by underlying etiology in the MEC

Table 2 shows the prevalence of CLD with and without cirrhosis in the MEC by underlying cause and race/ethnicity. We found that Japanese Americans (6.9%) and Latinos (6.7%) had the highest prevalence of CLD, while the prevalence was lower in whites (4.1%) and in African Americans and Native Hawaiians (3.9% each). The highest prevalence of NAFLD was observed in Japanese Americans (4.4%), followed by Latinos (3.1%), Native Hawaiians (2.3%), whites (1.7%) and African Americans (1.5%). The highest prevalence of ALD was observed in Latinos (1.6%), followed by whites (1.2%), African Americans (1.0%), Native Hawaiians (0.9%) and Japanese Americans (0.8%). The patterns were similar when analysis was restricted to participants with at least 12 months of FFS enrollment (data not shown). In a time trend analysis based on 3 periods (1999–2003, 2004–2008, 2009–2012), the proportion of NAFLD increased over time in all racial/ethnic groups (Supplemental Tables 2–3).

Analysis of Causes of CLD by Cirrhosis Status

We investigated the underlying cause of CLD without cirrhosis (N=3,575 cases) and with cirrhosis (N=2,208 cases) across race/ethnicity (Table 3). Because of the small numbers, we

combined HCV cases with those with both HCV and HBV. NAFLD was the most common cause of CLD without cirrhosis (65.6%) in all racial/ethnic groups, followed by alcoholic liver disease (ALD) (16.5%), HCV (5.3%) and others (5.3%). Interestingly, CLD without cirrhosis due to NAFLD was highest in Japanese Americans (72.9%) and Native Hawaiians (72.5%), followed by Latinos (61.1%) and whites (55.8%), and lowest in African Americans (49.3%). Compared to whites, the differences in Japanese Americans ($P \leq 0.0001$), Native Hawaiians ($P \leq 0.0001$) and African Americans ($P < 0.05$) were statistically significant. CLD without cirrhosis due to ALD was highest in African Americans (27.0%), followed by Latinos (20.8%), whites (23.6%), Native Hawaiians (17.6%) and Japanese Americans (10.4%). CLD without cirrhosis due to HCV was highest in African Americans (12.8%), followed by Latinos (7.7%), whites (5.9%), Japanese Americans (3.2%) and Native Hawaiians (2.6%).

NALFD was the most common cause of cirrhosis in the entire cohort (29.3%). When stratified by race/ethnicity, NAFLD was the most common cause of cirrhosis in Japanese Americans (32.3%), Native Hawaiians (31.5%), and Latinos (31.9%); these numbers were significantly different from whites (21.7%) (Table 3). ALD was the most common cause of cirrhosis in whites (38.2%), while HCV and NAFLD were the most common causes of cirrhosis in African Americans (29.8% and 29.2%), respectively.

Discussion

In this large cohort study, we showed racial/ethnic variations in the prevalence of CLD and cirrhosis by underlying etiology. NAFLD was the most common cause of CLD and cirrhosis in the entire cohort. When analyzed by race/ethnicity, NAFLD was the most common cause of CLD without cirrhosis in all ethnic groups and of CLD with cirrhosis in Japanese Americans, Latinos and Native Hawaiians. HCV and ALD were the most common causes of cirrhosis in African Americans and whites, respectively. Our study reports several novel findings: (1) NAFLD is the most common cause of cirrhosis in this US multiethnic cohort; (2) although the prevalence of NAFLD is lowest in African Americans, it is the most common cause of CLD and second most common cause of cirrhosis in this group; and (3) the prevalence of NAFLD in Japanese Americans is higher than in Latinos and other ethnic groups.

With the striking increase in the prevalence of obesity, NAFLD has become the most common cause of CLD (15). Our results underscored the importance of NAFLD as the major cause of CLD across all ethnic groups in the MEC. However, our data likely underestimated the prevalence of NAFLD although it is consistent with other epidemiological studies that did not include imaging studies (16, 17). Studies that used imaging modalities or liver biopsy have shown higher prevalence of NAFLD which can be up to 45% in the US population (18–21). In the US, the prevalence of NAFLD has been reported to be higher among Latinos than in other racial/ethnic groups and lowest among African Americans (20, 22–25). We found that the lowest prevalence of NAFLD was observed in African Americans but, surprisingly, the prevalence in Japanese Americans was higher than that of Latinos in our cohort.

Data on the prevalence of NAFLD in Japanese Americans are scant; most published data on this population originated from Japan. The prevalence of NAFLD in the general population in Japan was estimated to be 9–14% in 1988 (26). During the past 20 to 30 years, the prevalence of NAFLD in Japan has increased gradually (27). Recent data from Japan showed that the prevalence of fatty liver based on ultrasonography among healthy adults was 30% (28), and as high as 72% when at least two of the risk factors (obesity, diabetes or hyperlipidemia) were present (29, 30). In a prospective study, metabolic syndrome was shown to be a strong predictor of NAFLD in apparently healthy Japanese men and women (31).

Despite the relatively low prevalence of NAFLD in African Americans, NAFLD was the most common cause of CLD in this group. Browning *et al.* and others showed that African Americans have significantly lower NAFLD prevalence compared to Latinos and non-Hispanic whites after adjusting for obesity and diabetes (22, 32). One explanation for these ethnic differences in NAFLD prevalence may be due to ethnic differences in body fat distribution (33–35), fat metabolism (15) and/or genetics (*e.g.* a genetic variant in *PNPLA3* (rs738409) associated with hepatic fat content and hepatic inflammation was found to be less prevalent in African Americans) (36–38). Compared to whites with similar total adiposity, Latinos and Asians are more likely, and African Americans less likely, to accumulate fat in the abdominal visceral compartment and in the liver (22, 39, 40). In the MEC, we previously reported that compared to white women with comparable total adiposity, Japanese-American women had more visceral and liver fat as measured by MRI (41). It also has been suggested that African Americans have a different lipoprotein metabolism (42). One can argue that the diagnosis of NAFLD was possibly overestimated in African Americans in our cohort using the definition criteria. However, all of our subjects had a diagnosis of CLD and NAFLD cases were classified as such after ruling out other causes of liver disease. Indeed, in our study, CLD and cirrhosis due to HCV were higher in African Americans compared to others. HBV was also prevalent in in African Americans in our cohort. These findings are consistent with HCV and HBV data in African Americans shown in other studies supporting the methodology we have used (3, 43, 44).

Our study shows that NAFLD was the most common cause of cirrhosis in the entire MEC cohort. When we stratified by race/ethnicity, NAFLD was the most common cause in Japanese, Latinos and Native Hawaiians, while ALD and HCV were the most common causes of cirrhosis in whites and African Americans, respectively. However, a recent report based on a transplant database (2004 to 2013) (7) showed that NAFLD was the second leading indication for liver transplant among cirrhosis patients after HCV. It is possible that because NAFLD is a silent disease, underdiagnosed and associated with many comorbidities, NAFLD patients are not eligible for the transplant list (45). It is likely that ALD cases were excluded from the transplant list. A study among US Veterans (2001–2013) reported that HCV was the most common cause of cirrhosis (13). The most likely explanation of the discrepancy between the VA results and ours is that the VA participants were predominantly men and the prevalence of HCV in this population was approximately double that of comparable age and sex groups of US population (13). Our data highlights the importance of NAFLD as a leading cause of cirrhosis, especially in certain ethnic groups.

Alcohol continues to be one of the major contributors to liver disease in the US. Indeed, we found that ALD is the most common cause of cirrhosis in whites. While the prevalence of alcohol abuse among racial/ethnic groups have been characterized, data on racial disparities in CLD and cirrhosis due to alcohol are scant. Older data described higher heavy drinking in African Americans and Latinos (46, 47). However, recent data from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) showed that whites are more likely to die from alcoholic liver cirrhosis than blacks (http://pubs.niaaa.nih.gov/publications/Surveillance100/Cirr11.htm). Our data are consistent with the NIAAA findings.

The limitations of our study include case identification based on Medicare claim files, probable underestimation of the true prevalence of NAFLD given the lack of biochemical and imaging testing and possibly other etiologies (*e.g.* HCV and HBV related diseases due to under diagnosis), and restriction to FFS participants. Our data included mostly people aged on average 60 years and older when liver diseases are usually more severe (48, 49). The strengths of our study include its large size and population-based design, longitudinal follow up, inclusion of five racial/ethnic populations, including understudied populations, and information on several factors including alcohol intake, BMI, and diabetes status. Our study has investigated all major causes of liver diseases in these racial groups using a published method (13). To our knowledge, our study is the largest detailed study with multiple ethnic groups to date on racial disparities in liver diseases.

In conclusion, in this large multiethnic cohort, we found NAFLD to be the most important cause of CLD and cirrhosis. The high prevalence of NAFLD among Japanese Americans and Native Hawaiians is a novel finding and studies elucidating the causes of this are warranted. Better screening, diagnostic and management approaches need to be implemented to face this growing epidemic. Further studies are needed to confirm our findings and to investigate underlying genetic, metabolic and nutritional causes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We thank the MEC participants for their participation and commitment.

Grant support: National Cancer Institute grants CA164973 and CA186203

Abbreviations

ALD	alcoholic liver disease
BMI	body mass index
HBV	hepatitis B virus
HCV	hepatitis C virus
NAFLD	nonalcoholic fatty liver disease

SD standard deviation

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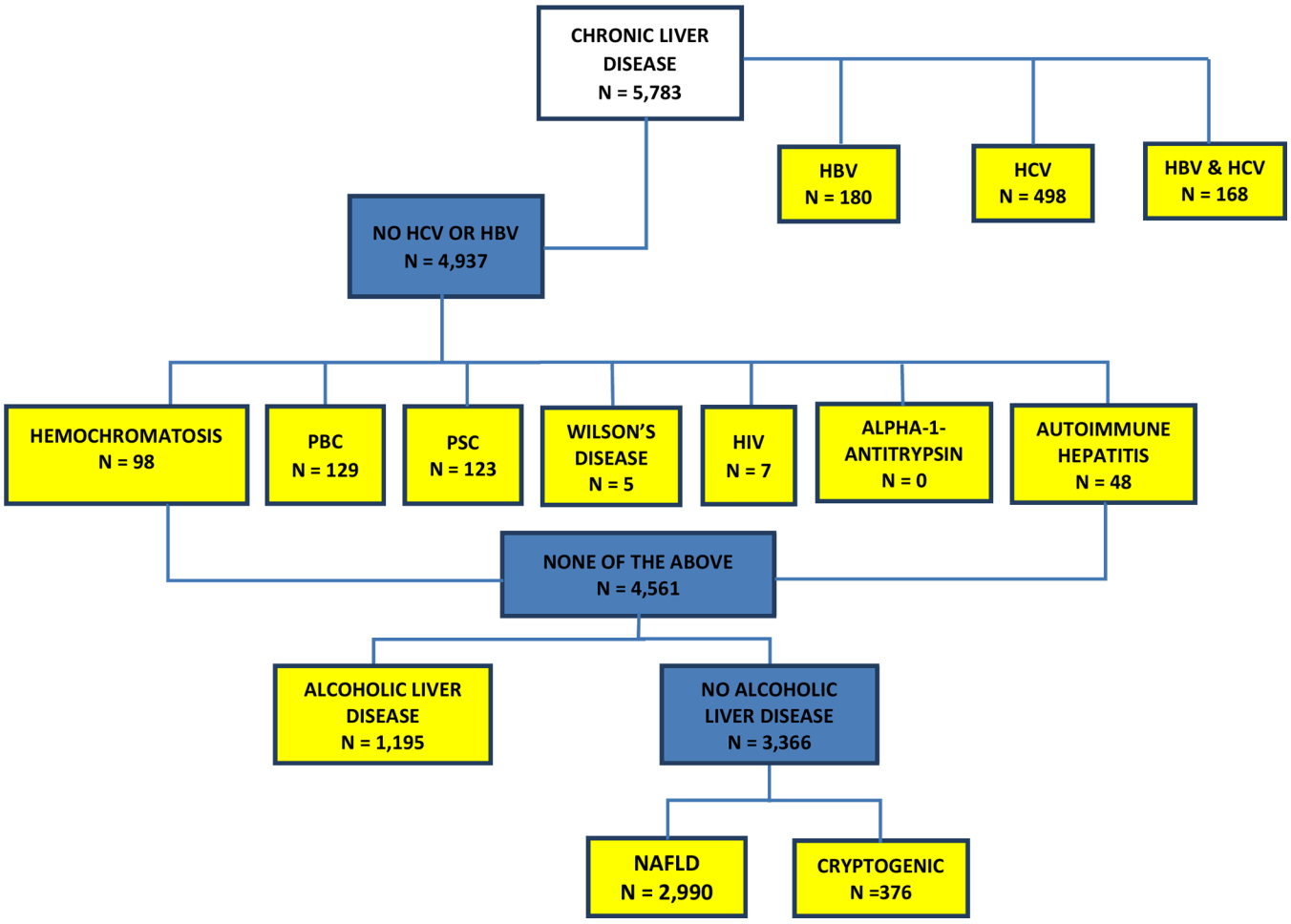

Figure 1. Identification of the cause of liver disease

Alcoholic Liver Disease: alcohol dependence (303.00–303.93), alcohol abuse (305.00–305.03), alcohol mental disorders (291.0–291.9), alcoholic polyneuropathy (357.5), cardiomyopathy (425.5), pancreatitis (577.0, 577.1), and alcoholic-related CLD (571.0–571.3); **Alpha-1-Antitrypsin**: 273.4; **Autoimmune Hepatitis:** 571.42; **Cirrhosis:** 571.2, 571.5, hepatic encephalopathy (572.2), hepatorenal syndrome (572.4), esophageal varices (456.0, 456.1, 456.20, 456.21), and spontaneous bacterial peritonitis (567.23); **CLD:** 571.0–571.9; **Hemochromatosis:** 275.0; **Hepatitis B:** 0702, 07020, 07021, 07022, 07023, 0703, 07030, 07031, 07032, 07033, V0261; **Hepatitis C:** 07041, 07044, 07051, 07054, 07070, 07071, V0262; **HIV:** 042; **NAFLD:** Diabetic or BMI ≥ 30 kg/m^2 or 571.8. Women with NAFLD who drank >14 drinks/week and men with NAFLD who drank >21 drinks/week were reclassified to alcoholic liver disease; **Primary Biliary Cirrhosis (PBC):** 571.6; **Primary Sclerosing Cholangitis (PSC):** 576.1; **Wilson's Disease:** 275.1

Table 1

Characteristics of Chronic Liver Disease Cases, Overall and by Etiologies in the Multiethnic Cohort, 1999–2012

	HBV	HBV & HCV	HCV	ALD	NAFLD	Other	Cryptogenic	All Cases
No. of cases, N (%)	180 (3.1%)	168 (2.9%)	498 (8.6%)	1,195 (20.7%)	2,990 (51.7%)	376 (6.5%)	376 (6.5%)	5,783
Age at date of 1st claim, mean (SD)	72.0 (6.8)	70.6 (6.0)	71.4 (6.3)	72.9 (6.3)	73.2 (6.6)	73.1 (6.5)	74.4 (6.7)	72.9 (6.5)
Race/Ethnicity, N (row %)								
White	31 (2.7%)	24 (2.1%)	98 (8.5%)	345 (30.1%)	467 (40.7%)	80 (7.0%)	103 (9.0%)	1,148
African American	19 (3.1%)	28 (4.6%)	102 (16.7%)	160 (26.3%)	239 (39.2%)	23 (3.8%)	38 (6.2%)	609
Native Hawaiian	9 (3.0%)	2 (0.7%)	8 (2.7%)	67 (22.3%)	174 (57.8%)	21 (7.0%)	20 (6.6%)	301
Japanese American	79 (3.5%)	55 (2.4%)	99 (4.3%)	276 (12.1%)	1,452 (63.6%)	171 (7.5%)	151 (6.6%)	2,283
Latino	42 (2.9%)	59 (4.1%)	191 (13.2%)	347 (24.1%)	658 (45.6%)	81 (5.6%)	64 (4.4%)	1,442
Sex, N (row %)								
Male	73 (2.9%)	78 (3.1%)	218 (8.7%)	741 (29.5%)	1,097 (43.7%)	144 (5.7%)	159 (6.3%)	2,510
Female	107 (3.3%)	90 (2.7%)	280 (8.6%)	454 (13.9%)	1,893 (57.8%)	232 (7.1%)	217 (6.6%)	3,273
Alcohol intake (g/day)								
Mean†	11.6	7.8	13.1	36.5	5.1	13.0	15.5	14.8
Body mass index (kg/m^2)								
Mean†	28.2	27.3	27.2	27.2	28.7	27.8	25.3	27.9

†Adjusted for sex and race/ethnicity distribution.

Table 2

Prevalence of Chronic Liver Disease and Cirrhosis by Etiologies in the Multiethnic Cohort, 1999–2012

All	HBV n=180	HBV & HCV n=168	HCV n=498	ALD n=1,195	NAFLD n=2,990	Others n=376	Cryptogenic n=376	Total Cases n=5,783	Base Population n = 106,458
White	31 (0.1 %)	24 (0.1 %)	98 (0.3%)	345 (1.2%)	467 (1.7 %)	80 (0.3 %)	103 (0.4 %)	1,148 (4.1 %)	28,225
African American	19 (0.1 %)	28 (0.2 %)	102 (0.6 %)	160 (1.0 %)	239 (1.5 %)	23 (0.1 %)	38 (0.2 %)	609 (3.9 %)	15,782
Native Hawaiian	9 (0.1 %)	2 (0.0 %)	8 (0.1 %)	67 (0.9 %)	174 (2.3 %)	21 (0.3 %)	20 (0.3 %)	301 (3.9 %)	7,647
Japanese American	79 (0.2 %)	55 (0.2 %)	99 (0.3 %)	276 (0.8 %)	1,452 (4.4 %)	171 (0.5 %)	151 (0.5 %)	2,283 (6.9 %)	33,312
Latino	42 (0.2 %)	59 (0.3 %)	191 (0.9 %)	347 (1.6 %)	658 (3.1 %)	81 (0.4 %)	64 (0.3 %)	1,442 (6.7 %)	21,492

* Using the following ICD-9 diagnosis codes for CLD: 571.0–571.9, we selected any 1 inpatient claim or 2 outpatient/carrier claims during a 1 year period for years 1999–2012 to identify CLD cases. Cirrhosis include cirrhosis with alcoholism (571.2), cirrhosis no mention of alcohol (571.5), esophageal varices (456.0, 456.1, 456.20, 456.21), spontaneous bacterial peritonitis (567.23), hepatic encephalopathy (572.2), and hepatorenal syndrome (572.4).

Table 3

Distribution of Chronic Liver Disease and Cirrhosis by Etiologies and Race/Ethnicity in the Multiethnic Cohort, 1999–2012

CLD without cirrhosis	**HBV n=98**	**HCV† n=190**	**ALD n=591**	**NAFLD n=2,344**	**Others n=191**	**Cryptogenic n=161**	**All Cases n=3,575**
White	15 (2.3%)	38 (5.9%)	151 (23.6%)	357 (55.8%)	41 (6.4%)	38 (5.9%)	640
African American	11 (3.6%)	39 (12.8%) **	82 (27.0%)	150 (49.3%) **	8 (2.6%) **	14 (4.6%)	304
Native Hawaiian	2 (1.0%)	5 (2.6%)	34 (17.6%)	140 (72.5%) *	7 (3.6%)	5 (2.6%)	193
Japanese American	50 (2.8%)	56 (3.2%) **	183 (10.4%) *	1,283 (72.9%) *	100 (5.7%)	88 (5.0%)	1,760
Latino	20 (2.9%)	52 (7.7%)	141 (20.8%)	414 (61.1%)	35 (5.2%)	16 (2.4%) **	678
Cirrhosis	**HBV** n=82	**HCV†** n=476	**ALD** n=604	**NAFLD** n=646	**Others** n=185	**Cryptogenic** n=215	**All Cases** n=2,208
White	16 (3.1%)	84 (16.5%)	194 (38.2%)	110 (21.7%)	39 (7.7%)	65 (12.8%)	508
African American	8 (2.6%)	91 (29.8%) *	78 (25.6%) **	89 (29.2%) **	15 (4.9 %)	24 (7.9%) **	305
Native Hawaiian	7 (6.5%)	5 (4.6%) **	33 (30.6%)	34 (31.5%) **	14 (13.0 %)	15 (13.9%)	108
Japanese American	29 (5.5%)	98 (18.7%)	93 (17.8%) *	169 (32.3%) **	71 (13.6%) **	63 (12.0%)	523
Latino	22 (2.9%)	198 (25.9%) *	206 (27.0%) *	244 (31.9%) *	46 (6.0%)	48 (6.3%) *	764

† HCV cases with HBV were also included in this group.

* P≤0.0001 compared with Whites and adjusted for sex and duration of Medicare enrollment.

** P<0.05 compared with Whites and adjusted for sex and duration of Medicare enrollment.