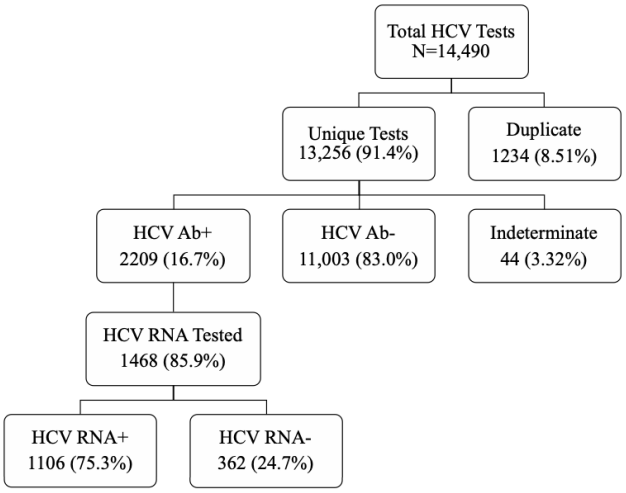
from the electronic medical record for all tested, with risk factors collected from those who tested positive for HCV Ab (HCV Ab+). Multivariate logistic regression was performed.

Figure 1. HCV Ab and HCV RNA positivity among people screened for HCV in the Dallas County Jail from 2015 to 2019 (N=14490).



Results. The prevalence of HCV Ab+ was 16.7% in the Dallas County Jail; 75.3% of those who tested HCV Ab+ were also HCV RNA+ (Figure 1). The HCV Ab+ incidence rate was 13.5 cases per 1000 person-years. People who were HCV Ab+ were more frequently (adjusted odds ratio [95% confidence interval], *p*-value): older (1.07 (1.06-1.07), *p*< 0.001), female (1.24 [1.07-1.44], p=0.004), white (2.12 [1.83-2.45], *p*< 0.001), and in the birth cohort 1945-65 (1.79 [1.44-2.23], *p*< 0.001; Table 1). In earlier birth cohorts (1940s), black men were more often HCV Ab+; in more recent birth cohorts (1990s), white and Hispanic females were more often HCV Ab+ (Figure 2). Among individuals who tested HCV Ab+, IDU was more frequently reported by white individuals, particularly women, compared to black individuals (p< 0.001; Figure 3).

Table 1. Demographic predictors of Hepatitis C Antibody positivity among those undergoing routine blood draws from 2015-19 at the Dallas County Jail (AIC 7041; BIC 7048; df 10; p<0.001).

	HCV Ab+ n=2209	Total Tested N=13212	*p*-value	Univariate Analysis n=10193 aOR (95% CI)	*p*-value	Multivariate Analysis n=7916 aOR (95% CI)	*p*-value
Age (n=13209)	47.0±12.2	38.0±11.8	**<0.001**	1.08 (1.07-1.08)	**<0.001**	1.07 (1.06-1.08)	**<0.001**
Gender			0.181		0.181		**0.003**
Female	447 (26.2%)	2535 (24.9%)		1.08 (0.96-1.22)		1.24 (1.08-1.44)	
Male	1261 (73.8%)	7648 (75.1%)		Referent		Referent	
Race/Ethnicity			**<0.001**				
Non-Hispanic White	862 (39.0%)	3799 (28.8%)		1.88 (1.67-2.12)	**<0.001**	2.08 (1.80-2.41)	**<0.001**
Hispanic White	378 (17.1%)	2296 (17.4%)		1.34 (1.16-1.55)	**<0.001**	1.83 (1.53-2.20)	**<0.001**
Non-Hispanic Black	948 (42.9%)	6967 (52.8%)		Referent		Referent	
Other	20 (0.9%)	140 (1.1%)		0.83 (0.43-1.61)	**0.582**	0.73 (0.34-1.59)	**0.415**
Year of Test			**0.003**				
2015-2016	500 (22.6%)	3019 (22.9%)		---------		---------	
2017	720 (32.6%)	3905 (29.6%)		Referent		Referent	
2018	639 (28.9%)	3976 (30.1%)		0.85 (0.75-0.95)	**0.005**	1.05 (0.91-1.21)	0.499
2019	350 (15.8%)	2312 (17.5%)		0.79 (0.69-0.91)	**0.001**	0.94 (0.79-1.12)	0.526
Birth Cohort (1945-65)			**<0.001**		**<0.001**		**<0.001**
Yes	912 (41.3%)	2027 (15.3%)		6.47 (5.73-7.33)		1.79 (1.44-2.23)	
No	1297 (58.7%)	11185 (84.7%)		Referent		Referent	
Months in Jail (n=7925)	2.43±2.35	2.63±2.91	**0.002**	0.97 (0.95-0.99)	**0.010**	0.97 (0.95-0.99)	**0.029**
Release to			**<0.001**		**<0.001**		
Prison	479 (43.7%)	2297 (36.6%)		1.43 (1.25-1.63)			
Not Prison	618 (56.3%)	3975 (63.4%)		Referent			

Figure 2. Trends of Hepatitis C Antibody prevalence and demographic prevalence ratios by birth year (prevalence ratio= proportion with disease/proportion with exposure) among people at the Dallas County Jail screened from 2017-2019 (N=10183). Demographic prevalence ratios were categorized by race (White, Hispanic, Black) and gender (Male, Female) into six categories. Gray bars represent the overall prevalence of HCV Ab+ by birth year.

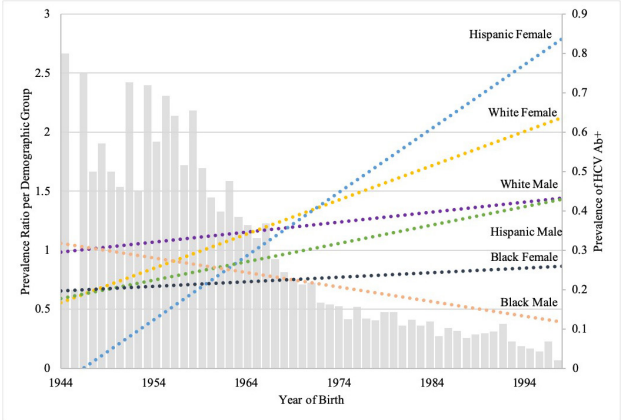
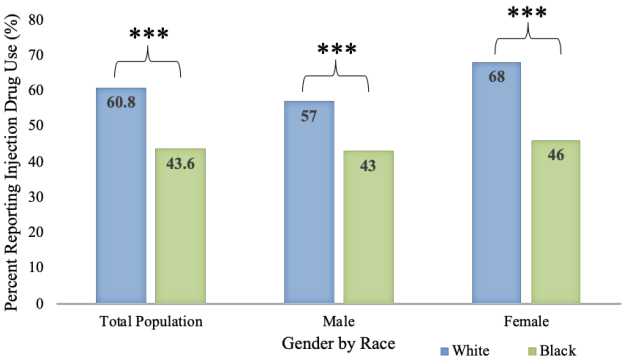


Figure 3. The racial demographics of injection drug use by gender among those who tested HCV Ab positive at the Dallas County Jail in 2017-2018 (n=672; total population p<0.001; male p=0.004; female p=0.008).



Conclusion. The high prevalence and incidence of HCV at the Dallas County Jail argues for routine, universal testing and linkage to treatment. Additionally, demographic trends mirror the IDU epidemic and have valuable implications for risk reduction and treatment interventions.

Disclosures. **Ank E. Nijhawan, MD, MPH**, **Gilead** (Grant/Research Support, Scientific Research Study Investigator, Research Grant or Support)

1065. Hepatitis C Virus Care Cascade Assessment–One Step Closer to Micro-Elimination

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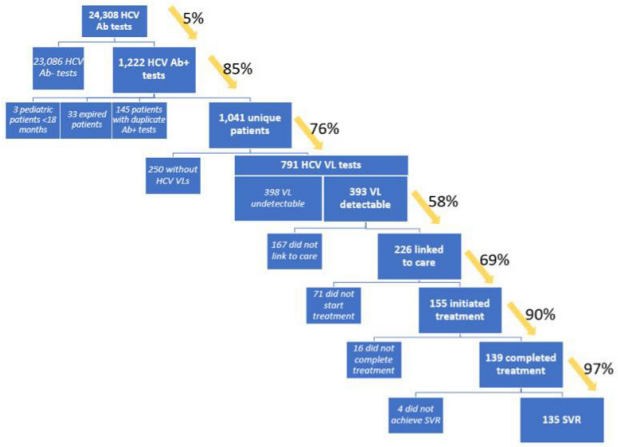
Session: P-48. Hepatitis

Background. Hepatitis C virus (HCV) is a leading cause of advanced liver disease and death. In the United States about 3.5 million people are living with HCV, but only 50% are aware of the infection, 16% are prescribed treatment, and only 9% achieve sustained viral response. The World Health Organization published an HCV elimination goal for 2030 that strives to achieve a 65% reduction in HCV-related deaths and 90% reduction in transmission. An important step toward this goal is micro-elimination at local hospitals by addressing care gaps in the HCV care cascade.

Figure 1



Methods. We created a retrospective cohort of patients who tested positive for HCV antibody (HCV Ab+) between 2016 and 2018 at Tufts Medical Center in Boston, Massachusetts. We assessed achievement of care cascade steps including HCV viral load (VL) testing, linkage to care, treatment initiation, and sustained viral response (SVR). We also assessed patient demographics, clinical factors and HCV risk factors. We used STATA/IC 14.1 to conduct bivariate analysis to identify factors associated with loss to follow-up across each care cascade step.

Results. A total of 24,308 HCV antibody tests were done during this timeframe, of which 5% (n=1,222) were HCV Ab+. After excluding duplicate tests, 1,041 unique patients with HCV Ab+ were included. This cohort had a mean age of 47 years and were 61% male, 66% white, 72% on public insurance, 12% HIV-positive, 13% HCV treatment-experienced. The most frequent HCV risk factor was injection drug use, occurring in 64% of patients. Of patients with HCV Ab+, 76% (n=791) were tested for an HCV VL, of which 50% (n=393) had detectable VL and 50% (n=398) had undetectable VL. Of the patients with a detectable VL, 58% (n=226) were linked with care. Following care linkage, 69% (n=155) initiated treatment, of which 90% (n=139) completed treatment, of which 97% (n=135) achieved

SVR (Figure 1). Factors that were significantly associated with getting a VL test and linking to care included private insurance, HIV co-infection, absence of intravenous drug use and cirrhosis; however, these factors were not significantly associated with achieving subsequent steps.

Conclusion. Assessment of the HCV care cascade at our hospital allowed us to identify clear care gaps and areas needing improvement towards a local micro-elimination.

Disclosures. **All Authors**: No reported disclosures

1066. Immune Escape Mutant Detection Using Commercially Available Methods for Hepatitis B Surface Antigen Serological Testing

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Session: P-48. Hepatitis

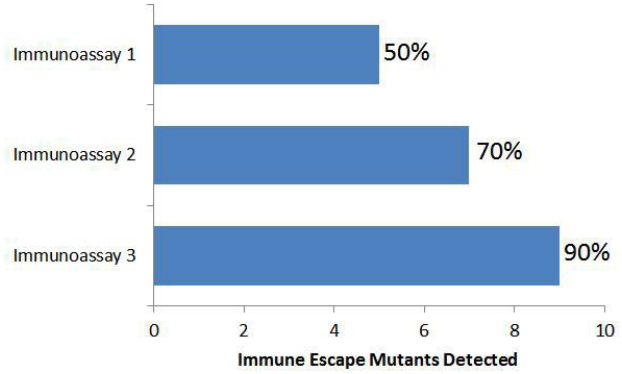
Background. Although overall infection rates of Hepatitis B virus (HBV) in the United States (US) remain stable, as many as 2.2 million persons are still chronically infected with Hepatitis B Virus (HBV)1. Persons who inject drugs (PWID) are at a higher risk of HBV infection and since 2009 three states (KY, TN, WV) have reported up to a 114% increase in cases of acute HBV infection due to higher infection rates among a non-Hispanic white populations (30–39 years), and injection drug users2. Hepatitis B vaccination is recommended as primary prevention for adults who are at increased risk for HBV infection, including PWID. However, data from the National Health Interview Survey indicate that hepatitis B vaccination coverage is low among adults in the general population3, and it is likely to be lower among injection drug users.

Hepatitis B Surface Antigen (HBsAg) is the first serological marker to appear after HBV exposure and infection; this marker is included in the recommended panel for acute hepatitis diagnosis and accurate detection is necessary for early and accurate diagnosis. Serological testing challenges exist for HBsAg due to the high degree of genetic variability which can further be exacerbated by endogenous and exogenous pressures. The immuno-dominant region may have one or more mutations described as immune escape mutations which can decrease or abrogate HBsAg binding to antibodies used in immunoassays. Although the prevalence of these mutations is not well documented in the United States, international studies have shown that up to 79% of HBV-reactivated patients (vs 3.1% of control patients; p< 0.001) carry HBsAg mutations localized in immune-active HBsAg regions4.

Methods. A study was conducted using a panel of 10 unique recombinant HBsAg immune escape mutants. Panel members were tested by commercially available HBsAg serological immunoassays.

Results. It was found that although commercially available HBsAg immunoassays are the primary diagnostic tool for HBV diagnosis, not all HBsAg immune escape mutants are detected, with some method detecting as few as 5 out of 10 of these mutant samples.

Figure 1



Conclusion. Improvement is needed in commercially available methods for the accurate detection of HBsAg.

Disclosures. **Robert Gish, MD**, **Abbott** (Consultant)**AbbVie** (Consultant, Advisor or Review Panel member, Speaker's Bureau)**Access Biologicals** (Consultant)**Antios** (Consultant)**Arrowhead** (Consultant)**Bayer** (Consultant, Speaker's Bureau)**Bristol Myers** (Consultant, Speaker's Bureau)**Dova** (Consultant, Speaker's Bureau)**Dynavax** (Consultant)**Eiger** (Consultant, Advisor or Review Panel member)**Eisai** (Consultant, Speaker's Bureau)**Enyo** (Consultant)**eStudySite** (Consultant, Advisor or Review Panel member)**Exelixis** (Consultant)**Fujifilm/Wako** (Consultant)**Genentech** (Consultant)**Genlantis** (Consultant)**Gilead** (Consultant, Advisor or Review Panel member, Speaker's Bureau)**GLG** (Consultant)**HepaTX** (Consultant, Advisor or Review Panel member)**HepQuant** (Consultant, Advisor or Review Panel member)**Intercept** (Consultant, Speaker's Bureau)**Ionis** (Consultant)**Janssen** (Consultant)**Laboratory for Advanced Medicine** (Consultant)**Lilly** (Consultant)**Merck** (Consultant)**Salix** (Consultant, Speaker's Bureau)**Shionogi** (Consultant, Speaker's Bureau)**Viking** (Consultant)

1067. Interferon-free Hepatitis C Treatment Increases Surrogates of Cardiovascular Disease Risk in Black Veterans

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Session: P-48. Hepatitis

Background. Sustained virologic response (SVR) after hepatitis C virus (HCV) treatment with either Interferon (IFN)-based or IFN-free regimens with direct-acting antivirals (DAAs) has been shown to reduce cardiovascular disease (CVD) events in majority white populations stratified by ASCVD score. However, the effect of IFN-free therapy on lipid profiles after SVR, as an indirect measure of CVD risk, is unknown in Black patients.

Methods. We evaluated HCV-infected Veterans from the Baltimore VA who were treated with DAAs between 2015-2019. We performed a retrospective analysis comparing lipid profile changes following SVR among those with early stage (F0-F2) fibrosis and advanced liver disease (ALD, F3-F4 fibrosis) using two-tailed paired t-tests. Independent t-tests were used to assess differences in lipid profiles based on fibrosis stage in patients with HIV and Type II Diabetes Mellitus (DM2).

Results. Of those treated for HCV (n=1,528), 96% (n=1,474) achieved SVR. Demographics are shown in Table 1. Most patients were Black males (75%) and a minority (2.7%) received statin therapy during treatment (Table 1). Of 1,094 patients for whom data was available, an increase in total cholesterol (TC) and LDL (p< 0.01 for both) was seen an average of 17 months after SVR, regardless of fibrosis stage (Figure 1). A significant decrease in triglyceride levels (p=0.04) was also seen in the ALD group after SVR (Figure 1). Mean pre-treatment HCV RNA level was comparable between fibrosis groups (F0-F2: 6.35 logs, F3-F4: 6.37 logs, p=0.46). There were 101 and 436 patients with HIV and DM2, respectively, for whom pre-treatment liver fibrosis data was available. In both groups, there were significant increases in LDL (p=0.008 (HIV), p=0.003 (DM2)) among patients with ALD following SVR.

Table 1. Baseline characteristics of treated HCV-infected persons who achieved SVR.

	N= 1474
Age, median years (SD)	64.6 (61,69)
Race % (N)	
Black or African American	75 (1110)
White	23 (345)
Other/Unknown/Declined to answer	2 (27)
Sex, %(N) male	97 (1426)
Diabetes, % (N)	35 (500)
Ethnicity, % (N)	
Not Hispanic or Latino	98 (1451)
Other/Unknown/Declined to answer	1.2 (18)
Hispanic or Latino	<1 (5)
HIV, % (N)	7 (101)
Median pre-treatment HCV RNA, IU/L	2,262,252.71
Fibrosis stage pre-treatment, % (N)	
F0-F2	56 (828)
F3-F4	36 (525)
Treatment regimens, % (N)	
Ledipasvir/Sofosbuvir	75 (1106)
Elbasvir/Grazoprevir	8 (124)
Paritaprevir/Ombitasvir/Dasabuvir/ritonavir	6 (85)
Sofosbuvir/Velpatasvir	3 (51)
Glecaprevir/Pibrentasvir	3 (40)
Sofosbuvir/Velpatasvir/Voxilaprevir	1 (15)
Other	4 (53)
Statin use during HCV treatment, % (N)	2.7 (40)