Chronic Hepatitis C in African Americans: Challenges and Opportunities

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Speaker Disclosure

The speaker serves on advisory boards and receives research support from companies with interest in the contents of this CME activity:

- Advisory Boards: AbbVie, Inc., Janssen, Inc.
- Research: Boehringer Ingelheim Pharmaceuticals; Bristol-Myer Squibb, Inc., Gilead Sciences; Eisai, Inc.
Outline

- **Challenges:**
  - Trends in HCV Epidemiology
  - Disease Course and Clinical Outcomes
  - Lower efficacy of PegIFN-based Treatment
  - Barriers to healthcare access and quality

- **Opportunities:**
  - New HCV Screening Recommendations
  - Affordable Care Act of 2010
  - More effective and safer treatments (interferon and ribavirin-free)
Hepatitis C Virus (HCV) in US

- ~3-4 million infected with HCV (NHANES 1999-2002)
- Most common blood-borne viral infection
- Main cause for cirrhosis & primary hepatocellular carcinoma (HCC)
- Indication for >40% of liver transplants
- Increasing mortality rate since 1999
- $2.0 billion in healthcare cost in 2003; rising to $6.7 billion in 2010-2019

Ly et al. AIM 156:271-278, 2012
HCV in African Americans (AA)

- HCV more prevalent in AA compared to Caucasian (CA) or White (3.0% vs. 1.5%)

- Race and Ethnicity (NHANES 1999-2002):
  - 2.6 million (95% CI 2.0-3.4 mill) CA (65%)
  - 920,000 (95% CI 0.7-1.2 mill) AA (22%)
  - 260,000 Mexican Americans (95% CI 0.15-0.43 mill) (6.5%)

HCV in African Americans (AA)

- 12-13% of US pop. 1999-2002 & 22% of HCV infected
- 9% of AA 40-49 years old infected compared to 3.8% of CA
- Est. 1.5-2 million AA may be infected with HCV
  - Liberal est. 5-8 million HCV infected in USA
  - Incarcerated and homeless not sampled by NHANES
  - Incarceration and homelessness more common in AA

Natural History of HCV Infection

- **Exposure (Acute Phase)**
  - 15% Resolved
  - 85% Chronic

- **Cirrhosis**
  - 20% progression
  - Rate accelerated with HIV, HBV, alcohol

- **5-year survival in patients with HCC is <5%**

- **Time (yr)**: 10-30
  - 10yr: Resolution, Chronic, Cirrhosis
  - 20yr: ESLD, HCC, Transplant/death
  - 30yr: Transplant/death

HCC = hepatocellular carcinoma
ESLD = end-stage liver disease
Trends in HCC Incidence in US

NCI SEER Data

Mortality Rate from HCV Exceeds that of HIV

Ly et al. AIM 156:271-278, 2012
Predictors of HCV-related Deaths in 2007

- 73.4% Age 45-59
  - 45-54 39%
  - 55-64 34%
- 70% Males [OR 2.4  95% CI (2.3-2.4)]
- 18% Non-Hispanic Black [OR 1.9 (1.8-2.0)]
- 15% Hispanic [OR 3.4 (3.2-3.50)]
- 2.9% HIV Co-infection [OR 5.8 (5.3-6.4)]
- 3.6% HBV Co-infection [OR 70.3 (63.5-77)]
- 57.2% Chronic Liver Disease [OR 57 (55-59)]
- 19.4% Alcohol-related condition [OR 13 (12.8-14)]

Ly et al. AIM 156:271-278, 2012
Annual Hepatitis C Mortality Rates: Race/Ethnicity

New Opportunities:
- IFN-free Antiviral Treatments
- New Screening Guidelines
- Greater Access to Healthcare
HCV Curable with Treatment: Sustained Virological Response (SVR)

- Tantamount to cure; HCV RNA remains negative in 99%
- Associated with improvement in liver histology (inflammation, fibrosis)
- Less frequent liver-related complications
- Reduced risk of liver decompensation
- Reduced risk of hepatocellular carcinoma
- Reduced liver-related mortality

Advances in Treatment for HCV Genotype 1: SVR (%)
Survival curves for each outcome were constructed using a clock-reset approach; patients who switched from the without SVR to the with SVR group due to successful retreatment during follow-up were censored in the without SVR group at the time of SVR. The time of SVR was then defined as time zero for their further follow-up in the SVR group. Statistical significance between the survival curves in the without and with SVR groups was assessed with univariate Cox proportional hazards regression analyses, including SVR as a time-dependent covariate. The 10-year cumulative occurrence rates for all-cause mortality were 8.9% (95% CI, 3.3%-14.5%) for with SVR and 26.0% (95% CI, 20.2%-28.4%) for without SVR; for liver-related mortality or liver transplantation, 1.9% (95% CI, 0.0%-4.1%) for with SVR and 27.4% (95% CI, 22.0%-32.8%) for without SVR; for hepatocellular carcinoma, 5.1% (95% CI, 1.3%-8.9%) for with SVR and 21.8% (95% CI, 16.6%-27.0%) for without SVR; and for liver failure, 2.1% (95% CI, 0.0%-4.5%) for with SVR and 29.9% (95% CI, 24.3%-35.5%) for without SVR.

Figure Legend:
Survival curves for each outcome were constructed using a clock-reset approach; patients who switched from the without SVR to the with SVR group due to successful retreatment during follow-up were censored in the without SVR group at the time of SVR. The time of SVR was then defined as time zero for their further follow-up in the SVR group. Statistical significance between the survival curves in the without and with SVR groups was assessed with univariate Cox proportional hazards regression analyses, including SVR as a time-dependent covariate. The 10-year cumulative occurrence rates for all-cause mortality were 8.9% (95% CI, 3.3%-14.5%) for with SVR and 26.0% (95% CI, 20.2%-28.4%) for without SVR; for liver-related mortality or liver transplantation, 1.9% (95% CI, 0.0%-4.1%) for with SVR and 27.4% (95% CI, 22.0%-32.8%) for without SVR; for hepatocellular carcinoma, 5.1% (95% CI, 1.3%-8.9%) for with SVR and 21.8% (95% CI, 16.6%-27.0%) for without SVR; and for liver failure, 2.1% (95% CI, 0.0%-4.5%) for with SVR and 29.9% (95% CI, 24.3%-35.5%) for without SVR.
In the US, 2.7–3.9 million people are living with chronic HCV infection; 75% are unaware they are infected.
Three-fourths Chronic HCV in the US Were Born Between 1945 and 1965

Estimated Prevalence by Age Group

New 2012 Recommendations—Individuals Who Should Be Screened for HCV

Baby Boomers Born from 1945–1965

- Adults born during 1945–1965 should receive one-time testing for HCV without prior ascertainment of HCV risk

- As of 2014, this includes all individuals between 49 and 69 years of age!

New 2012 Recommendations—Individuals Who Should Be Screened for HCV

- IDU
- Clotting factors made before 1987
- Received blood/organs before July 1992
- Hemodialysis
- Elevated alanine aminotransferase [ALT] levels
- HIV

Testing also should be performed based on the need for exposure management, including:

- Health care, emergency, and public safety workers after needlestick/mucosal exposure to HCV-positive blood
- Children born to HCV-positive women

New 2012 Recommendations—Reduce Alcohol Use in Patients with HCV

- All persons with identified HCV infection should receive brief alcohol screening, brief counseling if positive, followed by referral to appropriate care and treatment services.

- Screening tools:
  - National Institute on Alcohol Abuse and Alcoholism
  - WHO

Screening for hepatitis C virus infection in adults: clinical summary of U.S. Preventive Services Task Force recommendation.

<table>
<thead>
<tr>
<th>Population</th>
<th>Persons at high risk for infection and adults born between 1945 and 1965</th>
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<tbody>
<tr>
<td><strong>Recommendation</strong></td>
<td>Screen for hepatitis C virus (HCV) infection.</td>
</tr>
<tr>
<td><strong>Grade</strong></td>
<td>B</td>
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<tr>
<td><strong>Risk Assessment</strong></td>
<td>The most important risk factor for HCV infection is past or current injection drug use. Additional risk factors include receiving a blood transfusion before 1992, long-term hemodialysis, being born to an HCV-infected mother, incarceration, intranasal drug use, getting an unregulated tattoo, and other percutaneous exposures. Adults born between 1945 and 1965 are more likely to be diagnosed with HCV infection, either because they received a blood transfusion before the introduction of screening in 1992 or because they have a history of other risk factors for exposure decades earlier.</td>
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<tr>
<td><strong>Screening tests</strong></td>
<td>Anti-HCV antibody testing followed by confirmatory polymerase chain reaction testing accurately identifies patients with chronic HCV infection. Various noninvasive tests with good diagnostic accuracy are possible alternatives to liver biopsy for diagnosing fibrosis or cirrhosis.</td>
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<td><strong>Screening interval</strong></td>
<td>Persons with continued risk for HCV infection (such as injection drug users) should be screened periodically. Evidence on how often screening should occur in these persons is lacking. Adults born between 1945 and 1965 and persons who are at risk because of potential exposure before universal blood screening need only be screened once.</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Antiviral treatment prevents long-term health complications of HCV infection (such as cirrhosis, liver failure, and hepatocellular carcinoma). The combination of pegylated interferon (α-2a or α-2b) and ribavirin is the standard treatment for HCV infection. In 2011, the U.S. Food and Drug Administration approved the protease inhibitor boceprevir and telaprevir for the treatment of HCV genotype 1 infection (the predominant genotype in the United States).</td>
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<tr>
<td><strong>Balance of Benefits and Harms</strong></td>
<td>On the basis of the accuracy of HCV antibody testing and the availability of effective interventions for persons with HCV infection, the USPSTF concludes that there is a moderate net benefit to screening in populations at high risk for infection. The USPSTF concludes that there is also a moderate net benefit to 1-time screening in all adults in the United States born between 1945 and 1965.</td>
</tr>
<tr>
<td><strong>Other Relevant USPSTF Recommendations</strong></td>
<td>The USPSTF has made recommendations on screening for hepatitis B virus infection in adolescents, adults, and pregnant women. These recommendations are available at <a href="http://www.uspreventiveservicestaskforce.org">www.uspreventiveservicestaskforce.org</a>.</td>
</tr>
</tbody>
</table>

For a summary of the evidence systematically reviewed in making this recommendation, the full recommendation statement, and supporting documents, please go to www.uspreventiveservicestaskforce.org.
Affordable Care Act 2010

- ~30,000,0000 to gain access
- Requires private health plans to provide preventive services (HCV screening) given an A/B grade by USPSTF, without cost sharing
- Incentives for Medicaid programs to cover these preventive services
- Prohibits insurance companies from declining persons because of preexisting conditions
Disparities in HCV Treatment: Veterans Administration

- AA & Hispanics less likely to be a treatment candidate in according to clinicians
- AA and Hispanic receive fewer interferon prescriptions for HCV
- AA more likely to decline treatment
- Hispanics compared to CA
  - More likely to be considered eligible for treatment (VA and NIH Consensus 2002)
  - More medication dose reductions
  - More early treatment discontinuation and dropout for side effects
- Variability in Treatment
  - Patient effects- 23%
  - Provider & facility effects- 27%

Disparities: Primary Hepatocellular Carcinoma

- HCC incidence increased 200% 1975-2000
- Incidence and mortality rate 2 fold higher in African Americans and other ethnic minorities
- AA and Hispanics more likely to have HCV as risk factor for HCC
- AA have more advanced HCC tumor stage at diagnosis
- AA less likely to receive local or surgical (resection, liver transplantation) therapy than CA, even with tumor localized to liver

Davila Clin Gastro Hep 4:104-110, 2006; Sloane et al. JNMA 98 (12); 1934, 2006
Liver Transplantation for HCV 2002-2006 (MELD era)

- Liver disease more severe (MELD score) when placed on wait list (barrier to referral)
- No difference in removal for death/becoming too sick & odds of receiving transplant within 3 years
- AA more likely to undergo transplantation for HCV, and less likely to receive live donor
- AA have lower survival rate after transplantation that is abolished by adjusting for the higher MELD score and older age
- Liver graft loss more frequent in AA
- AA more likely to have recurrent HCV and allograft rejection as causes for graft loss than Whites
- AA with graft loss less likely to undergo repeat liver transplantation
Hepatitis C in African Americans: NMA Consensus Panel

- Increase HCV awareness
- Improve accuracy of HCV incidence and prevalence data
- Reduce or eliminate under-diagnosis
- Training to current and future health providers
- Greater inclusion of African Americans in clinical trials
- Increase access to care and treatment
- Address HCV in criminal justice system
- Increase monitoring of outcomes of HCV screening
- Apply and analyze new treatments

National Medical Association Available at www.NMAnet.org
Priority Area 1:
Evaluating Providers and Communities to Reduce Health Disparities
Aging of HCV-Infected Persons in the US: Disease Progression

Projected Prevalence of Chronic HCV, Cirrhosis, and Complications

Projected Number of Patients With Decompensated Cirrhosis and Hepatocellular Carcinoma

Number of Cases

- Decompensated Cirrhosis
- Hepatocellular Carcinoma (HCC)

Year

Minorities Underrepresented in HCV Clinical Trials

- Milestones in HCV Treatment in US
  - 1991: Interferon (IFN) approved
  - 1998: IFN & Ribavirin approved
  - 2001/2002: Peginterferon & ribavirin
  - AA and Hispanics < 5% of enrollees
- 2004-2006: 3 clinical trials of peginterferon & ribavirin for HCV genotype 1 in AA
- 2009: clinical trial of peginterferon & ribavirin for HCV genotype 1 in Latinos
- AA and Latinos poorly represented in direct acting antiviral clinical trials
Sofosbuvir, PEGIFN, and RBV for HCV GT1 Naïve (NEUTRINO)

Lawitz et al. n engl j med 368;20: 1878
NIH SPARE: Sofosbuvir and RBV in Difficult-to-Treat GT1 Pts

- Subjects primarily GT1a (70%), male (63%), black (83%), IL28B CT/TT (80%)
- BMI > 30: 48%; advanced liver disease: 23%; HCV RNA > 800,000 IU/mL: 62%

<table>
<thead>
<tr>
<th>Part 1 (early-stage fibrosis)</th>
<th>Wk 24</th>
<th>Viral Response, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sofosbuvir 400 mg + RBV 1000/1200 mg (n = 10)</td>
<td>90</td>
<td>90</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Part 2 (all stages of fibrosis)</th>
<th>EOT</th>
<th>SVR4</th>
<th>SVR12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sofosbuvir 400 mg + RBV 600 mg (n = 25)</td>
<td>88</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>Sofosbuvir 400 mg + RBV 1000/1200 mg (n = 25)</td>
<td>96</td>
<td>68</td>
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</tbody>
</table>

- In viral kinetic study involving 10 low-dose and 15 full-dose RBV subjects, HCV RNA decrease was rapid with median HCV RNA reduction of $4.14 \log_{10} \text{IU/mL}$ by Day 7
- Both regimens well tolerated and resulted in significant improvement of hepatic inflammation ($P < .0001$)

SYNERGY: Sofosbuvir/Ledipasvir FDC ± GS-9669 or GS-9541 in GT1

- NIAID nonrandomized parallel-arm phase II trial
- African American (88%), male (72%), infected with GT-1a (70%), had high HCV VL (>800k) (70%) and IL28B non-CC haplotype (82%).
- Primary endpoint: SVR12

Patients with GT1 HCV

- Treatment naive, F0-F4 (n = 20)
- Treatment naive, F0-F3 (n = 20)
- Treatment-experienced, F0-F3 (n = 20)

Sofosbuvir 400 mg QD/ledipasvir 90 mg QD; GS-9669 500 mg QD; GS-9451 80 mg QD.

SYNERGY: High Response Rates With 6-Wk Triple-Drug Regimens

- Very high response rates with all-oral therapy without RBV
- 1 patient in SOF/LDV FDC + GS-9669 arms relapsed and 1 missed SVR4 visit
- No serious AEs leading to discontinuation

Summary & Conclusions

- Chronic HCV is more prevalent and is associated with more morbidity and higher mortality in African Americans.
- AA with HCV face many barriers to healthcare access and quality.
- Maximum utilization of birth cohort and risk-based screening, better healthcare and more effective HCV treatments provide tools to decrease suffering.
- Coupled with awareness and strategies to overcome barriers could eliminate the racial disparities in HCV morbidity and mortality.
Hepatitis C: A Crisis in the African American Community

Peer-reviewed Consensus Paper

National Medical Association Available at www.NMAnet.org
Natural History of HCV Infection

15% Resolved
85% Chronic

20% Cirrhosis

~20 year progression rate accelerated with HIV, HBV, alcohol

5-year survival in patients with HCC is <5%²

6%/yr ESLD
4%/yr HCC
3-4%/yr Transplant/death

HCC = hepatocellular carcinoma
ESLD = end-stage liver disease
## Table 2. Response during and after Treatment Period.

<table>
<thead>
<tr>
<th>Response</th>
<th>NEUTRINO Study</th>
<th>FISSION Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SOF+PEG+RBV for 12 Wk (N=327)</td>
<td>SOF+RBV for 12 Wk (N=253)</td>
</tr>
<tr>
<td><strong>HCV RNA &lt;25 IU/ml — no./total no. (%)</strong></td>
<td></td>
<td></td>
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<tr>
<td>During treatment</td>
<td></td>
<td></td>
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<tr>
<td>At 2 wk</td>
<td>299/327 (91)</td>
<td>231/251 (92)</td>
</tr>
<tr>
<td>At 4 wk</td>
<td>321/325 (99)</td>
<td>249/250 (&gt;99)</td>
</tr>
<tr>
<td>At last observed measurement</td>
<td>326/327 (&gt;99)</td>
<td>249/253 (98)</td>
</tr>
<tr>
<td>After end of treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At 4 wk</td>
<td>302/327 (92)</td>
<td>187/253 (74)</td>
</tr>
<tr>
<td>At 12 wk</td>
<td>295/327 (90)</td>
<td>170/253 (67)</td>
</tr>
<tr>
<td>Virologic breakthrough during treatment — no. (%)</td>
<td>0</td>
<td>1 (&lt;1)</td>
</tr>
<tr>
<td>Relapse in patients with HCV RNA &lt;25 IU/ml at end of treatment — no./total no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients who completed treatment</td>
<td>25/320 (8)</td>
<td>71/242 (29)</td>
</tr>
<tr>
<td>Patients who did not complete treatment</td>
<td>3/6 (50)</td>
<td>3/7 (43)</td>
</tr>
</tbody>
</table>
### Sofosbuvir for HCV GT 1, 2, & 3 Treatment-Naive

#### Figure 1. Rates of Sustained Virologic Response in the NEUTRINO and FISSION Studies, According to Subgroup and Baseline Factors.
The HCV Treatment Paradigm is Evolving Rapidly

Mid 1980s | Late 1990s | 2002 | 2011 | 2014/5?

SVR in TN GT-1 (%)a

IFN1,2 | IFN/RBV1,3–5 | PegIFN/RBV3–8 | PI + PegIFN/RBV7,8 | IFN-free?

13–17%b | 29–38% | 38–52% | NSOC 67–75% | 70–95%

a. The red bar denotes the range in SVR for each treatment; b. All genotypes

NSOC, new standard of care; PegIFN, pegylated interferon; PI, protease inhibitor; RBV, ribavirin; SVR, sustained virological response; TN, treatment naïve

Additive relationship for hepatitis C virus clearance for HLA class II and IL-28B single nucleotide polymorphisms. Among 919 persons with spontaneous clearance and 1482 with viral persistence, the proportions with viral clearance are shown for rs12979860 in the IL-28B region by ethnicity, rs4273729 in the HLA class II region by ethnicity, and both rs4273729 and rs12979860 by ethnicity. Because these results are from case–control studies, the absolute effects may not be generally representative because they will vary by the ratio of clearance to persistence in the study population. See Figure S16 of Supplement 3 for a similar plot of the relative effects (odds ratio). IL-28B = interleukin-28B.
Peginterferon & Ribavirin for HCV GT-1 in AA and CA

48 Week Treatment

Subject Race
Jeffers et al. Hepatology, June 2005
IL28B (rs12979860) SNP
Associated with SVR

- IL28B (chr. 19q13) gene encodes IFN-lambda (type III IFN)
- Induced by virus infection and mediates antiviral response by Jak-STAT signaling pathway

Lower Ribavirin Exposure in AA

B. RBV AUC

<table>
<thead>
<tr>
<th>AUC&lt;sub&gt;0-7&lt;/sub&gt; (ng/mL*day)</th>
<th>AA (n=71)</th>
<th>CA (n=73)</th>
<th>P value</th>
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<tr>
<td>4142</td>
<td>5117</td>
<td>0.0034</td>
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<td>155541</td>
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<td>337390</td>
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<td>0.0606</td>
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</table>
SVR: RBV AUC\textsubscript{0-7} Conditional on IL28B SNP

RBV AUC\textsubscript{0-7} Threshold for SVR: \textgreater 4480 ng/ml x day
Why Peginterferon & Ribavirin less effective in AA

- AA have defect in HCV-Specific CD4 T helper 1 response (IFNγ) prior to treatment
- AA with poor response day 0-28 of therapy have higher T cell expression of Programmed Death-1 (exhausted phenotype)
- No major differences in HCV isolates between AA and CA
  - HCV genetic diversity in core, NS3, and NS5A associated with treatment response in AA and CA
  - Difference in E1 and NS2 in HCV genotype 1b unlikely to play major role in treatment outcome

NHANES III Demographic Characteristics of Persons Infected with HCV Genotypes 1, 2, and 3

AA Less Likely to Clear HCV Spontaneously

- Risk factors for acute HCV do not differ between AA and CA

- NHANES 2005-2008: 14,750 tested
  - 192 Anti-HCV + (1.32 ± .11%)
    - 149 HCV RNA+ (75.94 ± 4.72%) (i.e. CHC)
    - 43 HVC RNA- (Cleared)
      - AA: 9.25 ± 3.47%
      - White: 27.21 ± 6.49%
      - Hispanics: 31.21 ± 9.09%

- AA race only predictor for lack of HCV clearance

From: Genome-Wide Association Study of Spontaneous Resolution of Hepatitis C Virus Infection: Data From Multiple Cohorts


Figure Legend:

Manhattan plot summarizing the genome-wide association results in 919 persons with spontaneous resolution of hepatitis C virus infection and 1482 persons with chronic hepatitis C virus infection.

Each point corresponds to a P value from a test of association for a single nucleotide polymorphism. The $-\log_{10} P$ values are plotted by location of the person’s single nucleotide polymorphism across the genome. The dashed line represents an accepted level of genome-wide significance ($P = 5 \times 10^{-8}$). Single nucleotide polymorphisms in the HLA and IL-28B region on chromosomes 6 and 19, respectively, exceed this threshold. IL-28B = interleukin-28B.
Ledipasvir/Sofosbuvir ± Ribavirin
Genotype 1: Previously Untreated

Figure S2. Sustained Virologic Response by Patient Characteristics, Intention to Treat analysis

<table>
<thead>
<tr>
<th></th>
<th>LDV/SOF For 12 Weeks</th>
<th>LDV/SOF+RBV For 12 Weeks</th>
<th>LDV/SOF For 24 Weeks</th>
<th>LDV/SOF+RBV For 24 Weeks</th>
</tr>
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<tbody>
<tr>
<td><strong>Overall</strong></td>
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<tr>
<td>Age</td>
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<td>&lt;65 years</td>
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<td>≥65 years</td>
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<td>Non-black</td>
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<td>Interferon Eligibility Status</td>
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<td>HCV Genotype</td>
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<tr>
<td>Cirrhosis</td>
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<td>No</td>
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<td>Yes</td>
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<td>Baseline HCV RNA (U/mL)</td>
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<td>&lt;600,000</td>
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<tr>
<td>≥600,000</td>
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<tr>
<td>Baseline BMI (kg/m²)</td>
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<tr>
<td>&lt;30</td>
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<tr>
<td>≥30</td>
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<tr>
<td>Baseline ALT</td>
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<tr>
<td>≤1.5 x ULN</td>
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<tr>
<td>&gt;1.5 x ULN</td>
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<tr>
<td>IL28B genotype</td>
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<td>CC</td>
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<tr>
<td>Non-CC</td>
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</tbody>
</table>

SVR12 Rate (%)
Barriers to Healthcare for HCV

- Access (e.g., insurance status, ability to pay for healthcare) is the most important predictor of the quality of healthcare across racial and ethnic groups (IOM)
- Black Americans twice as likely to have no private health insurance
- Latina Americans 3 times less likely to have private health insurance