The Association of Race/Ethnicity, Sex, and Comorbidity with Hepatitis C Genotype 1 Treatment Response

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Citation

Abstract
Most studies comparing the response of African American (AA) patients to other groups for treatment of hepatitis C virus (HCV) have been performed in academic centers in the setting of clinical trials. In general, AA patients have lower response rates that are only partially explained by unfavorable prognostic factors, such as infection with genotype 1, high viral load, insulin resistance, obesity, hypertension, and others. We examined response rates associated with these adverse prognostic factors within race/ethnicity-sex strata for a cohort of patients treated at an inner-city hospital-based, safety-net clinic. From 2004-2008, 88 evaluable AA and 30 other patients received standard therapy for HCV consisting of weekly injections of peg interferon alfa-2 plus oral weight-based ribavirin. The predicted probability of a favorable virologic outcome ranged from 89.0% (95% CI: 63.7% - 97.3%) for other females to 4.1% (95% CI: 0.9% - 17.9%) for AA females. Conclusion: Important interactions occur among race/ethnicity, sex, and co-morbidity in response to HCV therapy. Increasing co-morbidity places AA patients at greatest risk of non-response and raises the possibility that more aggressive treatment of co-morbidity might increase response rates in the vulnerable patients.

INTRODUCTION
Although the number of Americans with HCV infection has declined about ten-fold from the 1980s to 2001[,], estimates are that as many as four million persons have been infected with HCV. No vaccine is available to prevent HCV, unlike for Hepatitis A and B, and carriers can transmit the disease via blood and body fluids throughout the remainder of their life. HCV has an enormous effect on morbidity and mortality. Perhaps eighty percent of infected people in the United States show signs of chronic infection, which over a period of 5-20 years may lead to liver cirrhosis (20%) and or liver cancer[]. Moreover, approximately 25% of patients with cirrhosis will die from hepatic failure or require liver transplantation. To some extent, treatment of HCV may slow progress of fibrosis.

The current standard for treatment of HCV is pegylated interferon alpha 2 (PEG-IFN) in combination with ribavirin (RBV)[,], which has improved clinical efficacy over single agent PEG-IFN. The overall sustained viral response rates following combined PEG-IFN+RBV is 54 – 56% after a 48-week course of therapy. Patients with genotype 1 infection typically have a 40 – 50% overall likelihood of response to 48 weeks of therapy[]. Those with genotypes 2 or 3 infection can expect to experience an improved response to treatment, with a 75 – 80% SVR after only 24 weeks of treatment[]. Other conventional predictors of low responsiveness, such as high initial viral load or advanced liver fibrosis, affect the response to PEG-IFN less than they do with non-PEG IFN[].

Although treatment is available for HCV, we lack a sound understanding of the complex interaction between race/ethnicity, comorbidity, and treatment response. For reasons that are not entirely clear, the sustained virological response (SVR) rate in AA patients was less than the response rate for other patients in two multicenter studies[]. To investigate these racial/ethnic differences more closely, we determined the overall antiviral response within a specified four year time period in a community based safety net setting, with emphasis on the interactions of race/ethnicity, sex and co-morbidity.

METHODS
We monitored the antiviral response for the cohort of all patients with HCV genotype 1 entered into treatment in the Hepatitis Clinic at Cooper Green Mercy Hospital (CGMH), an inner-city public hospital in Birmingham, Alabama from January 2004 thru July 2008. Patients with hepatitis not due to HCV (HBV, alcoholic, autoimmune, others) were
excluded. For all treated patients, we captured patient demographics, initial and subsequent viral loads, decision to treat, risk factors for drug response, and adverse effects. Before treatment was offered, all patients were engaged in an extensive education program including discussion with staff, formal classes, and brochures and videos illustrating the natural history of HCV, treatment options, anticipated response to treatment, and potential adverse effects. A directed search of the patient data was used to prepare this publication; no change in therapeutic decision was based upon the collection of this data. The CGMH Institutional Review Board (IRB) approved the use of anonymous demographic and incidence data collection for the purpose of preparing this publication.

Patients were offered treatment if their initial viral load was > 400,000 IU, they were symptomatic for HCV with chronic fatigue, weakness, abdominal pain, nausea), and they had elevated liver enzymes (AST > 2 times upper limit of normal). Patients not meeting these three criteria could still be considered for an individual treatment decision, if after an explanation of why they were not optimal candidates for treatment they still desired to be treated.

Patients received either PEG-IFN alpha 2a (180ug) or 2b (weight based) once per week, with weight based ribavirin twice a day (unless contraindicated). We did not show preference for the version of Peg-IFN offered other than that dictated by individual insurers. Patients were checked at 4 weeks of treatment when possible for a rapid virological response (RVR), and also at 12 weeks for an early virological response (EVR). Patients who were virus negative at 12 weeks of treatment had their medication continued for the complete 48 week duration. Patients who experienced at least a 2 log drop in viral load from baseline at 12 weeks of therapy but were not virus negative were continued to 24 weeks of therapy, and then if virus negative, were continued for 48 weeks of treatment, otherwise treatment was stopped.

Between three to six months after the end of a full course of treatment, a quantitative viral load was again measured (whenever the opportunity was available), to determine whether a SVR occurred. Not all patients made themselves available for all study time points, and not all patients started on therapy had finished therapy at the cutoff for collection of study data.

For this study, virologic response in patients was defined as meeting any of the following three criteria:

1. No detectable virus 3-6 months after stop of therapy (sustained viral response SVR);
2. Completed treatment with undetectable virus at end of treatment (EOT) even though no follow up assessment of virologic status was made at 3-6 month post-treatment; or
3. Still undergoing treatment at the time of data analysis with at least one favorable indicator of viral response: RVR or EVR.

We added the second and third criteria to reflect the real-world setting of a safety-net hospital. The second response option allowed us to include patients lost to follow-up after having responded up to EOT. The third criteria broadened the definition of Virologic Response to allow an overall picture of response rate in our patient population. Although these criteria are broader than those commonly used in randomized clinical trials, the more over-encompassing definition of response rate allowed us to analyze our single site data set to better understand within the purpose of our study the complex interactions between race/ethnicity, sex, and comorbidity with treatment response.

Non-response (NR) in patients was defined as either:

1. Did not experience at least a 2 log drop in virus titer at 12 weeks, or
2. Experienced at least a 2 log drop in virus titer at 12 weeks, but who were not virus-negative at 24 weeks.
3. Patients with detectable virus 3-6 months after cessation of therapy after an initial response (EOT) were classified as experiencing Relapse and were included for data analysis along with the non-responder group.
4. Patients with a drop in viral load during treatment, followed by an increase while still on therapy were classified as experiencing Breakthrough, and also were were included for data analysis along with the non-responder group.

Patients were excluded from the analysis if they were:

1. Not treated,
2. Lost to follow up,
3. Discontinued treatment due to an adverse effect of medication (AE), before a determination of viral response could be made
4. Deceased for unrelated reason before a determination of viral response could be made.

STATISTICAL ANALYSIS
We first examined distributions and univariate statistics for the overall sample. Next we performed bivariate analyses to study the associations among patient characteristics and response status using two-sample t-tests and chi-square tests with p-values ≤ 0.05 considered significant. Because of small cell sizes, exact tests of statistical significance were used for comparisons based on categorical independent variables.

The presence of diabetes mellitus (DM) and hypertension (HTN) were highly collinear, requiring these co-morbidities to be represented as a single, composite categorical variable. More specifically, we constructed a three-level categorical variable for the following states: (1) absence of both DM and HTN (no co-morbidity), (2) presence of either DM or HTN, and (3) presence of both DM and HTN.

Taking a dichotomous variable indicating virologic response as the patient-level outcome, multivariable logistic regression determined the independent associations for multiple clinical covariates. Because of the small number of outcomes, we exercised special caution to prevent over fitting. In particular, we omitted age and initial viral load from the final multivariable model because these variables were not significant in interim multivariable analyses. In addition, we entered the comorbidity score as a linear variable to conserve degrees of freedom and because the relationship between the comorbidity score and the outcome was monotonically decreasing. The power of the main logistic regression model to discriminate between the presence or absence of the outcome state (response to treatment) was examined with the c-statistic.

Based on the final multivariable model which included race/ethnicity, sex and the comorbidity score, we calculated the predicted probability of a favorable viral response. For each predicted probability, non-parametric re-sampling without replacement (bootstrapping) generated 95% bias-corrected and accelerated confidence intervals. Because of small cell sizes, predicted probabilities for certain patient profiles were not reported. All analyses were performed with STATA 10.0 SE.

RESULTS
Overall, 88 patients were treated for HCV with Genotype 1 and had follow up data available for analysis (Table 1).

Figure 1
Table 1: Overview of Patients Treated for Hepatitis C with Genotype 1, 2004 – 2007

<table>
<thead>
<tr>
<th>Included in Main Analysis</th>
<th>Non-responders</th>
<th>Responders</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean years</td>
<td>50.0 (24.0)</td>
<td>38.6 (13.9)</td>
<td>0.037</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>52</td>
<td>44</td>
<td>0.827</td>
</tr>
<tr>
<td>Male</td>
<td>36</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian American</td>
<td>57</td>
<td>37</td>
<td>0.000</td>
</tr>
<tr>
<td>Asian American female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic-Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other males</td>
<td>56</td>
<td>36</td>
<td>0.000</td>
</tr>
<tr>
<td>Other females</td>
<td>53</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>African American males</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American females</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>58</td>
<td>49</td>
<td>0.005</td>
</tr>
<tr>
<td>Diabetes</td>
<td>56</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>19</td>
<td>14</td>
<td>0.000</td>
</tr>
<tr>
<td>Co-morbidity Score</td>
<td>12</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Baseline Viral Load</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 400,000</td>
<td>8</td>
<td>5</td>
<td>0.000</td>
</tr>
<tr>
<td>≥ 400,000</td>
<td>80</td>
<td>72</td>
<td>0.782</td>
</tr>
</tbody>
</table>

Table 2 presents the characteristics of the study sample by treatment response. The average age was 50.4 years, 42% were female and 65% were AA. The co-morbidity profile was considerable with 69.8% of patients having diabetes, hypertension, or both. Most patients had a high (> 400,000) baseline viral load (80%).

Figure 2
Table 2: Patient Characteristics by Virologic Response

Table 2 presents the characteristics of the study sample by treatment response. The average age was 50.4 years, 42% were female and 65% were AA. The co-morbidity profile was considerable with 69.8% of patients having diabetes, hypertension, or both. Most patients had a high (> 400,000) baseline viral load (80%).

The overall response rate (Table 1) was 43% (38/88). We
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found the following (Table 2) gradient of decreasing favorable response to treatment: other females (80%), other males (67%), AA males (31%) and AA females (15%) (p < 0.001). There was a monotonically decreasing response rate with increasing comorbidity (p < 0.001). For patients with both diabetes and hypertension, the response rate was only 13%. There was also a trend for patients with higher baseline viral titers to have a lower response rates.

The final multivariable model, which included the sex-ethnicity groups and comorbidity scores as independent variables, discriminated between the presence and absence of response with a c-statistic of 0.86 (95% CI: 0.77 - 0.92). Based on the final multivariable model, Table 3 presents the predicted probabilities of a favorable virologic response (PPFVR) for ethnicity-sex strata. Overall, predicted response rates are lower for African Americans and lowest for African American females. Within each race/ethnicity group, the PPFVR decreased as comorbidity increased.

**Figure 3**

Table 3: Predicted Probability (95% CI) of Favorable Virologic Response (PPFVR) by Race/Ethnicity, Sex, and Comorbidity Score (n = 88)*

<table>
<thead>
<tr>
<th>Comorbidity Score</th>
<th>PPFVR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other males</td>
<td>0</td>
<td>87.2 - 97.2</td>
</tr>
<tr>
<td>Comorbidity Score 0</td>
<td>0</td>
<td>60.1 - 79.2</td>
</tr>
<tr>
<td>Comorbidity Score 1</td>
<td>66.0</td>
<td>34.2 - 89.6</td>
</tr>
<tr>
<td>Comorbidity Score 2</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Other females</td>
<td>0</td>
<td>89.0 - 97.3</td>
</tr>
<tr>
<td>Comorbidity Score 0</td>
<td>69.7</td>
<td>35.4 - 90.0</td>
</tr>
<tr>
<td>Comorbidity Score 1</td>
<td>69.7</td>
<td>35.4 - 90.0</td>
</tr>
<tr>
<td>Comorbidity Score 2</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>African American males</td>
<td>0</td>
<td>59.1 - 83.0</td>
</tr>
<tr>
<td>Comorbidity Score 0</td>
<td>59.1</td>
<td>29.4 - 83.0</td>
</tr>
<tr>
<td>Comorbidity Score 1</td>
<td>59.1</td>
<td>29.4 - 83.0</td>
</tr>
<tr>
<td>Comorbidity Score 2</td>
<td>59.1</td>
<td>29.4 - 83.0</td>
</tr>
<tr>
<td>African American females</td>
<td>0</td>
<td>10.5 - 31.6</td>
</tr>
<tr>
<td>Comorbidity Score 0</td>
<td>10.5</td>
<td>0.9 - 31.6</td>
</tr>
<tr>
<td>Comorbidity Score 1</td>
<td>10.5</td>
<td>0.9 - 31.6</td>
</tr>
<tr>
<td>Comorbidity Score 2</td>
<td>10.5</td>
<td>0.9 - 31.6</td>
</tr>
</tbody>
</table>

*Confidence intervals are bias corrected and accelerated from non-parametric sampling with replacement (bootstrapping) with 5,000 replicates. **Defined as baseline viral titer ≥ 400,000 copies/mL. PPFVR for sex-ethnicity-comorbidity combinations with cell sizes < 5 are not reported.

**DISCUSSION**

In this population of patients with HCV treated in an inner-city, safety-net setting, we found overall response rates similar to those documented from previous randomized controlled trials. We did use a more liberalized definition of viral response, because our primary interest was to examine the associations of race/ethnicity, sex, and comorbidity with viral response to treatment. In addition, we found a striking decrease in response rate with increasing co-morbidity, and the deleterious effects of co-morbidity were most pronounced for AA females. Overall antiviral response to HCV with standard treatment at our institution is 43%, which is slightly lower than both the VIRAHEP_C [12] and the Jeffers et al. multi-center studies (about 53%). We analyzed overall response data for genotype 1 only, to allow comparison with other studies for genotype 1. Our methodology is appropriate within the context of our study goals and our patient population, as discussed below. We interpreted out data in light of a number of factors which are known or suspected to affect the antiviral response to therapy, and which were measured in our study.

Gender - Data from several studies indicate that males have a lower SVR than do females [12, 10, 13]. At CGMH, we observed a 41% response for males, and 44% response for females. This difference was not statistically significant but the trend was consistent with published data.

Race/ethnicity – African Americans make up approximately 12% of the US population, but they account for 22% of cases of chronic HCV infection, and 65% of our treated and evaluable population presented herein [14]. For reasons that are not entirely clear, the SVR in AA patients has been found to be less than that for non-AA.

Two prospective studies, VIRAHEP-C [12] and Jeffers et al., Peginterferon alfa-2a (40kd) and ribavirin treatment for black American patients with chronic HCV genotype 1 [13], compared the effects of Pegasys (PEG-IFN alpha 2a) plus RBV treatment in AA and non-AA patients with HCV Genotype 1 infection. In the VIRAHEP-C study [12], 28% of AA patients and 52% of non-AA patients with untreated HCV Infection achieved a SVR with IFN alpha 2a + RBV. In Jeffers et al. [13], 26% of AA patients and 39% of non-AA patients achieved SVR with IFN alpha 2a + RBV. Prior to the development and results from these prospective studies, a retrospective analysis of pooled data from Phase II and III IFN alpha 2a + RBV studies had also showed that AA patients with chronic hepatitis C were generally less likely to respond to IFN therapy than non-AA patients with chronic hepatitis C [13].

Data from our treatment center (Table 3) showed a response rate for AA patients of 26%, which is consistent with other studies. Our overall response rate of 43% is also consistent with other researchers, but for non-AA the overall response rate (74%) was much higher than reported elsewhere (52% and 39%).

Comorbidity - Studies have shown a negative contribution of
many underlying and or concurrent medical conditions to the response rate for HCV. Several of these co-morbid conditions (DM, HTN, obesity, elevated cholesterol) have a particularly high prevalence in the AA community. Our data do not suggest an encouraging response to treatment for African Americans with diabetes and hypertension.

Mehta [18] found that among persons 40 years of age or older, those with HCV infection seemed to be twice as likely as those without HCV infection to have type 2 DM. After adjustment for other risk factors for DM (ethnicity, sex, body mass index, poverty index, and previous drug and alcohol use) the prevalence of type 2 DM with HCV infection who were 40 years of age or older increased nearly four-fold. In contrast, DM was not associated with HCV infection for those younger than 40 years.

Several lines of evidence point to the ability of HCV infection to cause type 2 DM through progressive liver damage[19,20]. This idea is supported by the liver's role in carbohydrate metabolism; the association of type 2 DM with other causes of cirrhosis; and evidence that liver transplantation can reverse both glucose intolerance and insulin resistance associated with diabetes. Moreover, many recent clinic-based investigations among patients with advanced HCV infection observed an increased prevalence of type 2 DM. This appeared most commonly among chronic HCV-infected patients with advanced fibrosis or cirrhosis and is possibly attributable to mediation by pro-inflammatory cytokines such as IL-6 and TNF-α [18,19]. In patients who develop Type 1 DM, during IFN treatment, there is most often a genetic predisposition and association with HLA haplotypes DR3, DR4, DQ2 and DQ8 [20,21]. Lastly, in our own clinic, we have also observed IFN therapy causing diabetes indirectly through pancreatitis.

From this perspective, diabetes may be a marker for more advanced liver damage from long-standing HCV infection. In addition, DM may accelerate liver damage from HCV. Although it is interesting to speculate that treating DM more aggressively may slow the progression of liver damage from HCV, this hypothesis remains to be proven.

Genotype - Overall, more than 70% of HCV clinical isolates in the United States are of Genotype 1[22]. AA are known to have a higher proportion of Genotype 1, which is associated with a lower response rate than for genotype 2 or 3. Eighty four percent of our patients had genotype 1, and for ease of data comparison to published studies, we only evaluated response in patients with Genotype 1. Our overall response rate for genotype 1 was clinically somewhat lower than those other researchers have noted based upon pure genotype 1 populations.

Initial High Viral Load – Although the response rate is known to be inversely proportional to initial viral load, 43% (n=34) of our (80) patients with initial high viral load (HVL) responded to treatment. We found that HVL seemed to have a consistent response across age and sex strata. Interestingly, modeling of response rate showed an interaction between baseline HCV RNA levels and race/ethnicity.[12] After controlling for sex, fibrosis scores, and amount of peginterferon taken, Conjeevaram[13] found that SVR rates were significantly lower in AA than for non-AA with high viral levels but were more similar at lower viral levels.

Multiple Simultaneous Risk Factors - Patients with multiple risk factors (DM, HTN, HVL) experience poor antiviral responses, as we have noted for AA with genotype 1. Unfortunately, there is evidence that patients with multiple risk factors are offered treatment less often. Kanwal[14], for example, studied treatment parameters for 5701 patients with HCV at a VA referral medical center. They found that patients with genotype 1 and in particular AA patients with genotype 1 were less likely to receive treatment. Data presented here show that AA M and AA F with multiple risk factors for poor response only rarely respond, and it may be reasonable to not be overly aggressive in treating this patient subgroup, especially if no signs of progressive liver fibrosis are present.

Alberti[15] evaluated the effect on response rate of gender, age, body mass index, HCV genotype, baseline viral load, baseline transaminase, histologic diagnosis (presence or absence of cirrhosis), comorbidities (present or absent) and type (2a or 2b) of PEG-IFN. Their patient population was in many ways similar to ours, as were their findings - a SVR rate for genotype 1 of 41.2%, compared to our 51%. Four factors increased the probability of achieving SVR: genotype 1a, absence of co-morbidities, baseline viral load < 1 mil, and pretreatment with PEG-IFN alpha 2a.

As we have observed in our own treatment population, AA patients with HCV genotype 1 are capable only infrequently of mounting a successful antiviral response. We have shown that not all patients are equally likely to mount an antiviral response, particularly AA females with comorbidity. There is some suggestion that optimizing the treatment of
comorbid conditions such as DM and HTN may improve the response to HCV, and at least one ongoing clinical study is trying to answer that question.

Trust in the Medical System: Several publications have pointed to the importance of a patient's trust in their medical system for having a favorable clinical response. At our own institution, we are completing several large clinical studies correlating trust in the medical system to the response of antihypertensive therapy, and preliminary results have been positive. It is possible that the trust factor could effect cytokine production, compliance, and other areas known to influence antiviral responses.

Adherence to the Therapeutic Regimen - Overall, Peg-IFN + RBV combination therapy was well-tolerated at our institution. Individuals can however experience significant problems with medication treatment, including flu-like symptoms with injection, bone marrow suppression, and neuropsychiatric problems. These adverse events could lessen the compliance rate, which has been related to viral response rates.

Poor therapeutic response is an expected outcome from reduction in doses and early discontinuation of treatment. We have incorporated aggressive management of treatment-related adverse effects into our therapeutic program, which others have shown to be of clinical use in maintaining compliance.

Indicators of good compliance included refill frequencies for medications; patient questioning on compliance at office visits; visit regularity; follow-up at regular intervals by the nursing staff; and the development of anemia from interferon and ribavirin. Patient education itself can influence compliance. Conjeevaram recorded that the proportion of the total maximum dose taken of both PEG-IFN and RBV during the first 24 weeks was significantly less among AA than for non-AA. 54% of AA compared with 73% of non-AA took at least 80% of the maximum doses of both drugs, the criteria typically used in assessing compliance in the criteria typically used in assessing compliance in combination therapy of HCV. It is possible that differential adherence to therapy could account for observed differences in response rates.

All patients at CGMH were required pre-treatment to attend a 2 hr long class taught by nurses experienced in the care of patients with HCV, and were also encouraged to attend as many refresher classes as they could. Patients were given access to nursing support by phone 24/7, and appeared to be very accepting of these teaching opportunities. Many of our patients have commented that these classes made them feel more a part of their therapy and understanding of the treatment and accepting of problems that could arise, consistent with findings of others.

CONCLUSIONS

Experience at our institution revealed an overall antiviral response to HCV that is comparable to studies previously reported. In particular, our data reveal that African Americans experience less favorable responses to treatment, that race-sex stratification is important, and that the presence of hypertension and diabetes are associated with drastically reduced response rates in African Americans, particularly in females. Further studies are planed to elucidate the poor treatment response in this subgroup of AA patients and to determine if optimizing treatment of comorbidities can increase their response rate to HCV.

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