## 1689a. Association Between Patient Portal Access and Viral Suppression Among People Living with HIV in a Large Southeastern Clinical Cohort

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**Session:** 188. HIV: Modern ART *Friday, October 6, 2017: 2:00 PM* 

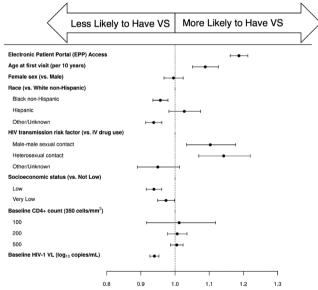
**Background.** Viral suppression (VS) among people living with HIV (PLWH), the goal of the HIV care continuum, leads to improved patient outcomes and decreased HIV transmission. Patient portals are online tools that enable patient interaction with healthcare systems and may increase patient engagement and improve health outcomes. We examined whether portal access was associated with VS among PLWH.

*Methods.* We conducted an observational cohort study among PLWH aged ≥18 years who had ≥1 HIV healthcare provider visit at the Vanderbilt Comprehensive Care Clinic (Nashville, Tennessee) from January 1, 2011–December 31, 2015. Patient portal access was defined as being registered for a portal account at any point in the year prior. VS was defined as having ≥1 viral load (VL) measured and the last VL ≤200 copies/ml within a given year. The adjusted relative risk (aRR) of VS was estimated with modified Poisson regression and robust standard errors for multiple outcomes per individual. Models were adjusted for all covariates in the Figure and for year since first kept appointment. Missing data were multiply imputed.

Results. The study population included 4,237 PLWH; median age was 43 years (IQR 33–50), 78% were male, 41% were black, and 60% reported malemale sexual contact (MSM). Of the 57% who had portal access during the study period, median age was 42 years (IQR 31–49), 86% were male, 30% were black, and 75% were MSM. In adjusted analysis, portal access was independently associated with improved VS (aRR = 1.19, 95% CI 1.16–1.21 vs. no portal access) (Figure). Increasing age and sexual contact (vs. injection drug use) remained associated with improved VS; black race (vs. white race), lower socioeconomic status, and higher baseline VL remained associated with poor VS after accounting for portal access (Figure).

Conclusion. Portal access was independently associated with improved VS, although sociodemographic disparities in VS persisted. Additionally, there were sociodemographic disparities in patient portal access. There may be important unmeasured confounders such as health literacy and educational attainment. Additional prospective studies are needed to determine whether patient portal access leads to improved VS among PLWH.

Figure. Adjusted Relative Risk of Viral Suppression



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## 1690. Performance of NS3, NS5a AND NS5b Hepatitis C Virus (HCV) Antiviral Resistance Sequencing Assays

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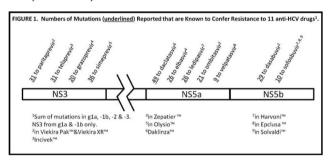
**Session:** 189. Hepatitis B and C Across the Lifespan *Friday, October 6, 2017: 8:30 AM* 

**Background.** HCV genotype 1a (HCV g1a) followed by HCV g1b, -g2, and -g3 are the most common etiologic agents in the  $\sim$ 3 million current HCV-infections in the US. To achieve effective therapy, antiviral drug resistance testing is often essential but not fully available. Knowledge of both the genotype and the presence of HCV mutations in the genes of the major drug targets (NS3, NS5A and NS5B) can assist in optimal treatment selection.

Methods. The HCV genotype of 1000 HCV positive clinical plasmas and sera was determined (HCVg Direct, GenMark). Ten independent Sanger sequencing assays detecting antiviral drug resistance mutations in the genes encoding NS3, NS5a, and NS5b were developed. Six of these assays address mutations in all three genes in the two most common genotypes (HCV g1a and g1b). In addition, four more assays address mutations in NS5a and NS5b of HCV g2 and g3. These mutations are resistance determinants against 11 anti-HCV drugs as shown in Figure 1. A stream-lined workflow employs conventional reverse transcriptase PCR, gel electrophoresis, spectrophotometry, bi-directional Sanger sequencing and reporting. The assays were designed to cover hot spot regions and capturing all known resistance mutations in NS3, NS5a and NS5b.

**Results.** Consistent with previous US HCV incidence reports, g1a, g1b, g2, and g3 comprised 99% of 1000 sequentially tested HCV patient specimens (62%, 12%, 11%, and 14%, respectively). Testing of more than 20 clinical samples each for g1a, g1b, g2, and g3 resulted in successful detection of NS3, NS5a, and NS5b mutations that confer drug resistance. The design successfully permitted detection of relevant mutations known to date for all 11 drugs. The number of reportable mutations range from 20 – 36, 9 – 49, and 10 – 29 for the NS3, NS5a, and NS5b inhibitors, respectively (Figure 1).

Conclusion. These assays provide the most comprehensive commercially available antiviral drug resistance information to date for mutations in HCV NS3, NS5A, and NS5B. This testing will assist physicians in deciding on the most appropriate treatment options for their patients.



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## 1691. Is there Failure to Screen for Hepatitis C in Newborns Suffering from Neonatal Abstinence Syndrome?

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