jurisdictions considering replicating the MCC program. Of the remainder, some costs related to protocol and assessment tool development could potentially be minimized by adapting materials already developed for LAC MCC. The annual cost per patient was $1100–$3100; although the annual costs of similar programs vary widely, it is within the range reported for these similar programs and is also comparable with the cost of a 1-month supply of a single-tablet coformulated antiretroviral therapy medication.9

Bruce R. Schackman, PhD
Philip J. Jeng, MS
Moses J. E. Flash, BA
Marisol Mejia
Sona Oksuzyan, MD, PhD, MPH
Justine A. Scott, MPH
Kenneth A. Freedberg, MD, MSc
Sonal P. Kulkarni, MD, MPH
Wendy H. Garland, MPH

Department of Healthcare Policy and Research, Weill Cornell Medical College, New York, NY
Medical Practice Evaluation Center, Massachusetts General Hospital, Boston, MA
Division of HIV and STD Programs, Los Angeles County Department of Public Health, Los Angeles, CA
Divisions of General Internal Medicine and Infectious Diseases, Massachusetts General Hospital, Boston, MA
Harvard University Center for AIDS Research, Harvard University, Boston, MA
Department of Health Policy and Management, Harvard T.H. Chan School of Public Health, Boston, MA

REFERENCES
1. Garland WH, Oksuzyan S, Mejia M, et al. Medical Care Coordination Services for Persons Living with HIV in Los Angeles County: A Robust Strategy to Strengthen the HIV Care Continuum. Los Angeles, CA: Los Angeles County Department of Public Health; 2017.
2. Garland WH, Kulkarni SP. Advancing “TasP” Using a Medical Care Coordination Program in Los Angeles County [Abstract]. Conference on Retroviruses and Opportunistic Infections, February 13–16, 2017, Seattle, WA.
3. Soto TA, Bell J, Pillen MB; For The HIV/AIDS Treatment Adherence, Health Outcomes Cost Study Group. Literature on integrated HIV care: a review. AIDS Care. 2004;16(suppl 1):43–55.
4. Division of HIV and STD Programs. County of Los Angeles Department of Public Health—Medical Care Coordination; 2015. Available at: http://publichealth.lacounty.gov/dhsp/MCC.htm. Accessed May 3, 2018.
5. Frick KD. Microcosting quantity data collection methods. Med Care. 2009;47(7 suppl 1):S76–S81.
6. Kim JJ, Maulsby C, Zulliger R, et al. Cost and threshold analysis of positive charge, a multisite linkage to HIV care program in the United States. AIDS Behav. 2015;19(10):1735–1741.
7. Jain KM, Maulsby C, Brantley M, et al. Cost and threshold analyses for 12 innovative US HIV linkage and retention in care programs. AIDS Care. 2016;28(9):1199–1204.
8. Maulsby C, Jain KM, Weir BW, et al. The cost and threshold analysis of retention in care (Ric): a multi-site national HIV care program. AIDS Behav. 2017;21(3):643–649.
9. Martin EG, Schackman BR. Treating and preventing HIV with generic drugs—barriers in the United States. New Engl J Med. 2018;378(4):316–319.

The Effect of Switching to Maraviroc + Darunavir/Ritonavir Dual Therapy in Virologically Suppressed Patients on the Progression of Liver Fibrosis: Findings From a Randomized Study

In vitro and animal studies revealed a potential protective role of CCR5 antagonists on reducing liver fibrosis progression and protecting from developing hepatocellular carcinoma.1 Hepatocytes bear CXCR4 and CCR5, the 2 main coreceptors for HIV entry into cells and the blockade of coreceptors on hepatic stellate cells, the major producers of extracellular matrix in the liver, will slow progression of liver fibrosis, especially due to HIV-envelope gp120–mediated fibrogenesis modulation.2–5

The aim of present analysis was to compare the evolution of liver fibrosis over time evaluated by surrogate biomarker assays in HIV–infected patients on a virologically successful

Supported by grants from Ministero della Salute, ISS, for Programma Nazionale AIDS project number 40H94; Janssen Europe provided Darunavir (DRV) tablets for patients in the study arm and supported the pharmacovigilance of the study, and ViV Healthcare Italy supported tropism testing for all patients for conducting the study. ViV Healthcare Italy also supported plasma antiretroviral drug monitoring for patients in the study arm for conducting the study. No additional external funding was received for this study. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Presented as poster at the 9th Italian conference on AIDS and Antiviral Research; June 12–14, 2017, Siena, Italy (P67).
A.B. reports nonfinancial support from Bristol-Myers Squibb; personal fees from Gilead Sciences; and nonfinancial support from ViV Healthcare. A.D.L. reports consulting fees from Gilead Sciences, Abbvie, Janssen, Bristol-Myers Squibb, ViV Healthcare Italy, and Merck Sharp and Dohme, outside the submitted work. B.R. reports nonfinancial support from Janssen, ViV Healthcare Italy, Abbvie, and Gilead and consulting fees from Merck Sharp and Dohme, outside the submitted work. A.D.M. reports grants and consulting fees from Bristol-Myers Squibb, Merck Sharp and Dohme, and Gilead and consulting fees from ViV Healthcare Italy, outside the submitted work. C.M. reports consulting fees and nonfinancial support from ABBVIE; consulting fees from Merck Sharp and Dohme, Gilead Sciences, ViV Healthcare Italy, and BMS; and nonfinancial support from ASTELLAS, outside the submitted work. F.V. reports nonfinancial support from Bristol-Myers Squibb, ViV Healthcare Italy, and Gilead Sciences and consulting fees from Merck Sharp and Dohme and BMS, outside the submitted work. M.C. reports consulting fees from Gilead Sciences, Janssen-Cilag, Merck Sharp and Dohme, Bristol-Myers Squibb, and ViV Healthcare Italy, outside the submitted work. L.M. reports grants and consulting fees from ViV Healthcare Italy, outside the submitted work. S.R. reports grants and consulting fees from ViV Healthcare Italy, Bristol-Myers Squibb, Merck Sharp and Dohme, Gilead Sciences, and Janssen, outside the submitted work. S.D.G. reports personal fees from Bristol-Myers Squibb, Janssen-Cilag, ViV Healthcare Italy, Gilead, and Merck Sharp and Dohme, outside the submitted work. The remaining authors have no conflicts of interest to disclose.

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Copyright © 2019 The Author(s). Published by Wolters Kluwer Health, Inc. www.jaids.com | e17
patients with HIV-1 RNA <50 copies/mL, randomized to switch to maraviroc + darunavir/r (MVC + DRV/r arm) qd or to continue the current MVC-free 3-drug antiretroviral therapy (ART) (3-drug ART arm).

Patients included in the study were enrolled in the GUided Simplification with Tropism Assay (GUSTA) trial, a multicenter, open-label, randomized study (www.clinicaltrials.gov, number NCT01367210), whose main results have been published.6

Briefly, GUSTA included patients with HIV-1 RNA <50 copies/mL for at least 6 months, R5 tropism and CD4 counts >200 cells/μL for at least 3 months before enrollment; hepatitis B virus–coinfected patients and those with Child-Pugh B/C cirrhosis were excluded.

We retrospectively evaluated Fibrosis-4 (FIB-4) Index and aspartate aminotransferase to Platelet Ratio Index (APRI) scores, at baseline and after 12, 24, 48, and 96 weeks. The cutoff points of serum marker tests of hepatic fibrosis were as follows: FIB-4 <1.45 (F0-F1), 1.45–3.25 (indeterminate), and >3.25 (F3-F4); APRI <0.5 (F0-F1), >1.5 (F2) and >2 (cirrhosis).

Differences between arms were assessed by McNemar test. The FIB-4 Index and APRI scores were used as continuous variables; their predictors at baseline and their change over time were investigated by linear regression.

We included 150 patients, 76 randomized to MVC + DRV/r arm and 74 to 3-drug ART arm. Baseline characteristics were homogeneous between arms except for relative younger age in the MVC + DRV/r arm (median 47 yrs; interquartile range [IQR] 40–52) than in the 3-drug ART arm (50 yrs; IQR 44–57) (P = 0.08), more frequent African ethnicity in the 3-drug ART arm than in the MVC + DRV/r arm (8% vs. 1%) (P = 0.05), and FIB-4 median value.

FIGURE 1. A, APRI score during follow-up. B, FIB-4 during follow-up. No significant difference between arms at each time-point.
higher in the MVC + DRV/r arm (1.15; IQR 0.82–1.32) than in the 3-drug ART arm (0.91; IQR 0.68–1.20) (P = 0.01). APRI score was similar between arms: 0.23 (IQR 0.18–0.29) in the MVC + DRV/r arm and 0.25 (IQR 0.20–0.33) in the 3-drug ART arm (P = 0.12).

Overall, 89% (134/150) were males and Caucasians; 41% (61/150) were heterosexuals; 38% (57/150) homosexuals/bisexuals; 7% (10/150) reported history of injected drug use, 11 years of HIV (IQR 7–18), 10 years of ART (IQR 6–15), CD4 at nadir 222 cells/mm (IQR 132–319) and at baseline 654 cells/mm (IQR 506–905). Eighteen patients presented positive serology for hepatitis C virus (HCV) and 8 had a detectable HCV RNA, 4 in each arms.

Sixteen (11%) presented diabetes mellitus: 12% (9/76) in the MVC + DRV/r arm and 9% (7/74) in the 3-drug ART arm (P = 0.04). At screening, nucloside reverse transcriptase inhibitors (NRTIs) were used in 95% (143/150), nonnucloside reverse transcriptase inhibitors (NNRTIs) in 12% (18/150), integrate strand transfer inhibitors (INSTIs) in 18% (17/150), and protease inhibitors (PIs) in 69% (103/150) of which boosted PI in 63% (94/150) and DRV/r in 31% (47/150). No differences between arms were observed in terms of disyndrome (9/76) in the MVC + DRV/r arm and 9% (7/74) in the 3-drug ART arm (P = 0.04). At screening, nucloside reverse transcriptase inhibitors (NRTIs) were used in 95% (143/150), nonnucloside reverse transcriptase inhibitors (NNRTIs) in 12% (18/150), integrate strand transfer inhibitors (INSTIs) in 18% (17/150), and protease inhibitors (PIs) in 69% (103/150) of which boosted PI in 63% (94/150) and DRV/r in 31% (47/150). No differences between arms were observed in terms of disyndrome (9/76) in the MVC + DRV/r arm and 9% (7/74) in the 3-drug ART arm (P = 0.04). At screening, nucloside reverse transcriptase inhibitors (NRTIs) were used in 95% (143/150), nonnucloside reverse transcriptase inhibitors (NNRTIs) in 12% (18/150), integrate strand transfer inhibitors (INSTIs) in 18% (17/150), and protease inhibitors (PIs) in 69% (103/150) of which boosted PI in 63% (94/150) and DRV/r in 31% (47/150). No differences between groups.

During observation in the 3-drug ART arm (n = 74), NRTIs were used in 92%, NNRTIs in 16%, INSTIs in 15%, PIs in 69%, boosted PI in 51%, and DRV/r in 43%.

According to the cutoff points of hepatic fibrosis, FIB-4 in the MVC + DRV/r arm was <1.45 in 83% (63/76), between 1.45 and 3.25 in 16% (12/76), and >3.25 in 1% (1/76); in the 3-drug ART arm, it was <1.45 in 88% (65/74) and between 1.45 and 3.25 in 12% (9/74) (no one had FIB-4 >3.25).

Overall, APRI was <0.5 in 91% (137/150), and no one had >1.5 at baseline.

Based on the FIB-4 score, at 48 weeks progression to a higher level was observed in 6% (4/63) in the MVC + DRV/r arm and in 6% (4/65) in 3-drug ART arm; in 3% (4/12) among those in MVC + DRV/r arm and in 3% (3/9) in 3-drug ART arm, FIB-4 improved by at least 1 stage, whereas the other patients did not modify their FIB-4 stratum.

Based on the APRI score, at 48 weeks, significant modification of the stratum was not observed.

In addition, no significant differences between arms were observed in platelet counts and alanine transaminase changes at 48 weeks from baseline. We observed a more profound decrease of aspartate transaminase (AST) levels in the MVC + DRV/r arm (mean change –4.19 IU/L, SD 7.2) vs. 3-drug ART arm (mean change +0.58 IU/L, SD 9.9) (P = 0.007).

In a multivariable model adjusting for risk factor for HIV acquisition and duration of ART exposure, longer time from HIV diagnosis (per 1 year increase +0.031, 95% confidence interval [CI]: +0.007 to +0.055, P = 0.01), lower nadir CD4+ cells count (+100 cells increase, –0.060, 95% CI –0.107 to –0.014, P = 0.01), and HCV antibody positive status (+0.321, 95% CI +0.000 to +0.642, P = 0.05) were associated with higher baseline FIB-4 values. No factor independently associated with baseline APRI values was observed. During follow-up, the APRI score decreased more prominently in the MVC + DRV/r arm vs 3-drug ART arm at week 12 (median change –0.02; IQR –0.06 to +0.12 vs –0.066; IQR –0.05 to +0.02; P = 0.28), at week 48 (–0.04; IQR –0.09 to +0.02 vs +0.001; IQR –0.037 to +0.049; P = 0.01), and at week 96 (–0.03; IQR –0.06 to +0.01 vs +0.02; IQR –0.01 to +0.10; P = 0.053) (Fig. 1A).

In a multivariable model, predictors of APRI change at 48 weeks were baseline APRI (–0.391; 95% CI –0.515 to –0.266; P < 0.001) and MVC + DRV/r arm vs 3-drug ART arm (–0.040; 95% CI –0.006 to –0.074; P = 0.021).

FIB-4 also showed a trend toward a more prominent reduction in the MVC + DRV/r arm (–0.02; IQR –0.21 to +0.13) vs 3-drug ART arm (+0.02; IQR –0.23 to +0.20) (P = 0.35) at week 48 (Fig. 1B). Baseline FIB-4, but not study arm, predicted FIB-4 modifications during follow-up.

In conclusion, we observed that switch to MVC + DRV/r in HIV-1–infected, but virologically suppressed patients on 3-drug ART, was associated with a slight but significant improvement of the APRI score over time as compared with continuing 3-drug ART without MVC. This MVC-containing regimen did not significantly influence the longitudinal change of the FIB-4 score, possibly due to the presence of age as a component of the score, which was increasing over time in the study patients, although a trend toward an improvement was observed. Our observations are in agreement with experiments showing a reduction of hepatic stellate cells activation and fibrosis progression and an improved survival in a murine model of hepatocellular carcinoma1 and in vitro observations on the inhibitory effect of MVC on the accumulation of fibrillar collagens and extra-cellular matrix proteins by human hepatic stellate cells.7 Results from this study are also in line with a previous retrospective non-comparative analysis on 71 HIV/HCV-coinfected patients treated with MVC, showing a potential beneficial effect on liver fibrosis measured by the APRI score.8 In a previous prospective, non-controlled pilot study on 24 HIV/HCV-coinfected patients starting a MVC-based regimen, liver fibrosis was slightly but not significantly reduced, although observation was limited to 6 months.9 In addition, a recent study suggests that a validated marker of liver fibrosis was reduced in HIV-1–infected patients carrying the variant allele CCR5 delta-32, associated with reduced CCR5 expression, and in patients exposed to cenicriviroc, a CCR5/CCR2 blockade agent.10

Our study adds to previous evidence and has its strengths in the randomized comparison, the study arm treated with a homogeneous MVC-containing regimen and the prospective follow-up of the patients up to 96 weeks. Its main limitation is the lack of information on the liver histological pattern modification rather than indirect biomarkers, as it remains unclear whether their change truly reflects hepatic fibrosis change. The lack of information on patients’ alcohol consumption and the absence of transient liver elastography measurements also represent limitations to this analysis.
Further studies are warranted to confirm an antifibrotic effect of CCR5 antagonist therapy.

ACKNOWLEDGMENTS

The authors thank the patients who shared their data, the GUSTA study group, ViViV Healthcare, Verona, that supported viral tropism determination, and TDM. Janssen who supported pharmacovigilance and gave darunavir.

GUSTA study group: S Di Giambenedetto, N Ciccarelli, R Gagliardini, S Lamonica, I Fant, F Lombardi, D’Avino Alessandro, Fabbiani Massimiliano (Clinic of Infectious Diseases, Catholic University of Sacred Heart, Rome); P Navarra, L Lisi, GMP Ciotti, (Pharmacology Department, Catholic University of Sacred Heart, Rome); A De Luca, B Rossetti, C Bianco, M Masini, (Infectious Diseases Unit, Azienda Ospedaliera Universitaria S.Orsola Malpighi, Bologna); P Caramello, G Orofino, M Farenga, S Carosella (Infectious Diseases Unit A, Amedeo di Savoia Hospital, Torino), Valeria Ghisetti (Microbiology and Virology Laboratory, Amedeo di Savoia Hospital, Torino); E Petrelli, B Canovari (Infectious Diseases Unit, Pesaro Hospital, Pesaro); C Catalani, M Trezzi (Infectious Diseases Unit, Pistoia Hospital, Pistoia); C Mastroianni, M Lichtner, R Marocco (Infectious Disease Unit, SM Goretti Hospital, Department of Public Health and Infectious Diseases, Sapienza University, Latina); A Bartoloni, G Serrantino, S Tekle Kiros, I Campolmi (Clinic of Infectious Diseases, Azienda Ospedaliera Universitaria Careggi, Firenze); A D’Arminio Monforte, T Bini, G Ancona, S Solaro (Infectious and Tropical Diseases Institute, Department of Health Sciences, University of Milan San Paolo Hospital, Milan); A Antinori, R Aciavarpa, S Ottou, R Libertone, S Mosti, C Pinnetti, (Infectious Diseases Unit, IRCCS L. Spallanzani, Roma); CF Perno, Ada Bertoli (Department of Experimental Medicine and Surgery, University of Rome Tor Vergata, Roma).

We are grateful to Alessandro Cozzi-Lepri, Annamaria Geretti and Jonathan Schapiro for their invaluable work in the Data Safety and Monitoring Board.

Barbara Rossetti, MD, PhDa,b
Roberta Gagliardini, MDc
Gaetana Serrantino, MDd
Vincenzo Colangeli, MDc
Alessandra Latini, MDf
Manuela Colafligih
Francesca Vignale, MDg
Stefano Rusconih
Antonio Di Biagioi
Giancarlo Orofino, MDj
Ivano Mezzaromaa
Vincenzo Vulloj
Daniela Franciscim
Claudio Mastroianni
Michele Trezzi, MD
Benedetta Canovari, MD
Silvia Lamonica, PharmD
Arturo Cicculio, MD
Alberto Borghetti, MD
Antonella D’Arminio Monforte
Simona Di Giambenedetto
Andrea De Lucaa,c

for GUSTA trial study group

aInfectious Diseases Unit, Azienda Ospedaliera Universitaria Senese, Siena, Italy
bClinical of Infectious Diseases, Catholic University of Sacred Heart, Roma, Italy
cDepartment of Medical Biotechnologies, University of Siena, Siena, Italy
dClinical of Infectious Diseases, Azienda Ospedaliera Universitaria Careggi, Firenze, Italy
eClinical of Infectious Diseases, Azienda Ospedaliera Universitaria S.Orsola Malpighi, Bologna, Italy
fInfectious and Tropical Diseases Unit, University of Siena, Siena, Italy

Copyright © 2019 The Author(s). Published by Wolters Kluwer Health, Inc.
PD-1+ and TIGIT+ CD4 T Cells Are Associated With Coronary Artery Calcium Progression in HIV-Infected Treated Adults

To the Editors:

With the advent of antiretroviral therapy (ART), AIDS-related morbidity and mortality has declined allowing age-related diseases, such as cardiovascular disease (CVD), to emerge as new challenges for this population. With an effect size of approximately 1.5–2.0, the impact of HIV on CVD is independent of traditional cardiovascular risk factors and antiretroviral medications. Immune activation of monocytes and macrophages has been implicated in the higher CVD risk in individuals with chronic HIV. Coronary artery calcium (CAC) is an indicator of subclinical coronary artery atherosclerosis predictive of coronary events including the onset of myocardial infarction and coronary-related deaths. Two research groups, including our own, have published that activated CD16+ monocytes/macroinphages predict greater CAC progression among HIV-infected persons over a 2-year period.

HIV-associated immune dysfunction is also characterized by T-cell dysfunction; their role in HIV-associated atherosclerosis has been less studied. T-cell exhaustion is characterized by an expansion of negative checkpoint receptors (NCRs) including PD-1 (programmed cell death protein 1), TIM-3 on CD4+ and CD8+ T cells and other immune cells associated with chemokine receptor inactivation through mutation or therapeutic blockade. During chronic infection or cancer, T-cell exhaustion results in the progressive loss of effector function, upregulation of inhibitory receptors, and failure to transition to a quiescent state. We have recently reported that the expansion of NCR on CD4+ T cells is associated with comorbidities of cognitive impairment and fat loss in HIV-infected individuals on ART. In this study, we sought to examine the impact of NCR-expressing T cells on CVD. We hypothesized that higher baseline PD-1-expressing and/or TIGIT-expressing CD4+ T cells will be associated with progression of CAC after 2 years in chronically HIV-infected individuals on stable ART.

T-cell immunophenotyping was performed on banked peripheral blood mononuclear cells from HIV-infected individuals enrolled in the Hawaii Aging with HIV-Cardiovascular Disease (HAHC-CVD) Cohort Study, a longitudinal study of subclinical CVD risk in individuals with chronic HIV age >40 years and on ART for ≥3 months. Data on CVD risk factors, as well as metabolic data from fasting blood, were available and allowed calculation of the Framingham Risk Score (FRS) using the National Cholesterol Education Program website (http://hp2010.nhlbi.nih.gov/atpiii/calculator.asp). As previously reported, computer tomography (CT) examinations for CAC were performed locally in Honolulu, HI, using a dual-source CT scanner (Siemens 64-slice Somaton) with quantification of CAC centrally at the Los Angeles Biomedical Research Institute (M Budoff).

Cryopreserved peripheral blood mononuclear cell were thawed and stained for viability and the frequency of expression at baseline of TIGIT, PD-1, and TIM-3 on CD4+ and CD8+ T cells were assessed by flow cytometry following previously published methods. Isotype controls or fluorescence minus one controls were used to facilitate gating. Software-based compensation was performed on FlowJo (Treestar).

The predictive impact of NCR-expressing T cells and other immunologic parameters on 2-year change in CAC was assessed by logistic regression, dichotomizing the participants into those who demonstrated progression of CAC and those whose level showed no progression. A multivariate logistic regression model was constructed. Because of the small sample size, FRS was used as a composite marker of traditional CVD-risk factors. We, as well as others, have reported a correlation between monocyte immune activation and change in CAC. We therefore further examined the relationship between NCR-expressing T cells and monocyte subsets based on CD16 expression (classical, intermediate, and nonclassical monocytes), hypothesizing that any impact of NCR-expressing T cells on CAC may be mediated through an increase in monocyte immune activation. A two-sided P-value (P) < 0.05 was considered as statistically significant. Analyses were performed using SPSS version 24 (IBM, Armonk, NY).

The data consisted of 43 HIV-infected participants who were predominantly male (88%) and Caucasian

REFERENCES

1. Ochoa-Callegjerro L, Pérez-Martínez L, Rubio-Medivallia S, et al. Maraviroc, a CCR5 antagonist, prevents development of hepatocellular carcinoma in a mouse model. PLoS One. 2013;8:e53992.

2. Friedman SL. Preface. Clin Liver Dis. 2008; 12:xii–xvi.

3. Seki E, De Minicis S, Gwak GY, et al. CCR1 and CCR5 promote hepatic fibrosis in mice. J Clin Invest. 2009;119:1858–1870.

4. Berres ML, Koenen RR, Rueland A, et al. Antagonism of the chemokine CCL5 ameliorates experimental liver fibrosis in mice. J Clin Invest. 2010;120:4129–4140.

5. Bruno R, Galastri S, Sacchi P, et al. Gp120 modulates the biology of human hepatic stellate cells: a link between HIV infection and liver fibrogenesis. Gut. 2010;59:513–520.

6. Rossetti B, Gagliardini R, Meini G, et al. Switch and liver

7. Coppola N, Perna A, Lucariello A, et al. Effects of treatment with Maraviroc a CCR5 inhibitor on a human hepatic stellate cell line. J Cell Physiol. 2010;223:6224–6231.

8. Gonzales E, Boix V, Deltoro MG, et al. The effects of Maraviroc on liver fibrosis in HIV/HCV co-infected patients. J Int AIDS Soc. 2014;17 suppl 3):19643.

9. Macos J, Villoria MM, Rivero A, et al. Lack of short-term increase in serum mediators of fibrogenesis and in non-invasive markers of liver fibrosis in HIV/hepatitis C virus coinfected patients starting maraviroc-based antiretroviral therapy. Eur J Microbiol Infect Dis. 2012;31:2083–2088.

10. Sherman KE, Abdel-Hameed E, Rooster SD, et al. Improvement in hepatic fibrosis biomarkers associated with chemokine receptor inactivation through mutation or therapeutic blockade. Clin Infect Dis. 2018. doi:10.1093/cid/ciy807. [epub ahead of print].

C.S. has received grants (R01HL095135 and U54MD007584) from the National Institutes of Health. For the remaining authors have no funding or conflicts of interest to disclose.