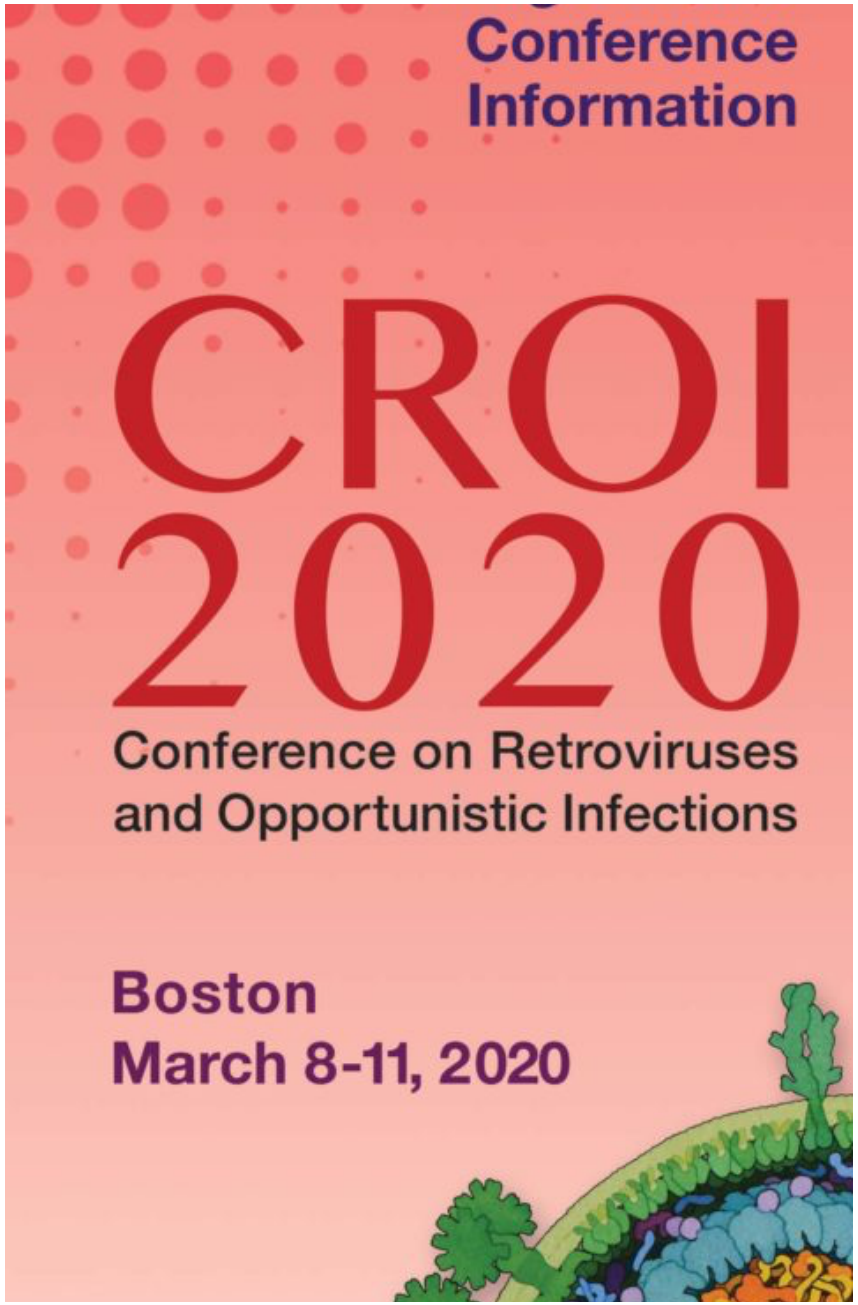


SELECCION COMUNICACIONES EN COINFECCION VIRUS DE HEPATITIS

Juan González-García

09 de Junio de 2020

Resumen comunicaciones

GONE BUT NOT FORGOTTEN: HCV AFTER DIRECT-ACTING ANTIVIRAL THERAPY

1. **INFERIORITY OF SHORT DURATION SOFOSBUVIR-VELPATASVIR FOR RECENT HCV (REACT STUDY). Gail Matthews *Kirby Institute, Sydney, Australia***
2. INDIVIDUAL AND POPULATION-LEVEL IMPACT OF HCV TREATMENT AMONG PEOPLE WHO INJECT DRUGS. Javier Cepeda *University of California San Diego, LA JOLLA, CA, USA*
3. LARGE HIV OUTBREAK AMONG PEOPLE WHO INJECT DRUGS, WEST VIRGINIA, 2018–2019. Robert Paul McClung *Centers for Disease Control and Prevention, Atlanta, GA, USA*
4. **HCV TRANSMISSION AMONG MSM: EXTERNAL INTRODUCTIONS COULD COMPLICATE MICRO-ELIMINATION Jelle Koopsen *Amsterdam University Medical Center***
5. NEWBORN TESTING REVEALS HIGH HCV SEROPREVALENCE IN PREGNANT WOMEN FROM NEW YORK STATE. Linda M Styer *New York State Department of Health, Albany, NY, USA*
6. HEPATOCELLULAR CARCINOMA RISK AMONG PERSONS WITH HIV IN NORTH AMERICA, 1996-2015. Jing Sun *Johns Hopkins University, Baltimore, MD, USA*
7. HIV/HCV VS HCV: PLASMA AND LIVER VIRAL DYNAMICS AND IP-10 LEVELS . Ashwin Balagopal *Johns Hopkins University, Baltimore, MD, USA*
8. **CLINICAL PREDICTORS OF LIVER FIBROSIS PRESENCE & PROGRESSION IN HIV-ASSOCIATED NAFLD. Lindsay T Fourman *Massachusetts General Hospital, Boston, MA, USA***

POSTERS 541-618

Resumen de otras presentaciones

Concurrent Preconference Workshops-

08 Marzo 2020 14:00-16:00 horas

Interactive Case-Based Workshop: What's Eating Your Liver
Conveners: Jürgen K. Rockstroh, *University of Bonn*, and Robert T. Schooley, *University of California San Diego*

When at Third You Don't Succeed
David L. Wyles, *Denver Health and Hospital Authority*

'A' Case To Remember: Hepatitis A - Managing an Old Virus in New Populations at Risk
Darcy Wooten, *University of California San Diego*

Hepatocellular Carcinoma
Susanna Naggie, *Duke University*

Nonalcoholic Steatohepatitis
Kathleen E. Corey, *Massachusetts General Hospital*

Concurrent Symposia.

09 Marzo 2020L 1600 a 1800

History Repeating Itself: Sexually Transmitted Infections in Women and Infants
Convener: Landon Myer, *University of Cape Town* , and Annette H. Sohn, *Therapeutics Research, Education and AIDS Training Asia*

Mother-to-Child Transmission of Hepatitis B: Can It Be Eliminated?
Yusuke Shimakawa, *Pasteur Institute*

Concurrent Symposia.

10 Marzo 2020 1600 a 1800

B Cured
Conveners: Robert T. Schooley, *University of California San Diego*, and Annette H. Sohn, *Therapeutics Research, Education and AIDS Training Asia*

Global Elimination of Hepatitis B Virus
Gilles Wandeler, *Bern University Hospital*

Adapting the Immune Response to Cure Hepatitis B
Barbara Rehermann, *National Institutes of Health*

Hepatitis B Virus: New Agents
Raymond T. Chung, *Massachusetts General Hospital*

The Mathematics of Hepatitis B Cure
Alan S. Perelson, *Los Alamos National Laboratory*

Concurrent Symposia.

11 Marzo 2020 1600 a 1800

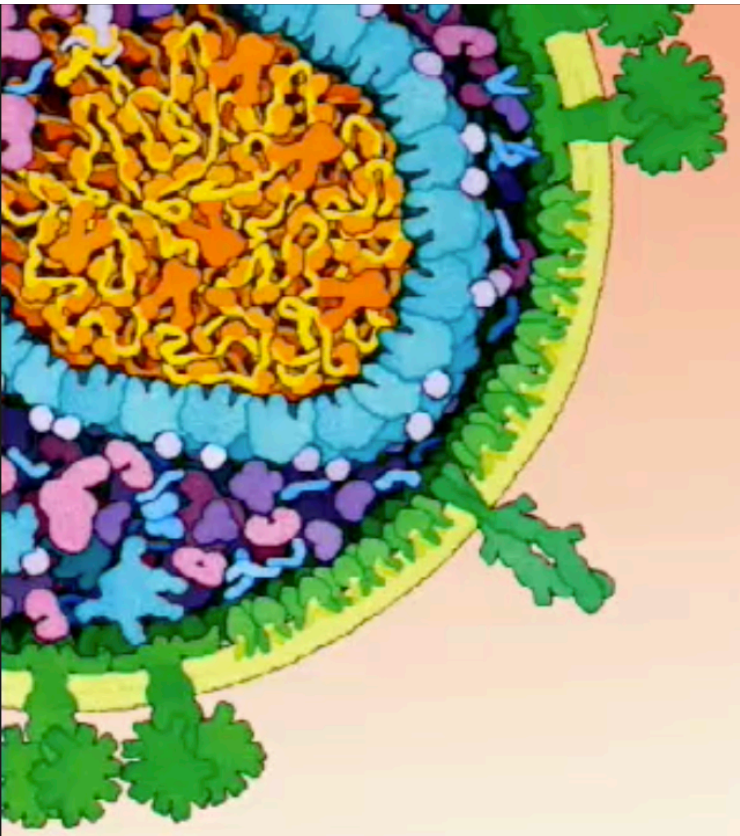
Hepatitis C: parallels, pitfalls, and promise

Global epidemiology of hepatitis C
Yvan J-F Hutin
World Health Organization, Cairo, Egypt

Modeling and examples of HCV elimination: possibilities, achievements, and next steps
Natasha Martin
University of California San Diego, San Diego, CA, USA

Vertical hepatitis C transmission: déjà vu all over again?
Ali Judd
University College London, London, United Kingdom

Hepatitis C treatment on a shoestring
Isabelle Andrieux-Meyer
Drugs for Neglected Diseases Initiative, Geneva, Switzerland



ORAL ABSTRACT: OL-10

Wednesday, March 11, 2020

INFERIORITY OF SHORT DURATION SOFOSBUVIR-VELPATASVIR FOR RECENT HCV (REACT STUDY)

Gail Matthews

Kirby Institute
Sydney, NSW, Australia

REACT was a NIH (NIDA)-funded randomized controlled trial comparing six (short) and 12 weeks (standard) sofosbuvir-velpatasvir for acute and recently acquired hepatitis C in people who inject drugs and people with HIV coinfection (clinicaltrials.gov: NCT 02625909).

Data Safety Monitoring Board

DSMB: international panel (hepatologist, ID physician and external statistician).

DSMB analysis was scheduled after the first 50 participants in each arm reached SVR12. DSMB met for first review in July 2018. A second review was then recommended to occur after 60 participants reached SVR12 in each arm.

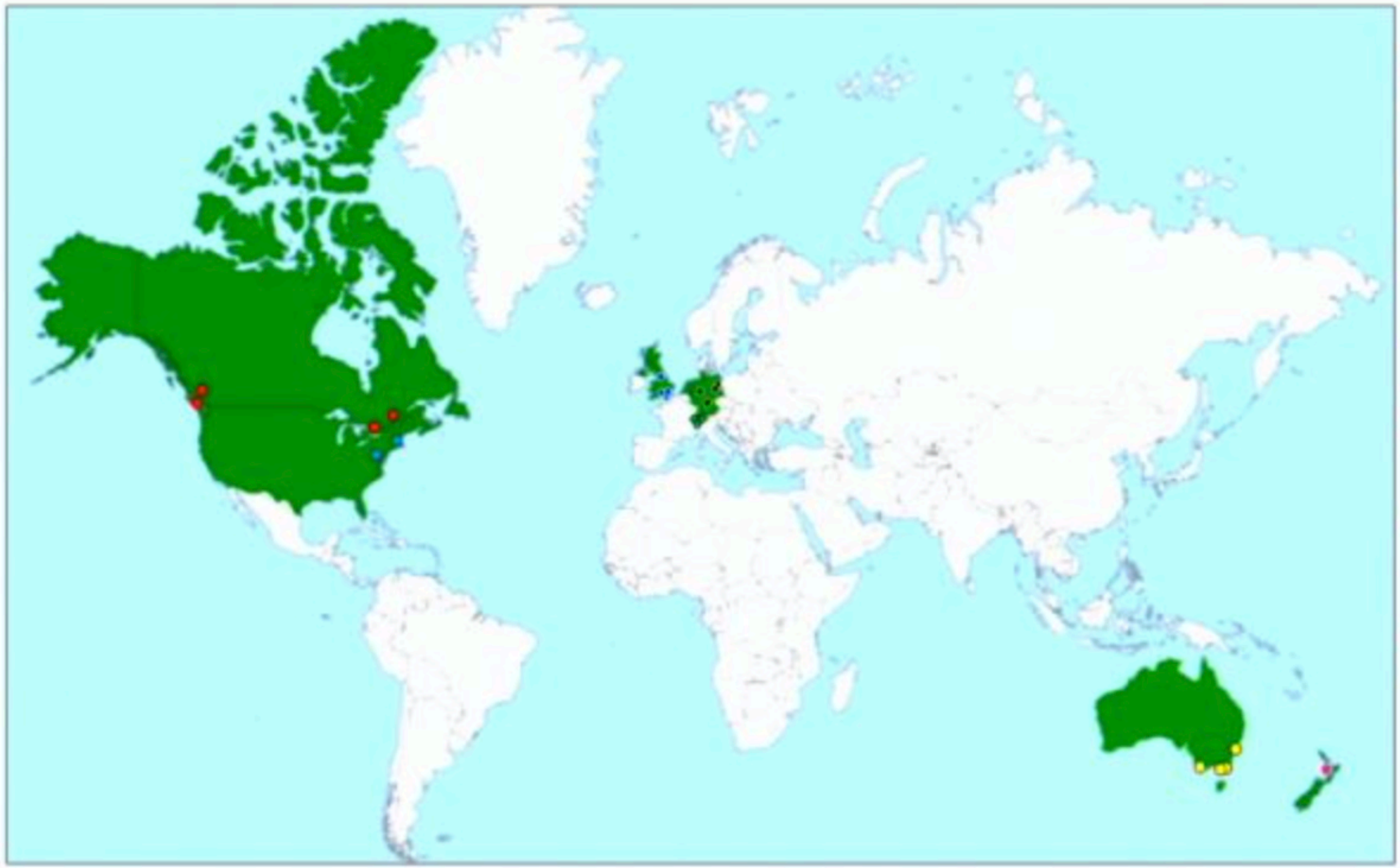
Recommendation of second DSMB review (May 2019) was cessation of study randomisation due to an unacceptably high rate of relapse in the short arm.

All enrolled non-randomised participants were switched to 12 weeks therapy with immediate effect.

Final study population for primary analysis consisted of all RANDOMISED participants

REACT: multicentre international phase 4 study

24 STUDY SITES COVERING 8 COUNTRIES



Australia

Canada

USA

UK

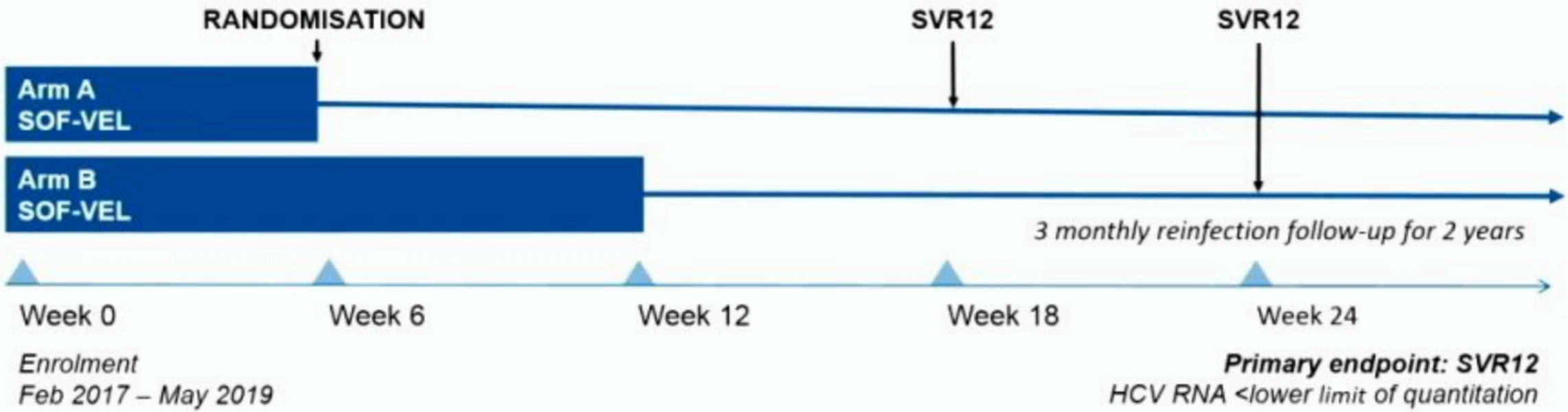
Switzerland

The Netherlands

New Zealand

Germany

Study design and methods



Randomisation was 1:1 and stratified by site and HIV status.

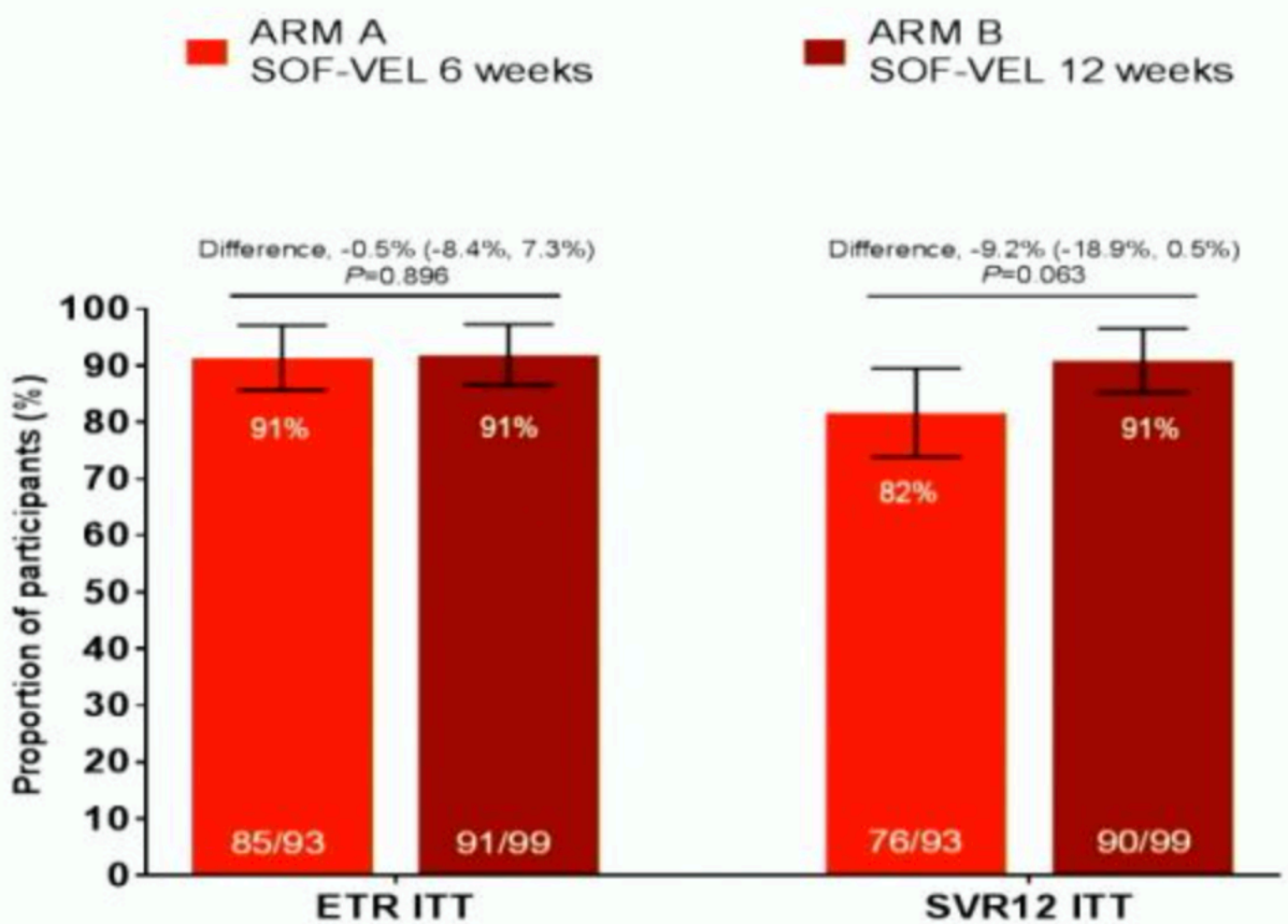
Analyses:

ITT population: all participants randomized (FAS)

Modified ITT: FAS excluding non-virological reasons for treatment failure (i.e., death, loss to follow up) and *confirmed* reinfection.

Per protocol: participants who received >90% of treatment with follow-up virologic data at SVR12.

Results: ITT analysis (n=192)



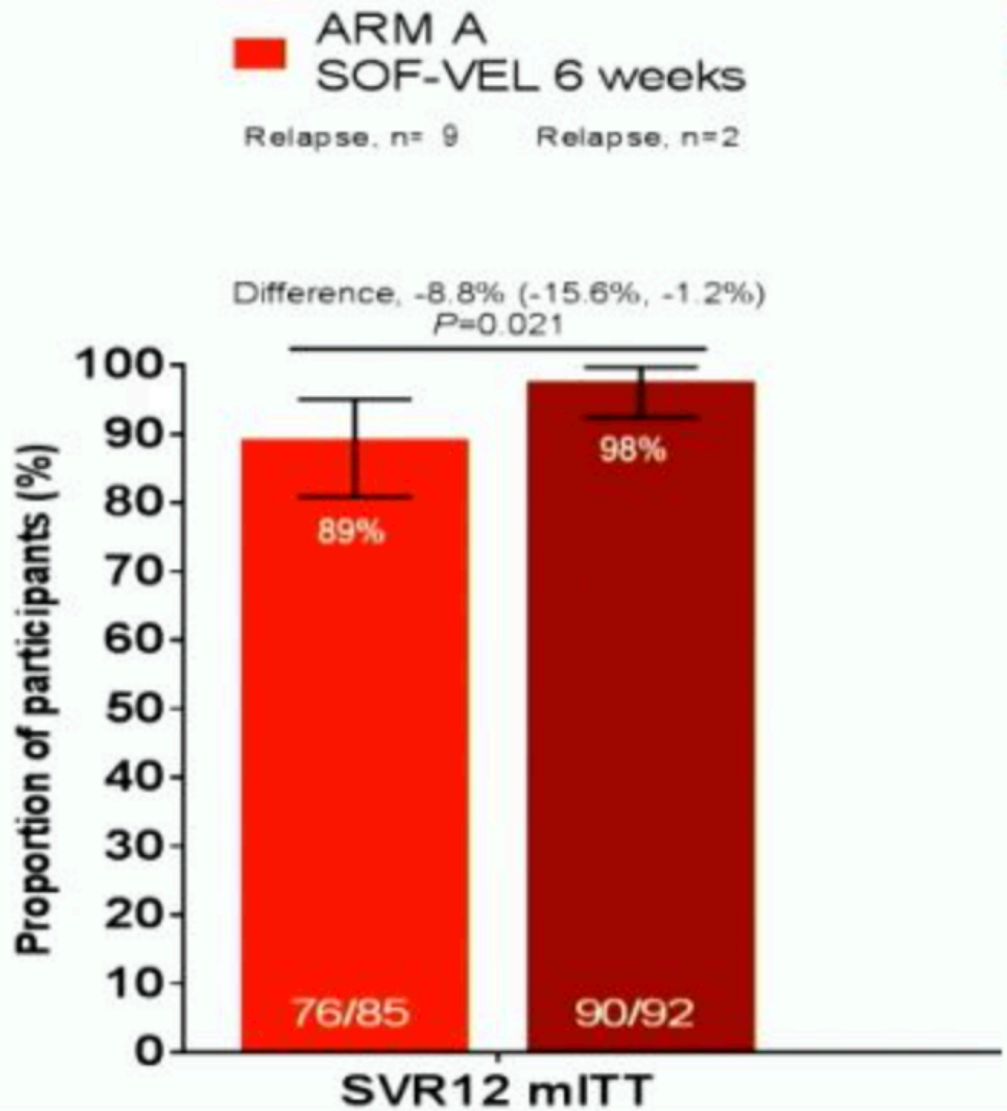
Failures	Arm A	Arm B
Total	17	9
Death	2	0
LTFU	3	2
Reinfection	3	2
Relapse	9	2

Reinfections before SVR12 (n=5)

Arm A	Arm B
3a → 1a	1a → 3a
4d → 1a	1a → 4d
1a → 1a*	

* Genetic distance core-E2 11%

Results: mITT analysis



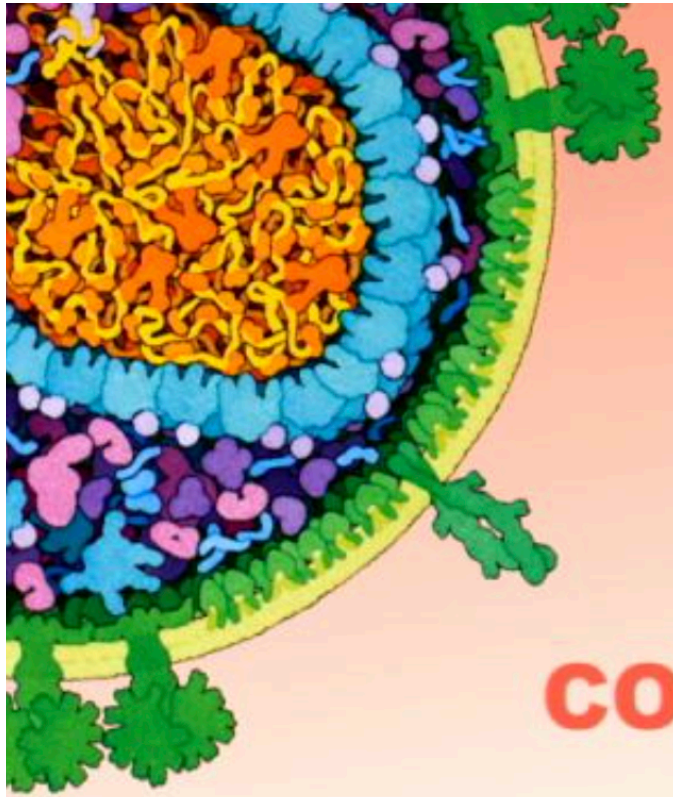
Relapse rate of 9% in short arm versus 2% in standard arm

Excludes LTFU (5), death (2) and confirmed reinfections (5)

Conclusiones del estudio REACT

- Aún siendo la tasa de respuesta alta en ambos brazos, la tasa de recaída en el brazo de 6 semanas con SOPF/VEL se consideró excesivamente alta comparado con el tratamiento estándar de 12 semanas

- El estudio apoya el tratamiento precoz de los casos de hepatitis aguda / reciente



ORAL ABSTRACT: OL-10

Wednesday, March 11, 2020

HCV TRANSMISSION AMONG MSM: EXTERNAL INTRODUCTIONS COULD COMPLICATE MICRO-ELIMINATION

Jelle Koopsen

Amsterdam University Medical Center
Amsterdam, Netherlands

CROI 2020

Métodos y Objetivos

- Revisión de la epidemia entre HSH en Amsterdan
- Clasificación filodinámica de 288 nuevas infecciones por VHC
- Descripción de los “clusters” de transmisión
- Tendencia temporal de la importación de casos

HCV epidemic among MSM in Amsterdam

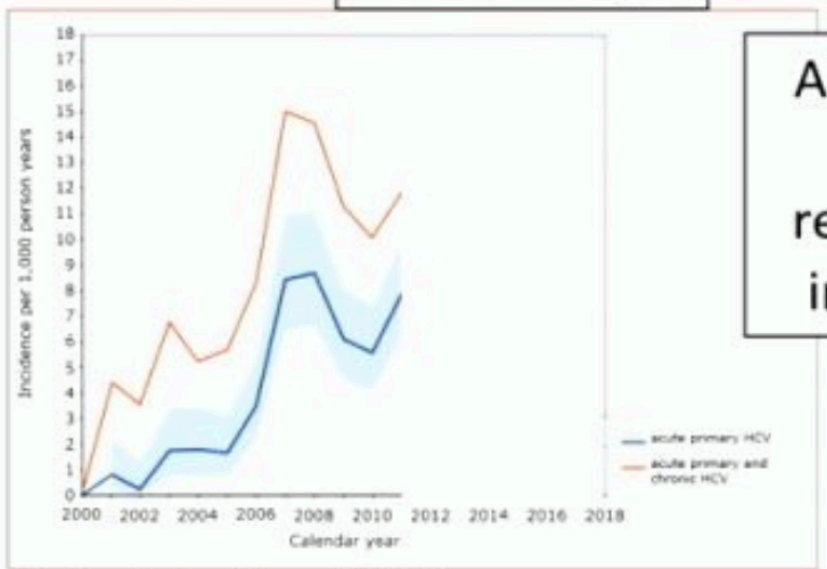
HCV incidence | Peak incidence | Increased testing of MSM at risk

2000 — 2007 — 2011 — 2015

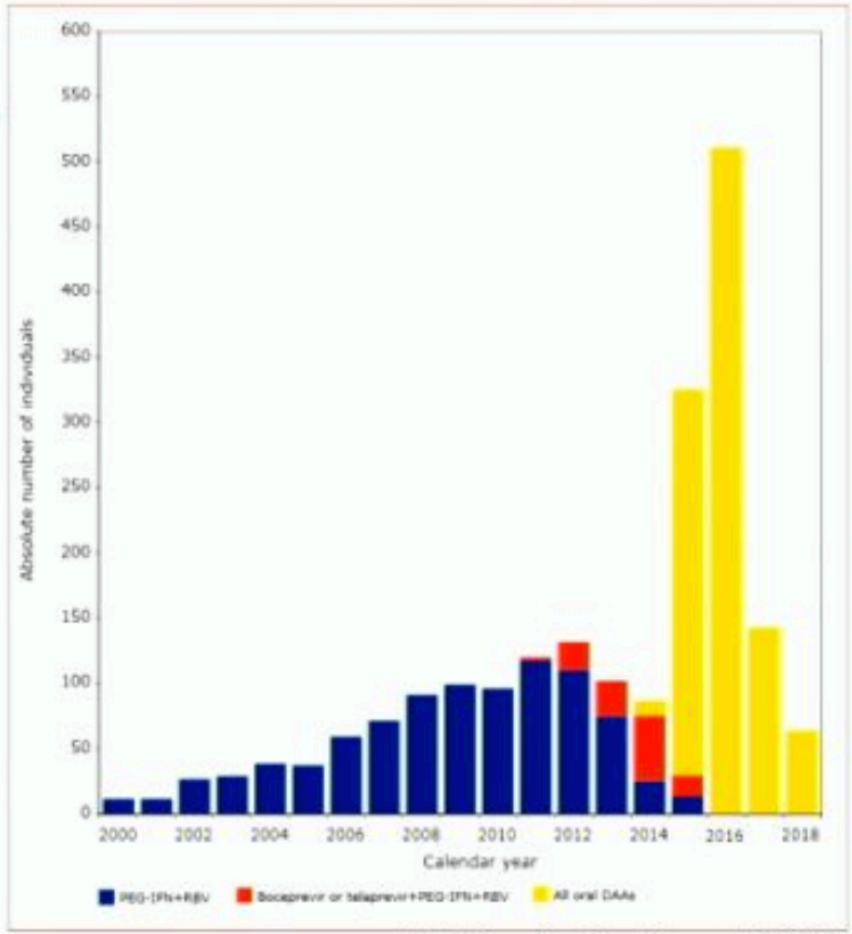
MSM-specific clustering

Universal DAA access

Alarmingly high reinfection incidence



HIV Monitoring Report 2019



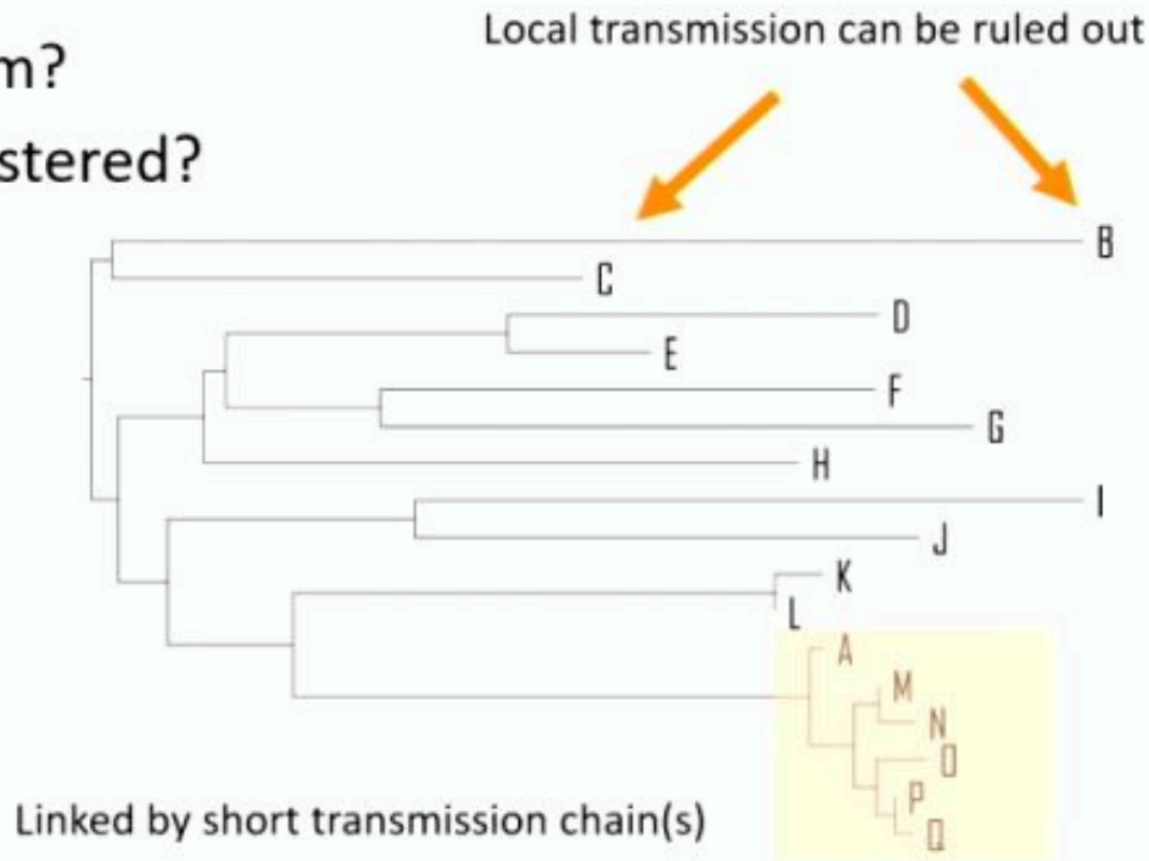
HIV Monitoring Report 2019

Phylodynamics to classify new infections

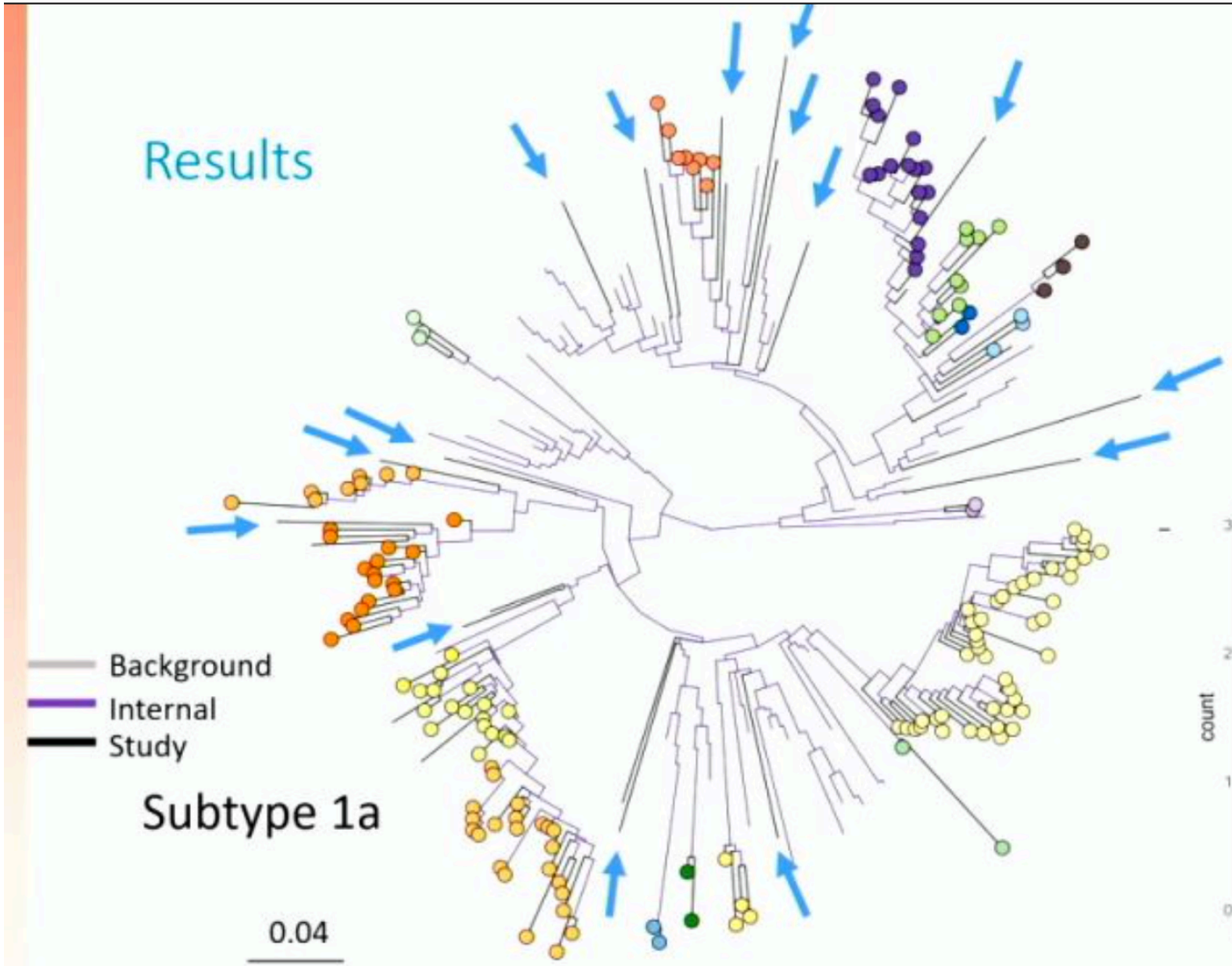
1. Where do the new infections come from?
    - Phylogenetically related or not clustered?

2. Transmission clusters in Amsterdam

3. External introductions over time

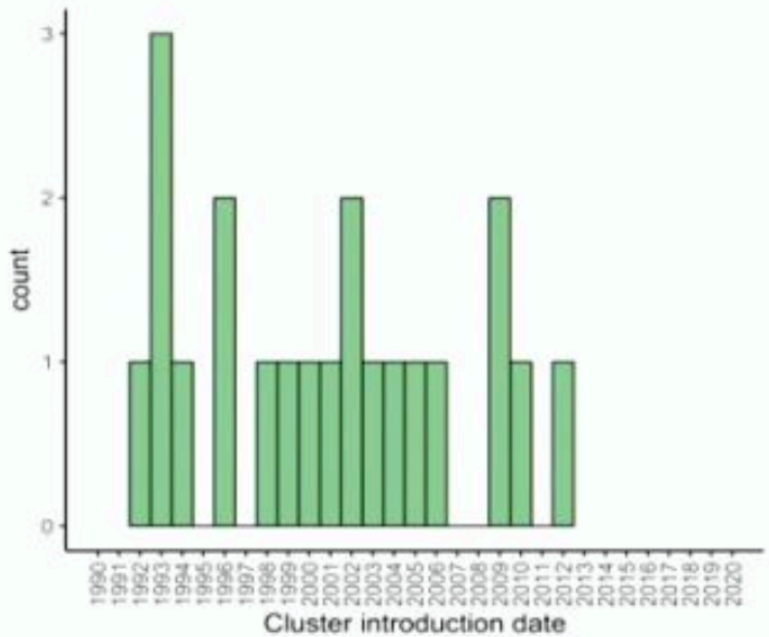


Results



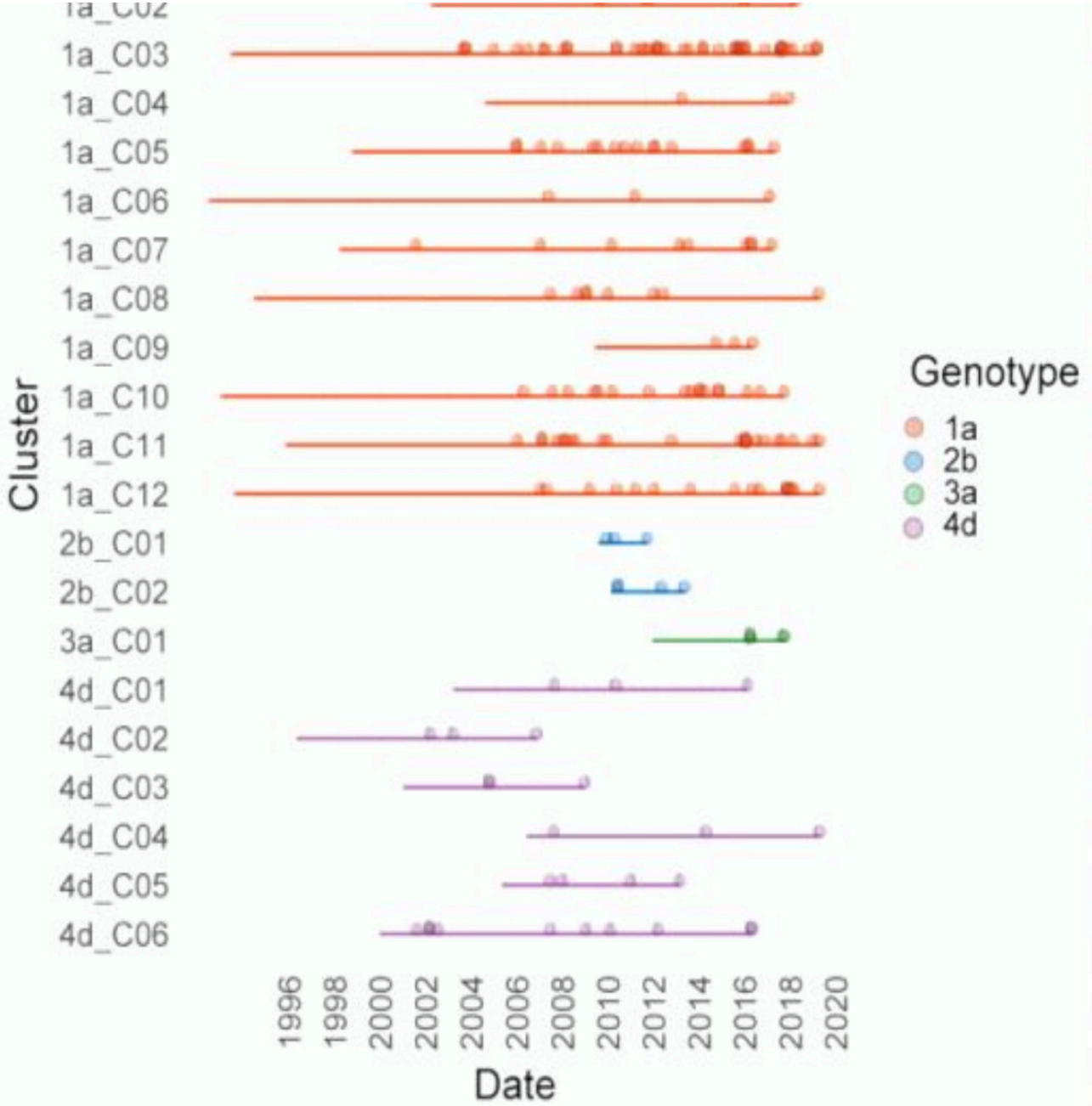
Subtype 1a

Clustered sequences	**86% (166/191)**
Clusters	12
Pairs	6
Cluster size	9
Cluster persistence	21.2 years

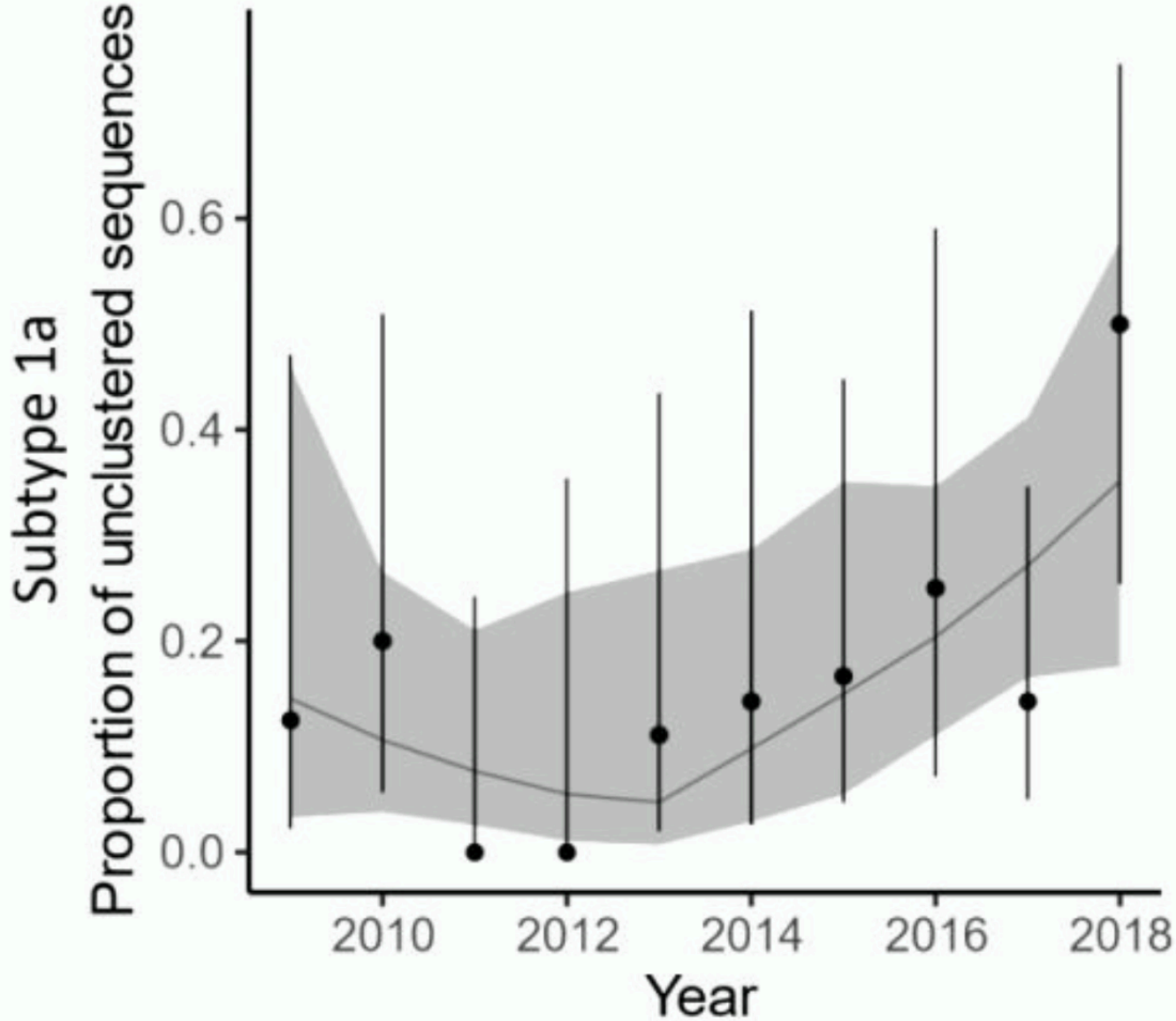


Transmission cluster characteristics

- Some clusters seem extinct
  (2b_C01, 2b_C02, 4d_C02, 4d_C03)
- Long sampling intervals
- Subtype 2b & 3a introduced later

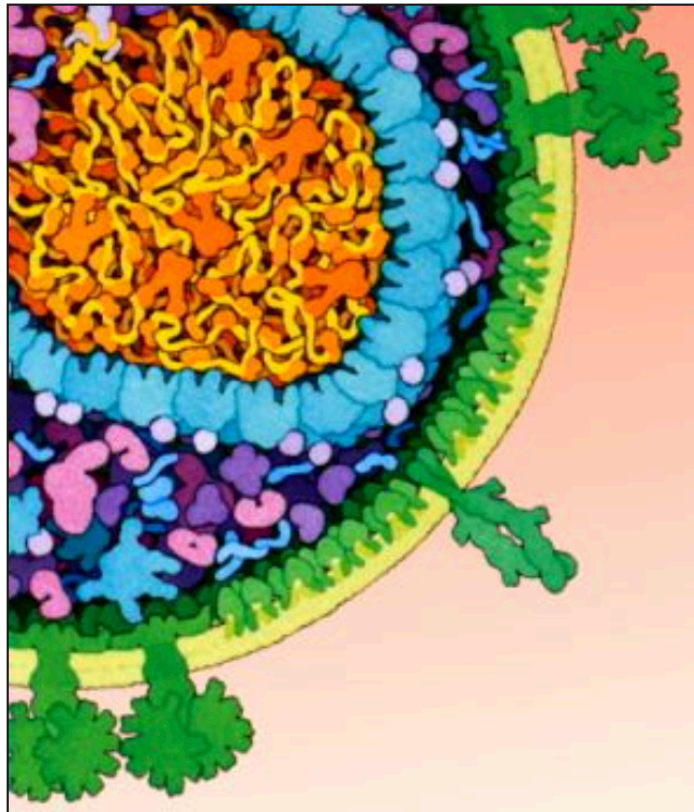


Proportion external introductions increases over time



Implications & discussion

- Local micro-elimination will be complicated if contribution of external introductions is high
- High DAA uptake → less local virus → more likely to acquire external virus
- 'Local transmission' may also be transmission within international clusters
- Lack of new clusters after 2012 could imply efforts to prevent new transmission chains are successful



ORAL ABSTRACT: OL-10

Wednesday, March 11, 2020

CLINICAL PREDICTORS OF LIVER FIBROSIS PRESENCE & PROGRESSION IN HIV-ASSOCIATED NAFLD

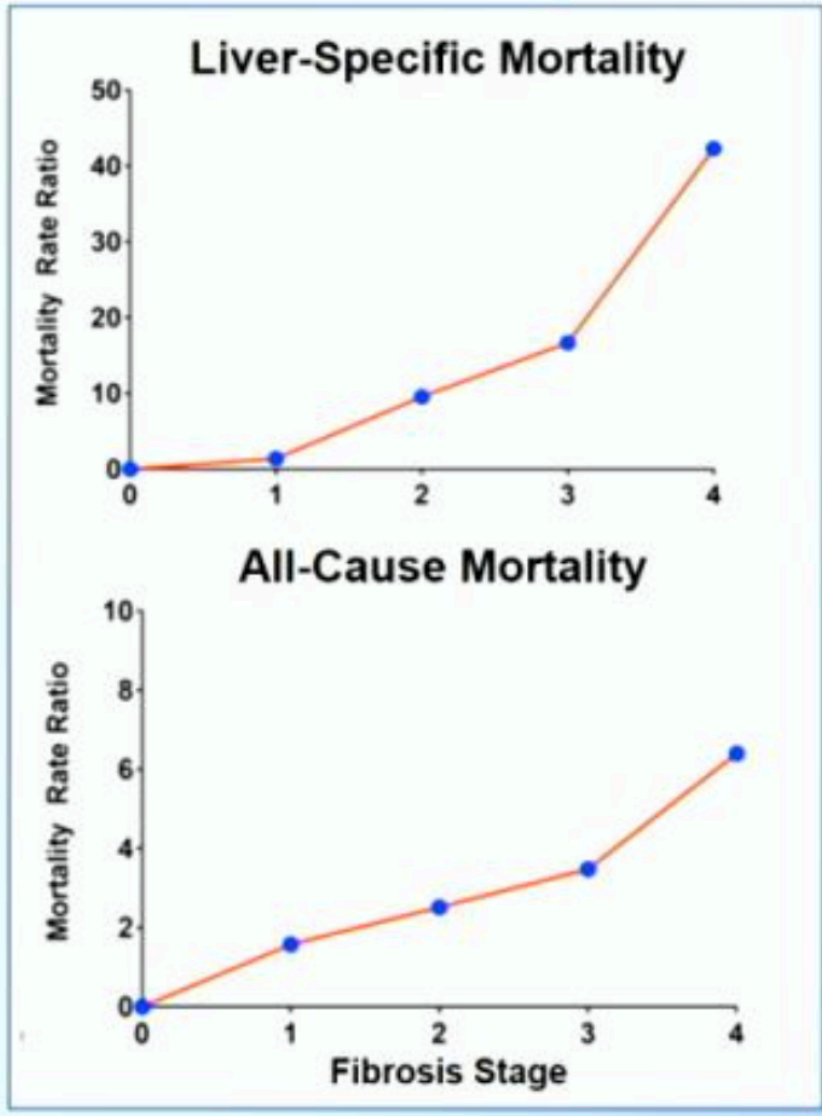
Lindsay T Fourman

Massachusetts General Hospital
Boston, MA, USA

CROI 2020

HIV-Associated Nonalcoholic Fatty Liver Disease (NAFLD)

- Fibrosis severity in NAFLD is the strongest predictor of liver-specific and all-cause mortality
- In the context of HCV, fibrosis is accelerated in those with HIV coinfection
- Natural history of HIV-associated NAFLD is not well understood
- Identifying clinical predictors of fibrosis presence and progression is imperative to target those with more aggressive disease



Dulai, *Hepatology* 2017

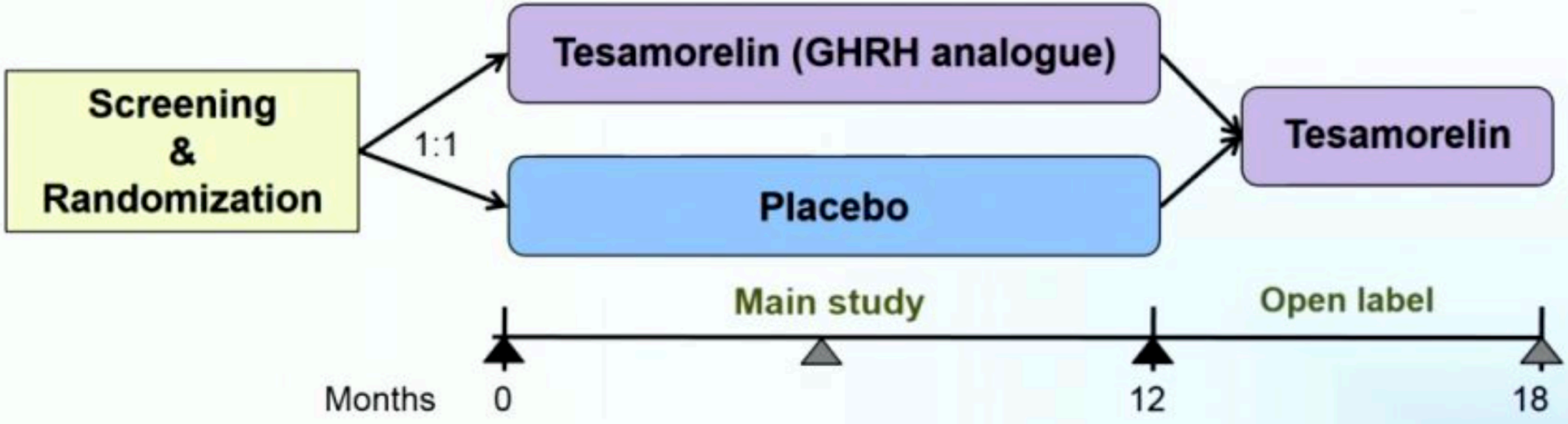
Tesamorelin Effects on Liver Fat and Histology in Individuals with HIV and NAFLD

Inclusion criteria

- Individuals with HIV on stable ART
- Hepatic fat ≥ 5% on ^{1}H-MRS

Exclusion criteria

- Active viral hepatitis
- Cirrhosis
- Heavy alcohol use
- HbA1c ≥ 7%



▲ Hepatic ^{1}H-MRS, abdominal MRI, liver biopsy

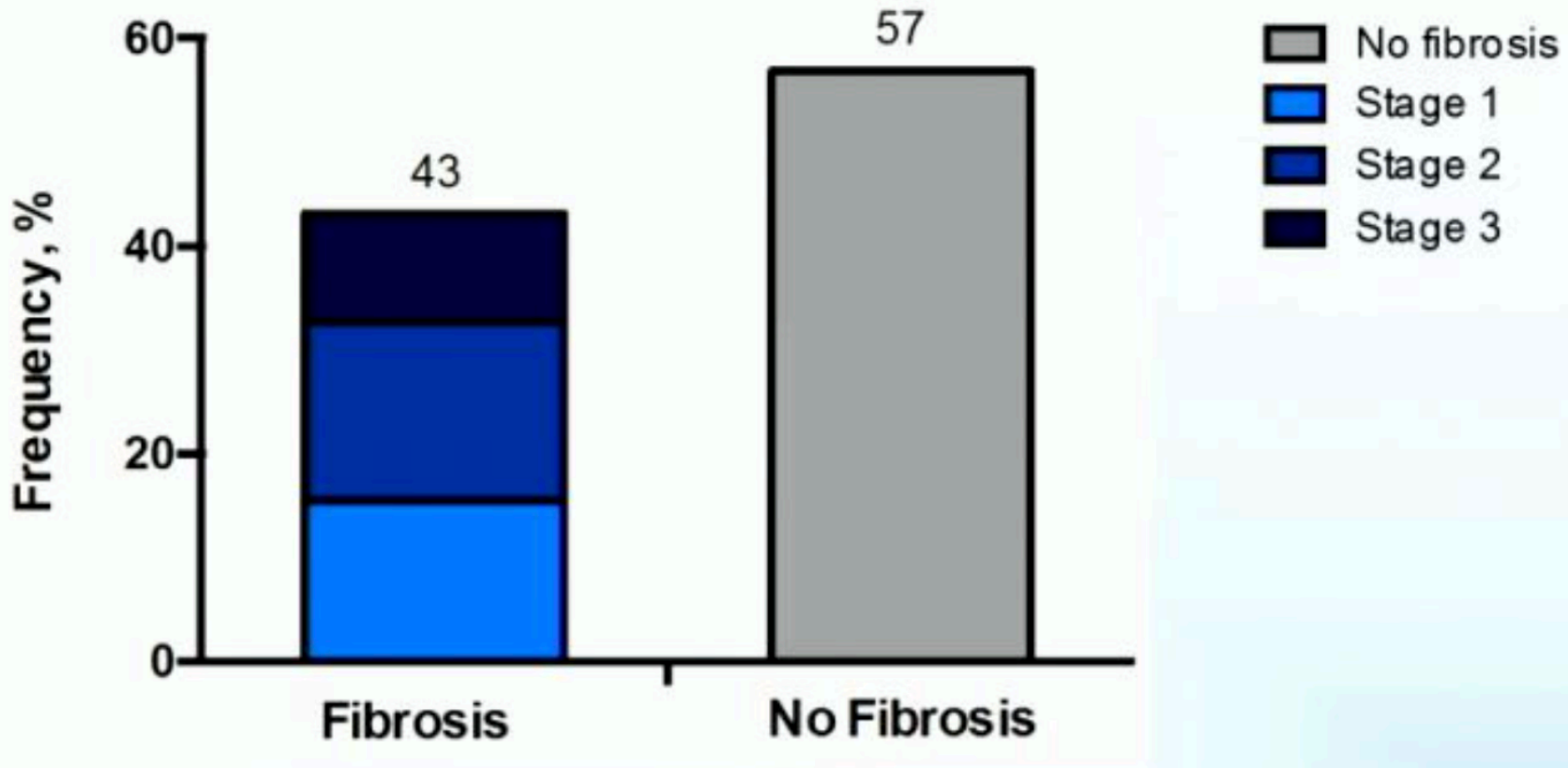
△ Hepatic ^{1}H-MRS and abdominal MRI

Stanley and Fourman, *Lancet HIV* 2019

Other Clinical Parameters

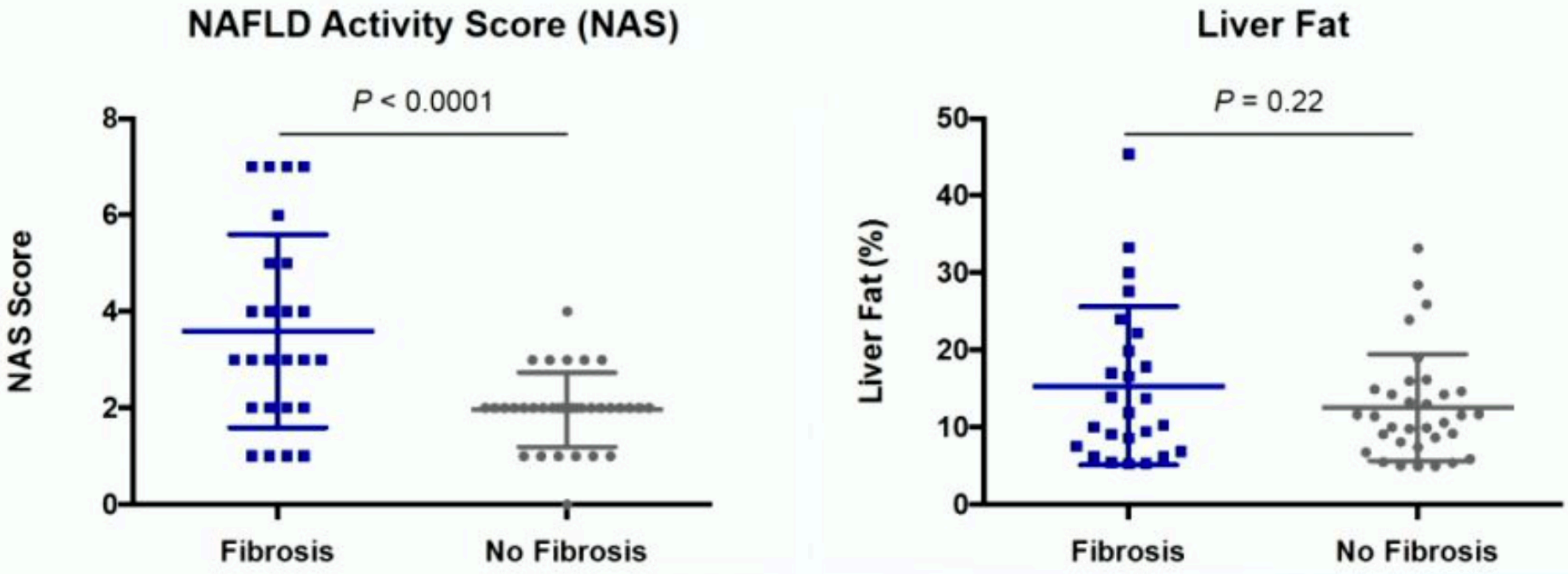
Hepatic	Body Composition	Laboratory
• Liver fat content by ^{1}H-MRS • NAFLD Activity Score (NAS) - Steatosis (0-3) - Lobular inflammation (0-3) - Hepatocellular ballooning (0-2)	• BMI • Waist circumference • Visceral and subcutaneous fat area by abdominal MRI	• Liver enzymes (ALT, AST) • HbA1c • CRP

Prevalence of Baseline Fibrosis in HIV-Associated NAFLD



- Baseline fibrosis had the following distribution: stage 1, 36%; stage 2, 40%; and stage 3, 24%
- Cirrhosis (stage 4 fibrosis) was a criterion for exclusion

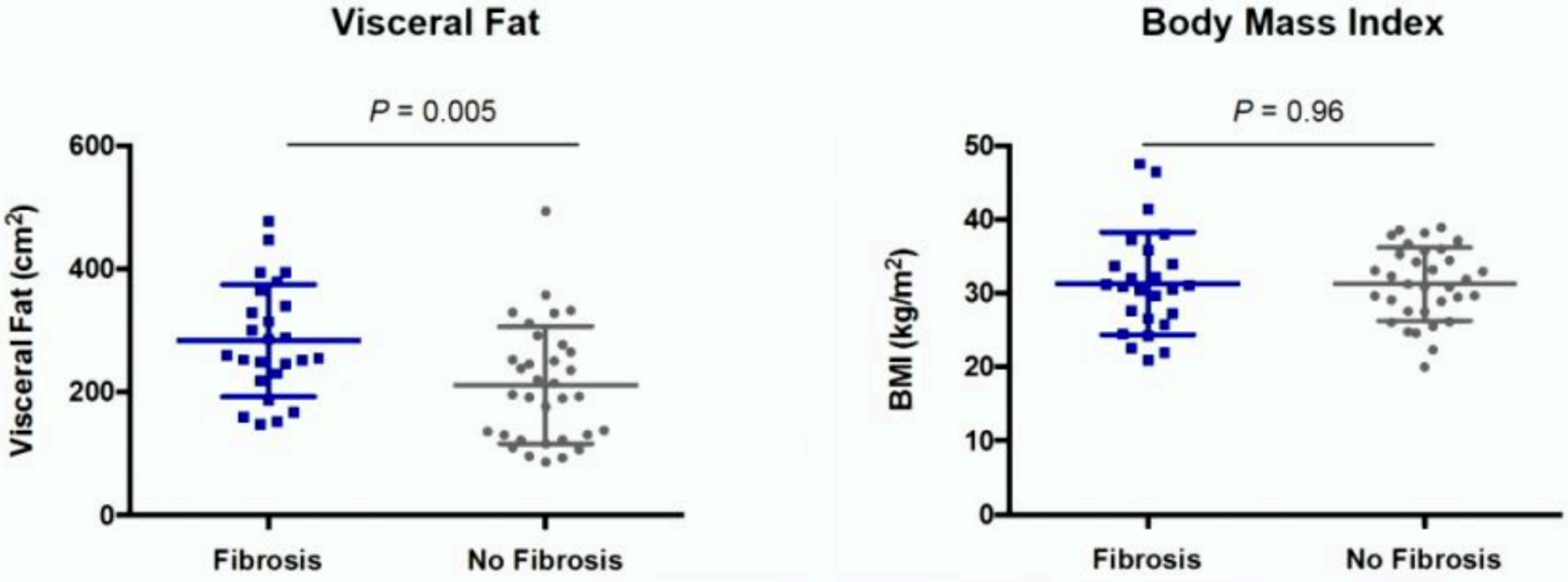
Clinical Correlates of Baseline Fibrosis in HIV-Associated NAFLD



Mean ± SD

- ALT ($P = 0.002$) and AST ($P = 0.0003$) were also higher in individuals with baseline fibrosis

Clinical Correlates of Baseline Fibrosis in HIV-Associated NAFLD

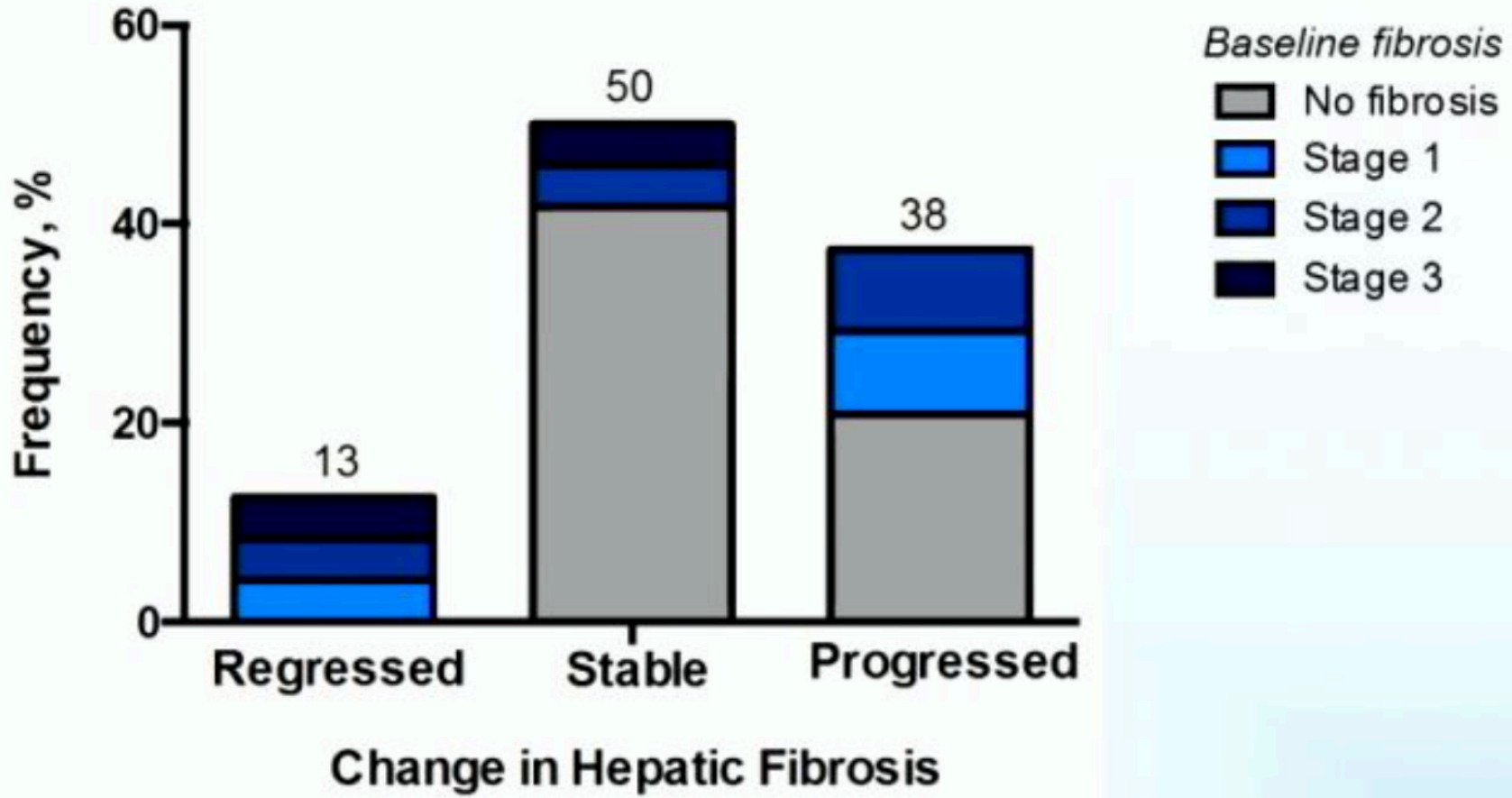


Mean ± SD

- Subcutaneous fat and waist circumference did not differ between groups

Progression of Fibrosis in HIV-Associated NAFLD

Placebo-Treated Participants



- A total of 56% of participants with fibrosis progression had no evidence of fibrosis at baseline
- Mean rate of fibrosis progression was 0.2 ± 0.8 stages per year

Clinical Predictors of Fibrosis Progression in HIV-Associated NAFLD

Odds of Fibrosis Progression in Placebo-Treated Participants

	Univariable Analyses (n = 24)		Multivariable Model (n = 24)	
Baseline Parameter	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
Visceral fat (cm^2)	1.3 (1.0, 1.7)	**0.03**	1.4 (1.0, 2.1)	**0.03**
Liver fat (%)	1.0 (0.9, 1.1)	0.57	1.0 (0.8, 1.2)	0.91
NAS score	0.9 (0.5, 1.5)	0.70	0.7 (0.2, 1.5)	0.34
BMI (kg/m^2)	1.0 (0.9, 1.2)	0.70	1.0 (0.8, 1.3)	0.90

Odds ratios correspond to a 1-unit change in the baseline parameter, except for visceral fat which is calculated based on a 25 cm^2 increment.

Summary

- To our knowledge, this study comprises the first longitudinal assessment of a well-defined sample with HIV-associated NAFLD
- We demonstrated a high prevalence and progression rate of biopsy-proven liver fibrosis
- Rate of fibrosis progression was more than six-fold higher in our study compared to reports of NAFLD in the general population
- Visceral adiposity was found to be a novel clinical predictor of accelerated hepatic disease progression
- Therapies to reduce visceral fat may be particularly effective in HIV-associated NAFLD

Presentaciones seleccionadas de posters en CROI 2020

Cápsulas

HIV CO-INFECTION AND RISK OF MORBIDITY AND MORTALITY IN HCV PATIENTS TREATED BY DAA

M. CHALOUNI[1], S. POL[2-3], P. SOGNI[4-5], H. FONTAINE[2], K. LACOMBE[6-7], JM. LACOMBE[8], L. ESTERLE[1], C. GILBERT[1], C. DORIVAL[6], D. SALMON[4-5], F. CARRAT[6-10]*, L. WITTKOP[1-11]* for the ANRS CO13 HEPAVIH and ANRS CO22 HEPATHER cohorts study grou

* equal contribution, [1]Univ. Bordeaux, ISPED, Inserm, Bordeaux Population Health Research Center, team MORPH3EUS, UMR 1219, CIC-EC 1401, F-33000 Bordeaux, France, [2]AP-HP, Hôpital Cochin, Unité d'Hépatologie, Paris, France, [3]Université Paris Descartes, INSERM U1223 and USM-20, Institut Pasteur, Paris, France, [4]Université Paris Descartes, Paris, France, [5]INSERM U1223, Institut Pasteur, Paris, France, [6]Sorbonne Université, INSERM, Institut Pierre Louis d'Epidémiologie et de Santé Publique, IPLESP, F75012, Paris, France, [7]APHP, Hôpital Saint-Antoine, Service de Maladies Infectieuses et Tropicales, Paris, F75012, France, [8]INSERM Transfert, Paris, France, [9]Service Maladies Infectieuses et tropicales, AP-HP, Hôpital Cochin, Paris, France, [10]AP-HP, Hôpital Saint-Antoine Unité de Santé Publique, Paris, France, [11]CHU de Bordeaux, Pôle de santé publique, F-33000 Bordeaux, France

METHODS

Population: Participants from the ANRS CO13 HEPAVIH cohort (HIV/HCV) and from the ANRS CO22 HEPATHER cohort (HCV) were included if they:

- Initiated a **DAA** treatment between **March 2014** and **December 2017**.
- Treated **outside a clinical trial**.
- **Without history of liver transplantation**.
- With an **available SVR** status.

- **Up to 4 HCV mono-infected** participants were **matched to each HIV/HCV co-infected** participants on **age** (+/- 3 years) and **sex**.

Baseline: Date of **DAA** treatment **initiation**.

Follow-up: **Follow-up ended** at time of **liver-related event**, **death** or **last follow-up**.

Outcomes:

- **Liver-related events:**
  - **Liver decompensation** (liver encephalopathy, hepatorenal syndrome, esophageal varices bleeding, ascites and jaundice)
  - **Hepatocellular carcinoma** (HCC)
  - **Liver transplantation**
  - **Liver-related death**
- **Liver-related mortality:** Death due to HCV
- **Non-liver-related mortality:** Death due to any cause other than HCV

- After adjusting on confounding factors, in **HCV participants treated by DAA**, **HIV co-infection** was:
  - **Not associated** with the risk of **liver-related events** (HR: 0.7 [0.3 ; 1.4]) **neither** with the risk of **liver-related mortality** (HR: 1.0 [0.3 ; 3,.2])
  - **Associated** with a significant **increased risk** of **non-liver-related mortality** (HR: 2.8 [1.3 ; 6.4])

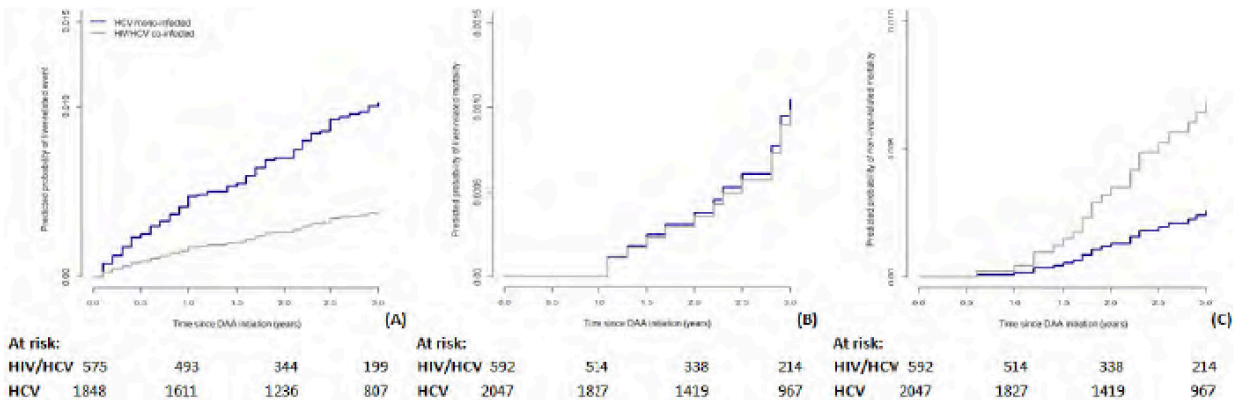
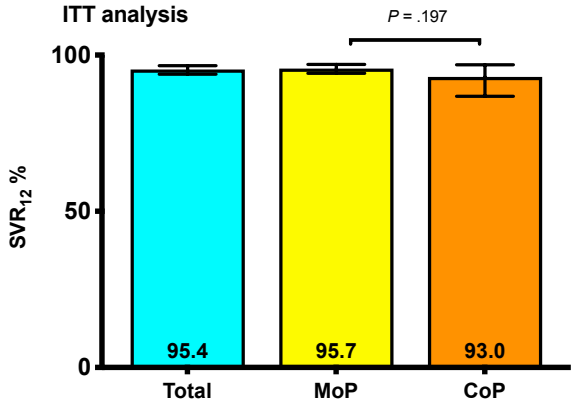
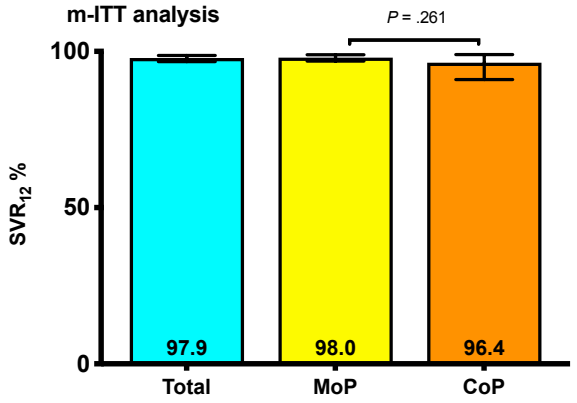


Figure 1. Predicted probabilities of liver-related events (A), liver-related mortality (B) and non-liver-related mortality (C) according to HIV co-infection in HCV infected participants from the ANRS CO13 HEPAVIH and ANRS CO22 HEPATHER cohorts, estimated for mean participants

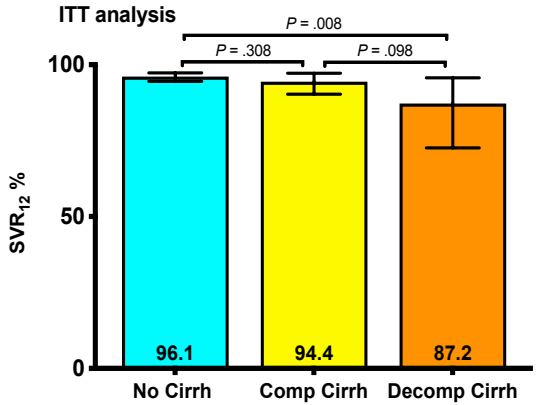
Real-world effectiveness of Sofosbuvir/Velpatasvir for Hepatitis C Virus Infection



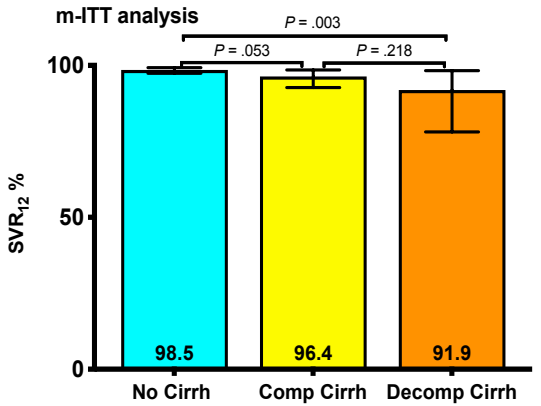
	Total	MoP	CoP
No.	1,103	888	115
SVR ITT	957 (95.4)	850 (95.7)	107 (93.0)
SVR (95% CI)	(93.9 - 96.6)	(94.2 - 97.0)	(86.8 - 96.9)
Relapse	17 (1.7)	14 (1.6)	3 (2.6)
DC due to AE	2 (0.2)	2 (0.2)	0
DC other	25 (2.5)	21 (2.4)	4 (3.5)
Death	2 (0.2)	1 (0.1)	1 (0.9)



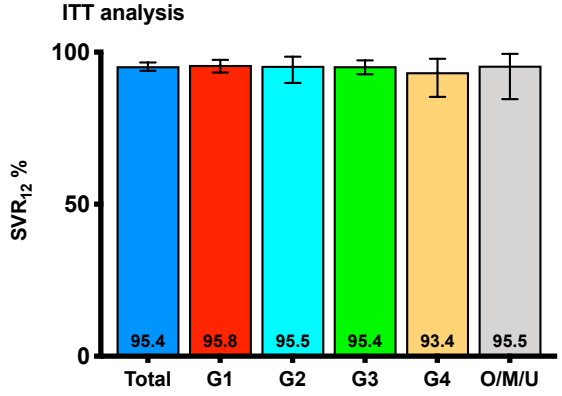
	Total	MoP	CoP
No.	978	867	111
SVR m-ITT	957 (97.9)	850 (98.0)	107 (96.4)
SVR (95% CI)	96.7-98.7	96.9-98.9	91.0-99.0
Relapse	17 (1.7)	14 (1.6)	3 (2.7)
DC due to AE	22 (2.2)	2 (0.2)	0
Death	2 (0.2)	1 (0.1)	1 (0.9)



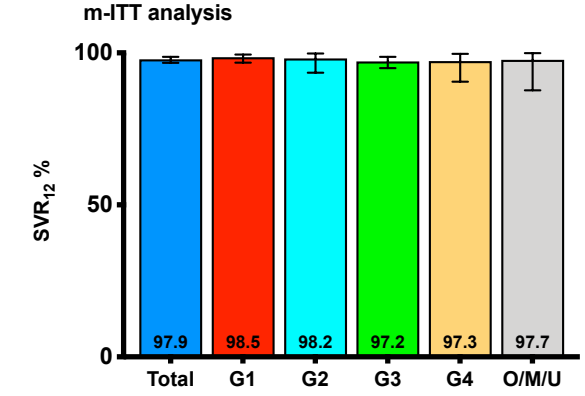
	No Cirrh	Comp Cirrh	Decomp Cirrh
No.	766	198	39
SVR ITT	736 (96.1)	187 (94.4)	34 (87.2)
SVR (95% CI)	94.5-97.3	90.3-97.2	72.6-95.7
Relapse	9 (1.2)	5 (2.5)	3 (7.7)
DC due to AE	2 (0.3)	0	0
DC other	19 (2.5)	4 (2.0)	2 (5.1)
Death	0	2 (1.0)	0



	No Cirrh	Comp Cirrh	Decomp Cirrh
No.	747	194	37
SVR ITT	736 (98.5)	187 (96.4)	34 (91.9)
SVR (95% CI)	97.4-99.3	92.7-98.5	78.1-98.3
Relapse	9 (1.2)	5 (2.6)	3 (8.1)
DC due to AE	2 (0.3)	0	0
Death	0	2 (1.0)	0



	Total	G1	G2	G3	G4	O/M/U
No.	1,003	401	112	370	76	44
SVR ITT	957 (95.4)	384 (95.8)	107 (95.5)	353 (95.4)	71 (93.4)	42 (95.5)
SVR 95% CI	93.9-96.6	93.3-97.5	89.9-98.5	92.7-97.3	85.3-97.8	84.5-99.4
Relapse	17 (1.7)	3 (0.7)	2 (1.8)	9 (2.4)	2 (2.6)	1 (2.3)
DC due to AE	2 (0.2)	1 (0.2)	0	1 (0.3)	0	0
DC other	25 (2.5)	11 (2.7)	3 (2.7)	7 (1.9)	3 (3.9)	1 (2.3)
Death	2 (0.2)	2 (0.5)	0	0	0	0



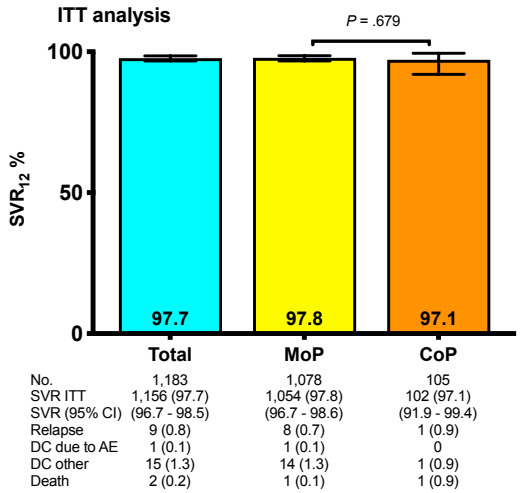
	Total	G1	G2	G3	G4	O/M/U
No.	978	390	109	363	73	43
SVR ITT	957 (97.9)	384 (98.5)	107 (98.2)	353 (97.2)	71 (97.3)	42 (97.7)
SVR (95% CI)	96.7-98.7	96.7-99.4	93.5-99.8	95.0-98.7	90.5-99.7	87.7-99.9
Relapse	17 (1.7)	3 (0.8)	2 (1.8)	9 (2.5)	2 (2.7)	1 (2.3)
DC due to AE	2 (0.2)	1 (0.3)	0	1 (0.3)	0	0
Death	2 (0.2)	2 (0.5)	0	0	0	0

MoP = HCV monoinfected patients
CoP = HIV/HCV coinfected patients

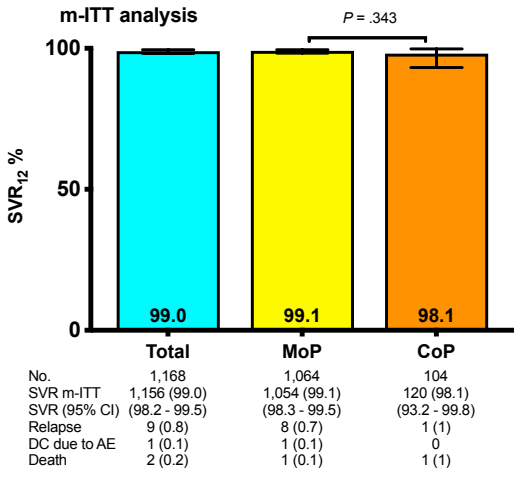
m-ITT analysis = exclusion of patients with DC for reasons other than AE

Aldamiz-Echevarría T et al. # 572

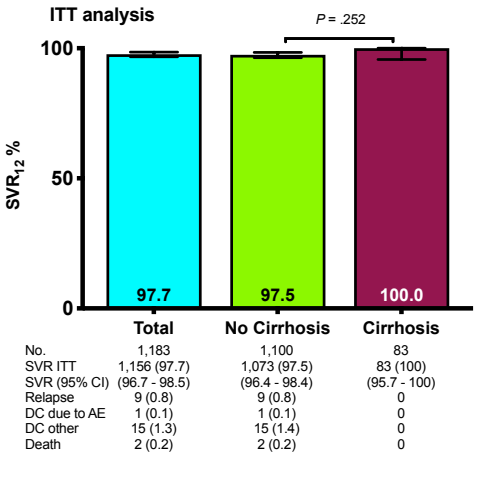
Real-world effectiveness of Glecaprevir/Pibrentasvir for Hepatitis C Virus Infection



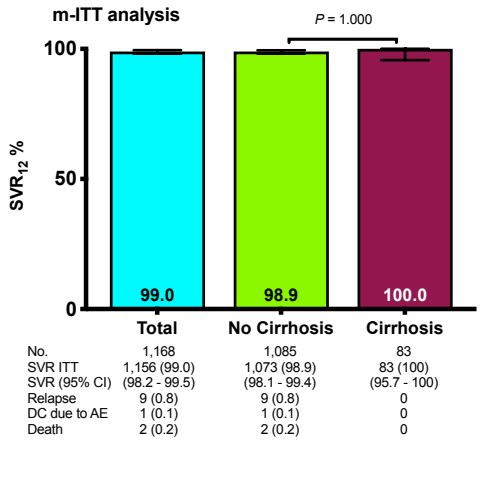
	Total	MoP	CoP
No.	1,183	1,078	105
SVR ITT	1,156 (97.7)	1,054 (97.8)	102 (97.1)
SVR (95% CI)	(96.7 - 98.5)	(96.7 - 98.6)	(91.9 - 99.4)
Relapse	9 (0.8)	8 (0.7)	1 (0.9)
DC due to AE	1 (0.1)	1 (0.1)	0
DC other	15 (1.3)	14 (1.3)	1 (0.9)
Death	2 (0.2)	1 (0.1)	1 (0.9)



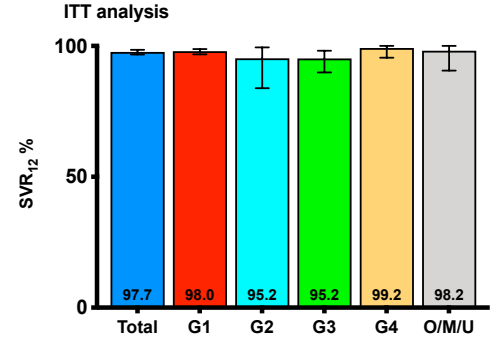
	Total	MoP	CoP
No.	1,168	1,064	104
SVR m-ITT	1,156 (99.0)	1,054 (99.1)	120 (98.1)
SVR (95% CI)	(98.2 - 99.5)	(98.3 - 99.5)	(93.2 - 99.8)
Relapse	9 (0.8)	8 (0.7)	1 (1)
DC due to AE	1 (0.1)	1 (0.1)	0
Death	2 (0.2)	1 (0.1)	1 (1)



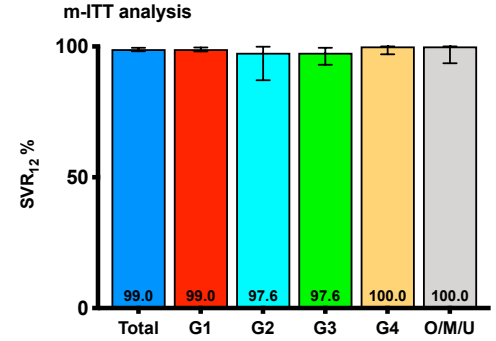
	Total	No Cirrhosis	Cirrhosis
No.	1,183	1,100	83
SVR ITT	1,156 (97.7)	1,073 (97.5)	83 (100)
SVR (95% CI)	(96.7 - 98.5)	(96.4 - 98.4)	(95.7 - 100)
Relapse	9 (0.8)	9 (0.8)	0
DC due to AE	1 (0.1)	1 (0.1)	0
DC other	15 (1.3)	15 (1.4)	0
Death	2 (0.2)	2 (0.2)	0



	Total	No Cirrhosis	Cirrhosis
No.	1,168	1,085	83
SVR ITT	1,156 (99.0)	1,073 (98.9)	83 (100)
SVR (95% CI)	(98.2 - 99.5)	(98.1 - 99.4)	(95.7 - 100)
Relapse	9 (0.8)	9 (0.8)	0
DC due to AE	1 (0.1)	1 (0.1)	0
Death	2 (0.2)	2 (0.2)	0



	Total	G1	G2	G3	G4	O/M/U
No.	1,183	837	42	126	121	57
SVR ITT	1,156 (97.7)	820 (98.0)	40 (95.2)	120 (95.2)	120 (99.2)	56 (98.2)
SVR (95% CI)	(96.7 - 98.5)	(96.8 - 98.8)	(83.8 - 99.4)	(89.9 - 98.2)	(95.5 - 100)	(90.6 - 100)
Relapse	9 (0.8)	6 (0.7)	1 (2.4)	2 (1.6)	0	0
DC due to AE	1 (0.1)	0	0	1 (0.8)	0	0
DC other	15 (1.3)	9 (1.1)	1 (2.4)	3 (2.4)	1 (0.8)	1 (1.8)
Death	2 (0.2)	2 (0.2)	0	0	0	0



	Total	G1	G2	G3	G4	O/M/U
No.	1,168	828	41	123	120	56
SVR ITT	1,156 (97.7)	820 (99.0)	40 (97.6)	120 (97.6)	120 (100)	56 (100)
SVR (95% CI)	(98.2 - 99.5)	(98.1 - 99.6)	(87.1 - 99.9)	(93.0 - 99.5)	(97.0 - 100)	(93.6 - 100)
Relapse	9 (0.8)	6 (0.7)	1 (2.4)	2 (1.6)	0	0
DC due to AE	1 (0.1)	0	0	1 (0.8)	0	0
Death	2 (0.2)	2 (0.2)	0	0	0	0

MoP = HCV monoinfected patients
CoP = HIV/HCV coinfected patients

m-ITT analysis = exclusion of patients with DC for reasons other than AE

Domínguez-Domínguez L et al. # 571

HEPATITIS E RABBIT GENOTYPE INFECTION in HIV-INFECTED PATIENTS

Antonio Rivero-Juarez[1], Mario Frias[1], Pedro Lopez-Lopez[1], Juan Berenguer[2], Federico García[3], Juan Macias[4], Begoña Alcaraz[5], Angeles Castro-Iglesias[6], Javier Caballero-Gomez[1,7], Angela Camacho[1], Isabel Machuca[1], Antonio Rivero[1], on behalf of CoRIS Cohort

1. Instituto Maimonides de Investigación Biomédica de Córdoba (IMIBIC). Hospital Universitario Reina Sofía de Córdoba. Universidad de Córdoba. Córdoba, Spain. 2. Hospital General Universitario Gregorio Marañón. Instituto de Investigación Sanitaria Gregorio Marañón (IISGM). Madrid, Spain. 3. Hospital Universitario San Cecilio. Instituto de Investigación Biosantaria Ibs.Granada, Spain. 4. Hospital Nuestra Señora de Valme. Seville, Spain. 5. Hospital General Universitario Santa Lucía. Cartagena, Spain. 6. Complejo Hospitalario Universitario a Coruña (CHUAC). A Coruña, Spain. 7. University of Cordoba - Agrifood Excellence International Campus (ceiA3), Cordoba, Spain.

Abstract 618

Results

Study population

- A total of 845 individuals were included in the study.
- Seven hundred and fifty-one (88.9%) were male and had a median age of 36.9 years (30.7-45.2 years).

Prevalence of HEV infection

- One hundred and one patients showed positivity of IgG against HEV (11.9%; 95% CI: 9.9%-14.3%). There were no variables associated with IgG positivity in the univariate and multivariate analyses (Table 1).
- Any of the patients included in the study were positive for IgM anti-HEV

Incidence of HEV infection

- Of the 744 patients negative for IgG antibodies at baseline, 733 (98.5%) had available serum samples in the follow-up.
- Forty-two patients showed seroconversion for IgG antibodies after 1 year of follow-up, supposing a cumulative incidence of 5.7% (95% CI: 4.3%-7.7%).
- In Table 2 we show univariate and multivariate analysis for HEV seroincidence.

SELECCION COMUNICACIONES EN COINFECCION VIRUS DE HEPATITIS

MUCHAS GRACIAS

Juan González-García

09 de Junio de 2020