Abstract P156 Table 1 Characteristics of the study participants

	BIC (429)	DOR (90)	p
Sex, n (%)			0,387
Male	300 (69,9)	61 (67,8)	
Female	129 (30,1)	29 (32,2)	
Age, years, median (IQR)	54 (45-59)	50 (45-56)	0,005
Risk Factor, n (%)			0,438
Heterosexual	186 (43,4)	41 (45,6)	
MSM	149 (34,7)	36 (40,0)	
PWID	62 (14,5)	9 (10,0)	
Other/Unknown	32 (7,5)	4 (4,4)	
HCV, n (%)	5 (1,2)	0 (0)	0,384
diabete	29 (6,8)	6 (6,7)	0,595
statina al BL	11 (2,6)	1 (1,1)	0,355
Evento CV major	23 (5,4)	1 (1,1)	0,058
dislipidemia	355 (82,8)	71 (78,9)	0,233
CDC C Stage, n (%)	155 (36,1)	32 (35,6)	0,716
Zenith HIV-RNA, log_{10} copies/mL, median (IQR)	5,00 (4,19-5,46)	5,15 (4,60-5,63)	0,048
CD4 nadir cells/mm³, median (IQR)	161 (40-297)	167 (52-289)	0,961
HIV-RNA TND, n (%)	368 (85,8)	78 (86,7)	0,489
CD4 cells/mm³, median (IQR)	599 (419-794)	742 (519-886)	0,002
CD4/CD8 ratio, median (IQR)	0,87 (0,57-1,27)	0,97 (0,68-1,25)	0,257
Time since HIV diagnosis, years, median (IQR)	17 (6-25)	14 (6-22)	0,200
Time on ART ,years, median (IQR)	13 (5-21)	13 (5-19)	0,505
Previous VF, n (%)	186 (43,4)	30 (33,3)	0,099
Cumulative time under suppression, months , median (IQR)	9 (3-14)	10 (3-13)	0,328

Abbreviations. MSM: Men who have sex with men; PWID: people who inject drugs; ART: Antiretroviral therapy; dr: drug regimen; NRTI: Nucleoside Reverse Transcriptase Inhibitor; INSTI: Integrase Strand Transfer Inhibitor; NNRTI: Non-Nucleoside Reverse Transcriptase Inhibitor; PI/b: Protease inhibitors/boosted.

significantly increased at 48W (+39, p=0.020), with a greater rise in the DOR group than in the BIC group (p<0.001); overall no significant variations was observed at 96W, however a greater gain was found in the DOR group (p<0.001). The probability of virological failure was similar between the groups (6.1% vs 4.4%, p=0.881), while TD was more frequent with DOR (13.1% vs 18.9%, p=0.044).

Conclusions Both BIC/FTC/TAF and DOR/3TC/TDF were effective switch options in virologically suppressed PLWH, ensuring durable virological control and favorable lipid effect up to 96W. These findings support their use in clinical practice, particularly when metabolic profile is a relevant consideration.

P164 SWITCHING TO DTG/3TC OR FTC/TAF/BIC IN PEOPLE LIVING WITH HIV ALREADY ON A STABLE TAF INCLUDING REGIMEN DOES NOT CHANGE WEIGHT AND LIPID PROFILE

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Background Our aim was to investigate the role of switching from emtricitabine/tenofovir alafenamide (TAF) based regimen to a dolutegravir and lamivudine (DTG) vs BIC/FTC/TAF (TAF) on metabolic parameters.

Material and Methods Consecutive people living with HIV infection (PWH) enrolled in a multicenter observational cohort (SCOLTA) project, on a stable FTC/TAF based regimen with a HIV-RNA<50 copies/ml were included. Changes from baseline (T0) to follow-up (T1, week 24 and T2, week 36) were analyzed.

Abstract P164 Table 1 Blood lipids change to 6- and 12-month follow-up visit (multivariate model)

	T1-T0 Mean (95% CI)			T2-T0 Mean (95% CI)		
All PWH	**3TC/DTG n=126**	**FTC/TAF/BIC n=394**	**P**	**3TC/DTG n=100**	**FTC/TAF/BIC n=317**	**P**
Weight (Kg) (n=404)	0.4 (-0.6 to 1.4)	0.4 (-0.3 to 1.1)	0.99	0.3 (-1.4 to 1.9)	0.0 (-1.1 to 1.2)	0.78
BMI (Kg/m²) (n=370)	0.18 (-0.16 to 0.52)	0.16 (-0.08 to 0.41)	0.92	0.19 (-0.28 to 0.67)	0.01 (-0.33 to 0.34)	0.42
TC (mg/dL) (n=479)	**-9 (-18 to -1)**	-7 (-13 to 0)	0.37	-11 (-22 to 0)	-7 (-17 to 2)	0.38
HDL (mg/dL) (n=467)	-2 (-5 to 1)	0 (-2 to 2)	0.17	-1 (-6 to 4)	-1 (-5 to 3)	0.98
LDL-c (mg/dL) (n=462))	-4 (-12 to 4)	-3 (-9 to 3)	0.68	**-12 (-23 to -2)**	**-9 (-17 to -0)**	0.32
TGL (mg/dL) (n=475)	-8 (-25 to 8)	-10 (-23 to 2)	0.78	10 (-11 to 32)	8 (-9 to 25)	0.73
No COBI in previous regimen	**n=84**	**n=138**	**P**	**n=64**	**n=118**	**P**
Weight (Kg)	0.3 (-0.0 to 1.5)	0.2 (-0.7 to 1.2)	0.92	0.2 (-1.6 to 2.0)	-0.3 (-1.8 to 1.1)	0.57
BMI (Kg/m²)	0.15 (-0.24 to 0.55)	0.10 (-0.22 to 0.42)	0.77	0.13 (-0.43 to 0.69)	-0.06 (-0.51 to 0.39)	0.50
TC (mg/dL)	-4 (-18 to 9)	-3 (-14 to 8)	0.84	-6 (-23 to 11)	-7 (-21 to 7)	0.82
HDL (mg/dL)	-1 (-5 to 3)	-0 (-4 to 3)	0.75	1 (-7 to 9)	2 (-5 to 9)	0.67
LDL-c (mg/dL)	1 (-12 to 13)	2 (-8 to 13)	0.70	-5 (-21 to 11)	-4 (-17 to 10)	0.76
TGL (mg/dL)	-6 (-29 to 17)	-10 (-29 to 8)	0.63	11 (-24 to 48)	2 (-28 to 32)	0.42
With COBI in previous regimen	**n=42**	**n=256**	**P**	**n=36**	**n=199**	**P**
Weight (Kg)	0.6 (-1.0 to 2.1)	0.7 (-0.1 to 1.5)	0.89	0.3 (-2.5 to 3.1)	0.4 (-1.1 to 1.9)	0.94
BMI (Kg/m²)	0.19 (-0.35 to 0.74)	0.28 (-0.03 to 0.56)	0.80	0.28 (-0.49 to 1.06)	0.11 (-0.31 to 0.54)	0.66
TC (mg/dL)	**-18 (-29 to -6)**	**-12 (-20 to -4)**	0.24	**-19 (-35 to -4)**	**-12 (-23 to -0)**	0.20
HDL (mg/dL)	-3 (-7 to 2)	0 (-2 to 3)	0.10	-2 (-9 to 5)	-3 (-8 to 1)	0.59
LDL-c (mg/dL)	**-12 (-22 to -1)**	**-10 (-17 to -3)**	0.69	**-20 (-34 to -6)**	**-14 (-24 to -4)**	0.32
TGL (mg/dL)	-16 (-40 to 9)	-12 (-27 to 3)	0.72	10 (-18 to 38)	11 (-8 to 30)	0.93

Bold: p<0.05 for change from baseline. Overall estimates and comparison between groups are adjusted for initial value of the variable, previous regimen class (with or without COBI), and CDC stage; models for blood lipids also included statin use at T0 and HCV co-infection. In strata of COBI use, estimates and comparison between groups are adjusted for initial value of the variable, previous regimen class, and CDC stage; models for blood lipids also included statin use at T0 and HCV co-infection.
BMI: body mass index; COBI: cobicistat; FTC: emtricitabine; IQR: interquartile range; LDL-c: low-density lipoprotein cholesterol; PWH: people with HIV; SD: standard deviation; SE: standard error; TAF: tenofovir alafenamide; TC: total cholesterol; TGL: triglycerides.

Results Five hundred and twenty PWH met the inclusion criteria, 408 (78.5%) were males, 298 (57.3%) were on a previous cobicistat including regimen (COBI-IR).

At T0 main characteristics were (mean ± standard deviation [SD]): age 49.7 ± 11.9 years, weight 75.9 Kg ±13.9, body mass index (BMI) 25.7 ± 4.1 kg/m^2, total cholesterol (TC) 192 ± 41 mg/dL, LDL-cholesterol (LDL-c) 114 ± 36 mg/dL, CD4+ cell count median value was 680 cell/μL (interquartile range [IQR] 498-908), triglycerides (TGL) 113 mg/dL (IQR 82-158). Median time of observation was 32 months (IQR 16-48).

PWH switching to DTG differed significantly by TAF for: INSTI and NNRTI in previous regimen (65.9% vs 81.0% and 25.4% vs 5.3% respectively; p<0.001), risk factor for HIV infection (Intravenous Drug users 6.3% vs 18.3%, sexual 87.3% vs 66.5%, other/unknown 6.4% vs 15.2%, p<0.0001), HCV coinfection (7.9% vs 18.3%, p<0.0001), baseline CD4 (median 772 [IQR 585-978] and 656 [IQR 469-882] , p=0.0005), CDC stage C (10.3% vs 18.3%, p=0.001), years of Antiretroviral Therapy (median 6.9 [IQR 3.1-13.3] vs 8.6 [IQR 3.8-17.1], p=0.01) and previous COBI-IR (33.3% in 2DR vs 65.9% in TAF, p<0.0001). PWH with previous COBI-IR had higher levels of TC (210 ± 39 mg/dl vs 196 ± 41 mg/dL, p=0.046) at enrollment. PWH on statin therapy were 11.1% in DTG vs 18.0% in TAF group (p=0.07).

In a multivariate model adjusted for initial value of the variable, previous regimen class (with or without cobicistat), CDC stage, statin use and HCV-coinfection, no significant differences were found between DTG and TAF group as regards weight, BMI and lipid profile (see table 1).

On the entire sample, TC reduced significantly in DTG group at T1 (-9 mg/dl [95% confidence interval -18 to -1], whereas at T2 the levels were still lower than T0 but not statistically significant. At T2 LDL-Cholesterol (LDL-c) showed a further decrease, both in DTG and TAF (-12 mg/dl [95% CI -23 to-2] and -9 mg/dl [95% CI -17 to -0] respectively) with no differences between the two groups.

In PWH on previous COBI-IR we observed significant declines in TC and LDL-C both at T1 and T2 with no differences between DTG and TAF.

Conclusions In PWH already on TAF, stopping TAF does not have an impact on lipid profile and BMI. Switching to DTG+3TC or BIC/FTC/TAF is associated with TC and LDL reduction independently of previous regimen. Weight and BMI were not influenced by switching TAF or COBI.

Pharmacology

P168 COMPREHENSIVE PHARMACOGENETIC, PHARMACOKINETIC AND CLINICAL ASSESSMENT OF BULEVIRTIDE IN CHRONIC HEPATITIS D PATIENTS

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Background Bulevirtide represents the first authorized entry inhibitor indicated for the management of chronic hepatitis D (CHD), exerting its antiviral activity by blocking the sodium taurocholate co-transporting polypeptide (NTCP). Although clinical results have been promising, marked variability among patients in drug exposure and therapeutic effectiveness has been reported. So far, limited evidence has explored the variability in bulevirtide plasma concentrations and the potential role of genetic polymorphisms. The present study sought to evaluate bulevirtide plasma levels, along with biochemical and virological markers at different time points, and to investigate the impact of NTCP gene variants on clinical outcomes, taking into account virological and biochemical responses at week (W) 60.

Material and Methods patients with CHD treated with bulevirtide were included in the study. Bulevirtide plasma levels, together with biochemical and virological markers, were measured at baseline and at weeks (W) 4/12/24/36/48 and 60 using liquid chromatography-mass spectrometry. The pharmacogenetic evaluation targeted NTCP genetic variants (rs8011311-rs2296651), analyzed through PCR genotyping.

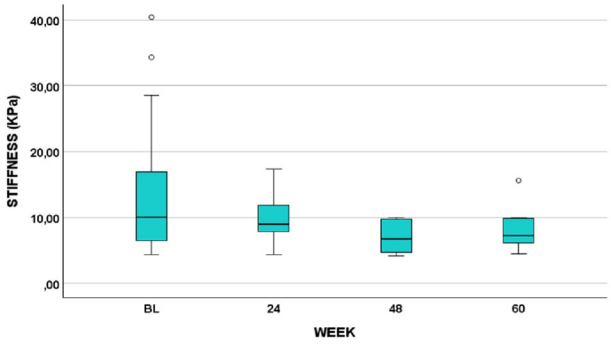
Results Seventeen patients were initially recruited; five were later excluded due to incomplete follow-up and inadequate adherence to therapy. Among the twelve patients included in the final analysis, 75% were male. Median age was 51.5 years and median BMI was 24.5 kg/m^2; cirrhosis was present in 50% of cases. At baseline, median ALT level was 80.5 IU/L and median HDV RNA was 5.3 Log. Liver stiffness significantly decreased at week 48 compared with baseline (p=0.041, figure 1). Lipid parameters were not affected by treatment, while a slight, non-significant increase in bile acids was observed. No adverse events were reported.

At week 60, virological response was achieved in 4 patients (33.3%), biochemical response in 4 (33.3%), and a combined response in 3 (25%).

Median plasma bulevirtide concentration was 0.4 ng/mL (IQR 0.3–0.5) at W4, 0.9 ng/mL (IQR 0.4–1.9) at W12, 0.6 ng/mL (IQR 0.3–1.6) at W24, 1.0 ng/mL (IQR 0.5–1.3) at W36, 1.2 ng/mL (IQR 0.5–7.2) at W48, and 0.7 ng/mL (IQR 0.3–2.1) at W60. Concomitant medications did not appear to influence bulevirtide exposure, whereas liver stiffness values >12 kPa were associated with differences in drug levels, particularly at week 12 (p=0.026).

Regarding pharmacogenetic findings, patients carrying the NTCP rs8011311 GC/CC genotype exhibited lower bulevirtide concentrations compared with those with the GG genotype at week 4 (p=0.047).

Conclusions This exploratory study is the first to describe bulevirtide plasma concentrations in patients undergoing 60 weeks of treatment and to investigate the influence of genetic factors, although sample size was limited. Larger-scale studies are needed to support the development of individualized dosing



Abstract P168 Figure 1 Stiffness levels during the different weeks