staying on current ART, switching to another oral ART or switching to a LAI ART. DCE treatment attributes include dosing frequency, side effects, forgivability, food/mealtime restrictions, and mode of administration. Pilot data for US patients is included here; the main survey will include approximately 550 patients and 450 physicians.

Results. Of 51 PLHIV completing the pilot survey, 80% were male, mean age was 54 years, and 63% were on ART for ≥10 years. Switching ART was common, with 55% reporting changing their ART ≥3 times. Just under half of patients (47%) were not totally satisfied with their current ART. Most common reasons for dissatisfaction included daily reminder of having HIV (31%) and having to take medicine every day (28%). Just over a quarter of patients (28%) reported forgetting to take their ART in the prior month. Across all DCE choices, patients preferred to remain on their current treatment 47% of the time, while 45% of the time patients preferred switching to the LAI, and for the remaining 8%, patients chose switching to another oral ART regimen.

Conclusion. Despite advances in ART treatment challenges remain. Among the treatment-experienced PLHIV in this pilot survey, over half of their choices resulted in switching to an alternative regimen, and when opting to switch, most patients preferred the long-acting injectable treatment regimen.

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2500. Fostemsavir Drug–Drug Interaction Profile, an Attachment Inhibitor and Oral Prodrug of Temsavir, for Heavily Treatment Experienced HIV-1-Infected Patients

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Background. Fostemsavir (FTR) is a first-in-class attachment inhibitor being evaluated in heavily treatment-experienced (HTE) HIV-1-infected patients. Active temsavir (TMR) binds to viral envelope glycoprotein 120 and prevents viral attachment and entry into host CD4+ T cells. TMR is primarily metabolized by esterase-mediated hydrolysis with contributions from cytochrome P450 (CYP) 3A4. TMR is not inhibited/induced major CYP or uridine diphosphate glucuronosyltransferase (UGT) enzymes and is a P-glycoprotein (P-gp) and breast cancer resistance protein (BCRP) substrate. TMR and/or its metabolites inhibit BCRP and organic anion transporter protein 1B1/3 (OATP1B1/3). FTR DDI profile informs coadministration with antiretrovirals (ARV) and other therapeutic classes.

Methods. DDI data from 13 studies were collected to impact the effect of 17 drugs or drug combinations on TMR and the impact of TMR on 15 drugs such as ARVs, rifampycins, opioid substitutes, statins, oral contraceptives (OC), and H2-antagonists.

Results. FTR with CYP3A4, P-gp, and/or BCRP inhibitors increase TMR concentrations; but, do not pose clinical concern at therapeutic dose. TMR may be administered with weak/moderate inducers with or without coadministration of CYP3A4, P-gp, and/or BCRP inhibitors such as RVT or COBL. Coadministration with strong inducers is contraindicated. FTR may be coadministered with RBT with or without a PK enhancer. However, co-administration of FTR with RIF is contraindicated. FTR can be given with drugs that increase gastric pH; famotidine did not impact TMR PK. FTR may increase concentrations of drugs that are substrates of OATP1B1/3 and BCRP; therefore, most statins require dose reduction (e.g., rosuvastatin dose is limited to ≤10 mg QD). TMR increased EE exposure 40% with no impact on NE; therefore, FTR may be coadministered with OCs containing ≤30 μg EE. TMR had no clinically meaningful impact on TDF, DRV/RTV, ATV/RTV, ATV, RTV, ETR, MET, or BUP/riton/BUP FK (Table 1).

Conclusion. FTR can be coadministered with ARVs and most common treatments used to manage HIV co-infections or comorbidities without dose adjustment of either drug except for select HMG-CoA reductase inhibitors and EE-containing OCs. Strong CYP3A4 inducers are contraindicated.

Disclosures. All authors: No reported disclosures.