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 ≥2 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 not forgeting 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 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.

**Disclosures. All authors:** No reported disclosures.

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

**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 does not inhibit/induce 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 compiled to inform the impact of 17 drugs or drug combinations on TMR and the impact of TMR on 15 drugs such as ARVs, rifampicin, 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 in switching to an alternative regimen, and when opting to switch, most patients preferred the long-acting injectable treatment regimen.

**Disclosures. All authors:** No reported disclosures.