

Preexisting and Post-Baseline Resistance Analyses in Pooled Pediatric Studies of Emtricitabine/Tenofovir Alafenamide (F/TAF)–Based Antiretroviral Therapy

Supplementary Materials

Kristen Andreatta,¹ Stephanie Cox,¹ Kulkanya Chokephaibulkit,² Carina A. Rodriguez,³ Afaaf Liberty,⁴ Eva Natukunda,⁵ Vinicius A. Vieira,¹ Kathryn Kersey,¹ Christian Callebaut¹

¹Gilead Sciences, Inc., Foster City, California, U.S.A.; ²Mahidol University, Bangkok, Thailand; ³Morsani College of Medicine, University of South Florida, Tampa, Florida, U.S.A.; ⁴Chris Hani Baragwanath Hospital, Johannesburg, South Africa; ⁵Joint Clinical Research Centre, Kampala, Uganda

Methods: HIV-1 Drug Resistance Substitutions (Based on IAS-USA list¹)

NRTI-R	K65R/E/N, T69 insertions, K70E, L74V/I, Y115F, Q151M, M184V/I, TAMs (M41L, D67N, K70R, L210W, T215F/Y, K219E/N/Q/R)
NNRTI-R	L100I, K101E/P, K103N/S, V106A/M, V108I, E138A/G/K/Q/R, V179L, Y181C/I/V, Y188C/H/L, G190A/E/Q/S, H221Y, P225H, F227C, M230I/L
PI-R	D30N, V32I, M46I/L, I47A/V, G48V, I50L/V, I54M/L, Q58E, T74P, L76V, V82A/F/L/S/T, N83D, I84V, N88S, L90M
INSTI-R	Primary: T66I/A/K, E92Q/G, T97A, F121Y, Y143R/H/C, S147G, Q148H/K/R, N155H/S, R263K

IAS-USA, International Antiviral Society—USA; INSTI, integrase strand transfer inhibitor; NNRTI, non-nucleoside reverse transcriptase inhibitor; NRTI, nucleos(t)ide reverse transcriptase inhibitor; PI, protease inhibitor; R, resistance; TAM, thymidine analog mutation

1. Wensing AM, et al. Top Antivir Med 2022;30:559-574

Results: Summary of Study Treatments

	F/TAF pooled pediatric participants N = 341	TN participants n = 50	VS participants n = 291
Study regimen, n (%)			
B/F/TAF	122 (36)	0	122 (42)
E/C/F/TAF	179 (53)	50 (100)	129 (44)
F/TAF + 3rd agent*	40 (12)	0	40 (14)
Low-dose version of B/F/TAF, E/C/F/TAF or F/TAF†	52 (15)	0	52 (18)

*3rd agents were boosted (n = 24: atazanavir/ritonavir, darunavir/cobicistat, darunavir/ritonavir, lopinavir/ritonavir) or unboosted (n = 13: dolutegravir, efavirenz, nevirapine)

†Low-dose B/F/TAF (n = 22), E/C/F/TAF (n = 27), F/TAF + 3rd agent (n = 12)

B/F/TAF, bictegravir/emtricitabine/tenofovir alafenamide; E/C/F/TAF, elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide; F/TAF, emtricitabine/tenofovir alafenamide; TN, treatment-naïve; VS, virologically suppressed

Results: Preexisting Resistance

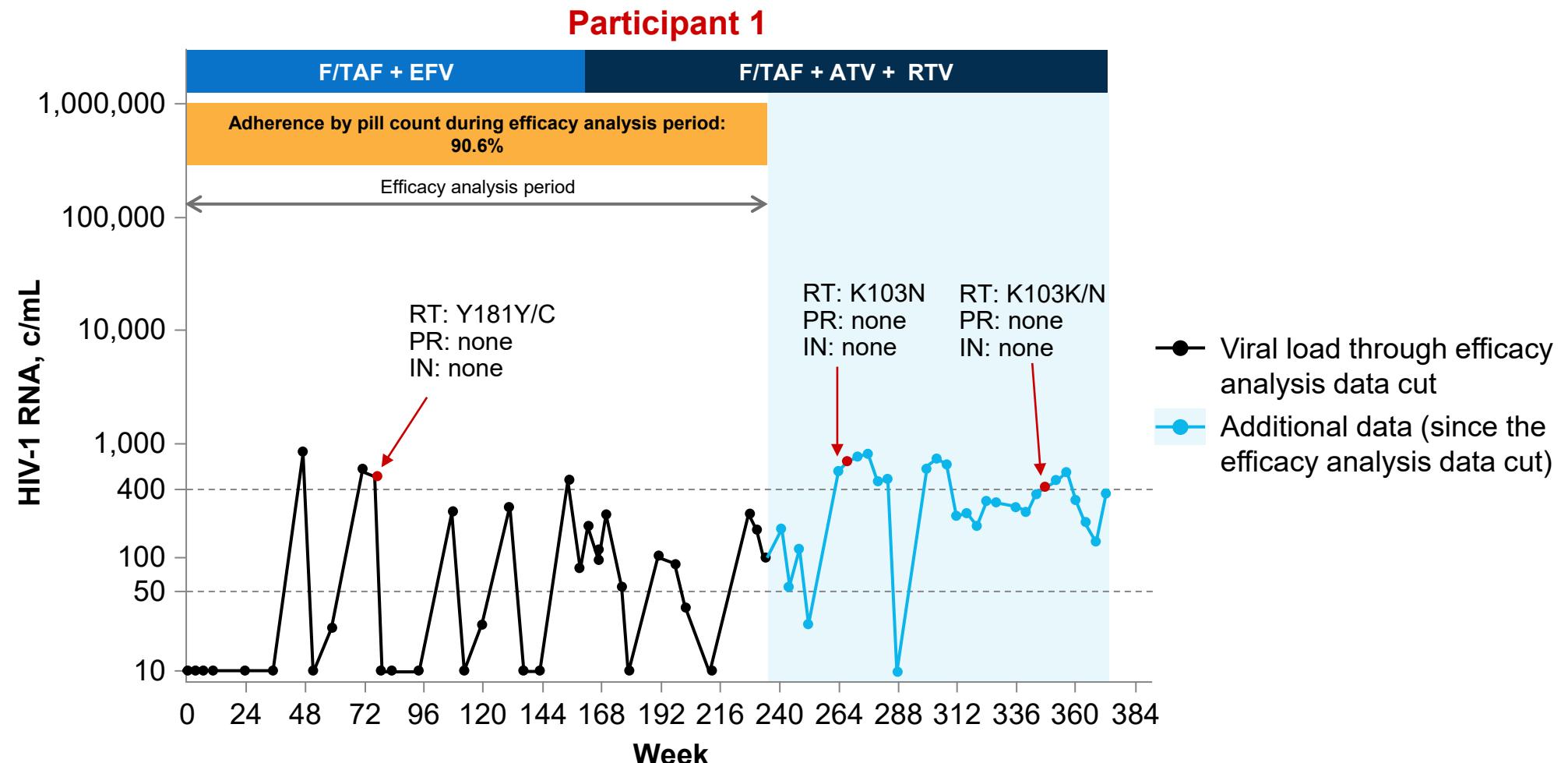
Participants with BL data, n/N (%)	F/TAF pooled N = 341	B/F/TAF (VS) n = 122	E/C/F/TAF (TN) n = 50	E/C/F/TAF (VS) n = 129	F/TAF + 3rd agent (VS) n = 40
No BL resistance*	105/152 (69)	56/95 (59)	45/50 (90)	4/7 (57)	ND
Any BL resistance*	47/152 (31)	39/95 (41)	5/50 (10)	3/7 (43)	ND
NRTI resistance	21/152 (14)	17/95 (18)	1/50 (2)	3/7 (43)	ND
M184V/I	13/152 (9)	11/95 (12)	0	2/7 (29)	—
Any TAM	14/152 (9)	12/95 (13)	1/50 (2)	1/7 (14)	—
K65R	1/152 (1)†	1/95 (1)†	0	0	—
NNRTI resistance	32/152 (21)	28/95 (59)	3/50 (6)	1/7 (14)	ND
K103N	8/152 (5)	6/95 (6)	2/50 (4)	0	—
PI resistance	11/152 (7)	11/95 (11)	0	2/7 (29)	ND
INSTI resistance	7/141 (5)	6/92 (7)	1/49 (2)	ND	ND
E92G	2/141 (1)	2/92 (2)	0	—	—
T97A	2/141 (1)	2/92 (2)	0	—	—
Y143C	1/141 (1)	1/92 (1)	0	—	—
R263K	2/141 (1)	1/92 (1)	1/49 (2)	—	—

*Participants without IN data are imputed as having no INSTI resistance substitution

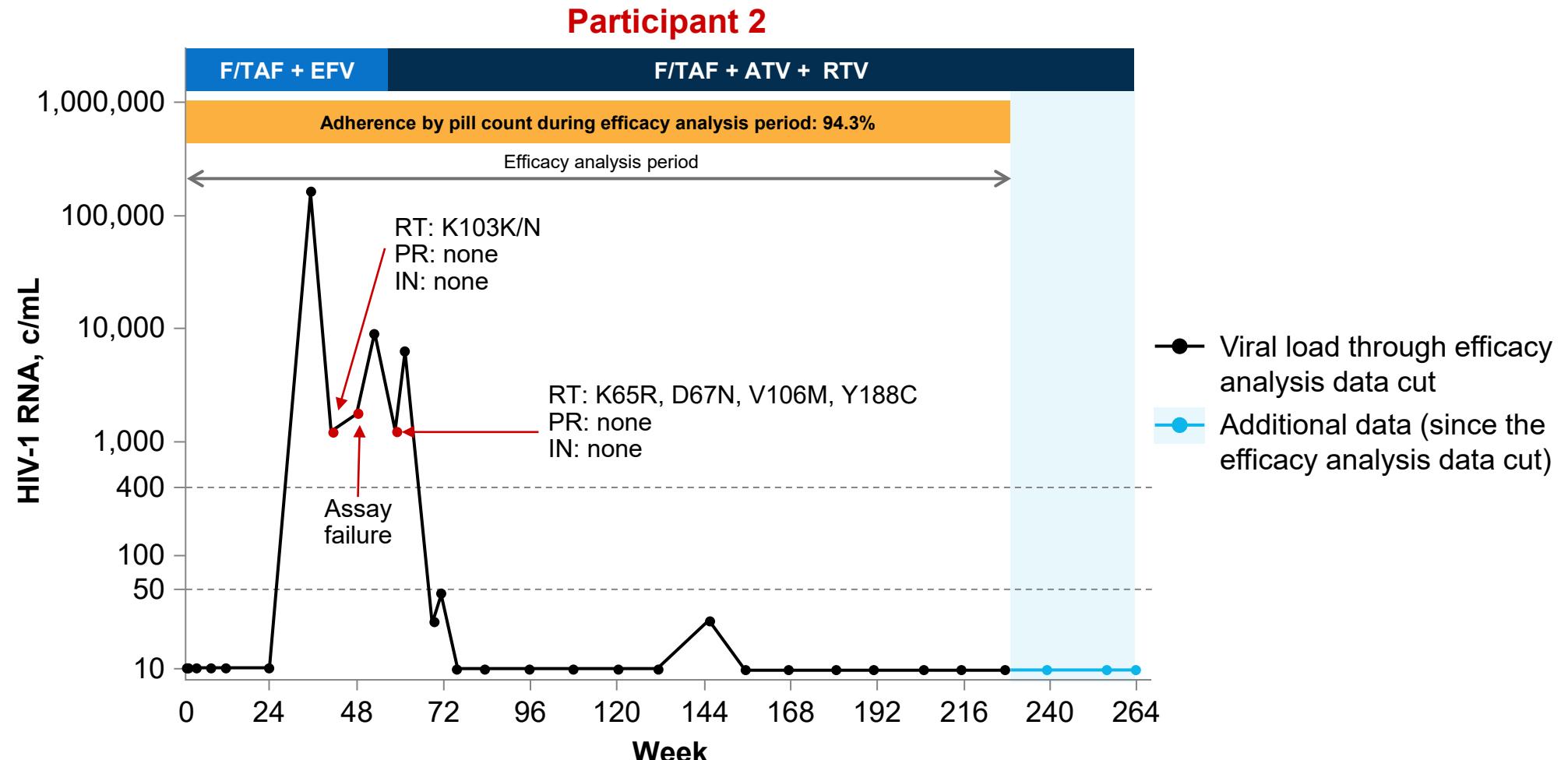
†Participant with K65R maintained virologic suppression through 223 weeks of B/F/TAF treatment

B/F/TAF, bictegravir/emtricitabine/tenofovir alafenamide; BL, baseline; E/C/F/TAF, elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide; F/TAF, emtricitabine/tenofovir alafenamide; IN, integrase; INSTI, integrase strand transfer inhibitor; ND, no data; NNRTI, non-nucleoside reverse transcriptase inhibitor; NRTI, nucleos(t)ide reverse transcriptase inhibitor; PI, protease inhibitor; TAM, thymidine analog mutation; TN, treatment naïve; VS, virologically suppressed

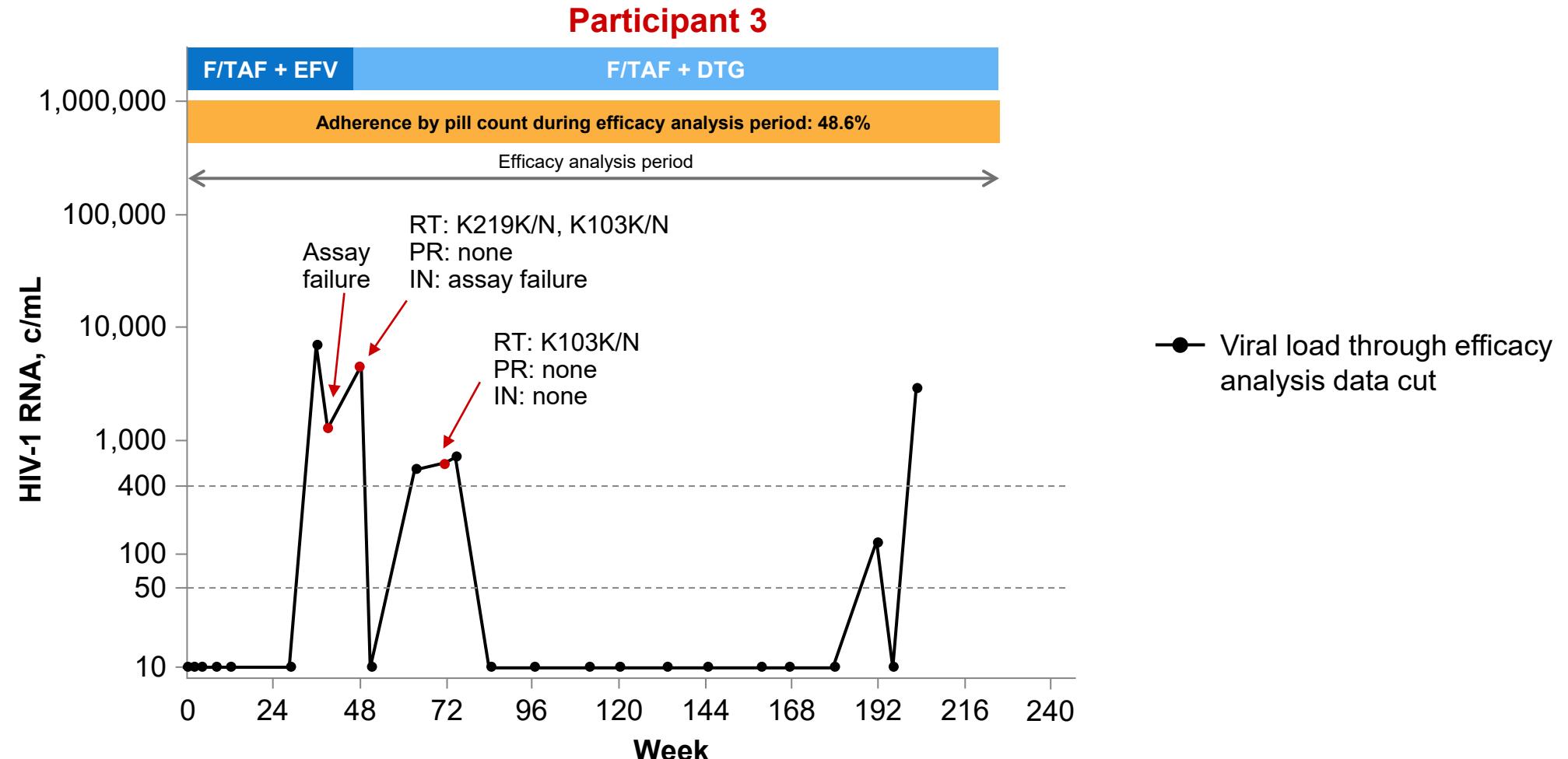
Results: Virologic Profiles of Presumed Emergent Resistance



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