Received: 18 June 2021 | Accepted: 22 September 2021

DOI: 10.1111/hiv.13186

SHORT COMMUNICATION

No overall impact on rate of weight gain with integrase inhibitor-containing regimens in antiretroviral-naïve adults

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Abstract

Objectives: Integrase strand transfer inhibitors (INSTIs) are commonplace in modern antiretroviral therapy (ART). Increased weight gain with their use is increasingly scrutinized. We evaluated weight changes in treatment-naïve adults with HIV-1 attending a UK centre who started regimens including raltegravir or dolutegravir.

Methods: A retrospective cohort study of adults prescribed an INSTI between January 2015 and March 2020 were categorized as having started an ART regimen containing raltegravir, dolutegravir, a protease inhibitor or a nonnucleoside reverse transcriptase inhibitor. Individuals with one or more weight measurement ≤ 5 years both pre- and post-ART initiation, who started a three-drug regimen with ≥ 6 months duration and achieved virological suppression (< 50 copies/mL) within 6 months were included. A random effects model with linear slope pre- and post-ART was used, adjusting for age, gender, ethnicity, ART regimen, backbone and year of initiation.

Results: The cohort included 390 adults; 88.7% were male, 66.4% were of white ethnicity, their median age was 40 years, there was a median of six weight measurements, 2.2 years from diagnosis to ART initiation, 2.9 years from ART to the last weight measurement, and weight and body mass index at initiation were 75 kg and 24.1 kg/m^2 respectively. Of these, 254 (65%) started an INSTI. The average pre-ART rate of weight gain was 0.44 kg/year [95% confidence interval (CI): 0.19–0.70], increasing to 0.88 kg/year (0.63–1.10, $p = 0.04$) after ART initiation. Our adjusted model found no evidence of an association between ART regimen and rate of weight gain.

Conclusions: Weight increased in the cohort both pre- and post-ART. We found no evidence of a higher rate of weight gain following ART initiation with an INSTI compared with other regimens.

KEYWORDS

antiretroviral therapy, integrase inhibitors, naïve, weight gain

INTRODUCTION

Integrase strand transfer inhibitors (INSTIs) are one of the recommended third agents for initial antiretroviral therapy (ART) regimens [1]. Raltegravir (RAL) and dolutegravir (DTG) remain the most widely used INSTIs in the UK [2]. 'Excessive' weight gain with INSTI-based regimens has been reported in a variety of settings, although the magnitude of the weight gain impact attributable to INSTIs, and what level of change should be considered significant remains unclear.

In ART-naïve cohorts, weight gain after ART initiation is seen as part of a 'return-to-health' phenomenon, particularly in individuals with advanced immunosuppression. This is a manifestation of reduced immune activation and the associated high metabolic turnover following viral suppression [3]. By contrast, more recent cohorts are typically started on treatment earlier, and therefore weight change may be more reflective of an effect of the ART regimen and/or obesogenic environments that similarly influence the general population.

We conducted a retrospective analysis of an adult, ART-naïve HIV-1 cohort in London, UK, to evaluate the rate of weight change after starting a RAL- or DTG-containing regimen relative to other regimens.

METHODS

Data were extracted from clinic records of people living with HIV (PLWH) who were prescribed INSTI-containing regimens between January 2015 and March 2020. The inclusion criteria were: ART-naïve; started a three-drug regimen containing DTG or RAL; initial regimen duration ≥ 6 months; evidence of viral load (VL) suppression (< 50 copies/mL) within 6 months of ART initiation; one or more weight measurement ≤ 5 years before (or at) ART initiation; and one or more weight measurement ≤ 5 years post-ART initiation. If consecutive VL 'blips' (> 50, < 200 copies/mL) or viral failure (>200 copies/mL) occurred after suppression, only weights before that point were included. There were no restrictions on year of ART initiation.

The analysis was restricted to individuals with dual nucleos(t)ide reverse transcriptase inhibitor (NRTI) backbones comprising abacavir/lamivudine (ABC/3TC) or tenofovir disoproxil fumarate/emtricitabine (TDF/FTC). Tenofovir alafenamide fumarate (TAF) was excluded because of low use. Weight measurements while on ART were only included for participants remaining on their initial ART regimen.

The comparator group were the identified ART-experienced individuals who previously started a non-INSTI-containing initial regimen; all other inclusion criteria were the same. Weight/height measurements were performed in outpatient clinics; devices were presumed to have undergone regular calibration in compliance with local standard operating procedures. Pregnant women and individuals with reported adherence issues or treatment interruptions were completely excluded from the analysis. Elvitegravir, bictegravir and cabotegravir were excluded because use as initial therapy was limited during the study period. Previous HIV pre-exposure prophylaxis (PrEP) use was not an exclusion criterion.

Statistical analysis

A random-effects model with linear slope before and after ART initiation was used to analyse weight change. Person-level correlated random effect terms were incorporated for intercept and pre- and post-ART slopes. The model parameters were defined in terms of the average weight at ART initiation, annual rate of weight gain pre-ART, and change in rate of weight gain post-ART. This approach was replicated for body mass index (BMI). Categorical weight change (e.g. 5% gain) was not adopted because of the heterogeneity when weight measurements were recorded.

Associations with age, gender, ethnicity (black African or other non-white/unknown vs. white), baseline CD4, baseline VL, initial ART regimen and year of ART initiation were evaluated. Control ART regimens were grouped into those containing a protease inhibitor (PI) and those containing a nonnucleoside reverse transcriptase inhibitor (NNRTI). Associations between continuous variables at ART initiation and weight/BMI trajectory characteristics were modelled using four-knot natural cubic splines (2.5^{th}, 33^{rd}, 66^{th} and 97.5^{th} centiles). A sensitivity analysis was conducted with additional adjustment of each model term for comorbidities of depression, renal impairment, high cholesterol/lipids, hypertension and non-alcoholic fatty liver disease (NAFLD). Models were fitted using the 'nlme' package [4] in R [5].

RESULTS

Of 682 identified ART-naïve individuals, 492 were started on regimens meeting the inclusion criteria; of these, 486 received this regimen for ≥ 6 months. In 424/486 participants, an undetectable VL was observed ≤ 6 months after ART initiation. A further 25 were excluded because pre- and post-ART weight measurements were outside the specified time-frame; nine were excluded due to no baseline HIV-1 VL and CD4 T-cell data. The final sample for

analysis included 390 individuals with 926 pre-ART and 1415 post-ART weights. The median [interquartile range (IQR)] number of weight measurements was 2 (1–3) pre-ART and 3 (2–5) post-ART. There was a median (IQR) of 2.2 (0.3–4.7) years from HIV-1 diagnosis to ART initiation, and 2.9 (1.3–4.1) years between ART initiation and last weight.

The cohort was predominantly male (n = 346, 89%), of white ethnicity (n = 259, 66%), Centres for Disease Control category A (n = 345, 89%), with a median (IQR) age at ART initiation of 40 years (34–46), and a median pre-ART CD4 count and VL of 390 (290–528) cells/μL and 4.5 (3.9–5.1) $\log_{10}$copies/mL, respectively. The median (IQR) weight and BMI at ART initiation were 75.0 (68.0–83.0) kg and 24.1 (22.2–26.8) kg/m^2, respectively. Of the 254 (65%) starting an INSTI, 196 (77%) started RAL and 58 (23%) DTG; 122 (48%) started TDF/FTC and 132 (52%) started ABC/3TC. None had received PrEP. Additional cohort characteristics are presented in Table S1.

The unadjusted mixed-effects model showed higher weights at initiation in the INSTI groups [NNRTI, 75.5 kg (95% CI: 73.2–77.7); PI, 73.9 kg (70.1– 77.7); DTG, 77.1 kg (74.0–80.1); RAL, 78.9 kg (76.7–81.0)] and an average pre-ART rate of weight gain of 0.44 kg/year (0.19–0.70) with an increase to 0.88 kg/year (0.63–1.10, $p = 0.04$) after ART initiation (Table S2). The adjusted model spline plots show trends of greater weight loss per year prior to ART initiation with lower CD4, higher baseline VL and higher age (Figure 1b,f,j; predicted average rates of change are shown for CD4, while differences in the predicted average rate of change relative to a reference value are shown for age and VL). Characteristics associated with weight loss prior to ART initiation were associated with a greater relative change in the rate of weight loss/gain at ART initiation (Figure 1d,h,l), with low CD4 and high VL at baseline associated with the larger absolute rates of weight gain on ART (Figure 1c,g).

Overall, when adjusting for the characteristics in Table 1, individuals had a higher rate of weight gain pre-ART for all other regimens compared with those starting an NNRTI, although not to a significant degree. The change in rate of weight gain after starting ART is positive where the rate of gain increased and negative where it decreased (e.g. black Africans, rate increased by 0.49 kg/year from a loss of −0.3 kg/year to a gain of 0.46 kg/year). This did not significantly differ by ART regimen relative to NNRTI [RAL, –0.76 kg/year (−2.36–0.84, $p = 0.36$); DTG, −1.01 kg/year (−2.97–0.95, $p = 0.32$); PI, −0.45 kg/year (−1.97–1.07, $p = 0.57$); NNRTI (reference); Table 1]. Similar findings were seen for BMI (Table 1, Figure S1). There was some evidence that individuals starting ART more recently had higher baseline weights [2007–2010, −0.82kg (−6.82–5.18, $p = 0.79$); 2011–2013, −0.13 kg (−5.29–5.03, $p = 0.96$); 2014–2016 (reference); 2017–2020, +2.57 kg (−0.99–6.13, $p = 0.16$)].

Further adjustment for patient comorbidities showed an increased rate of weight gain post-ART in those with depression (+1.29 kg/year, 95% CI: 0.32–2.26, $p = 0.01$) and greater weight at ART initiation for those with hypertension (+8.07 kg, 95% CI: 3.24–12.91, $p = 0.001$) and NAFLD (+9.48 kg, 95% CI: 5.04–13.91, $p < 0.0001$), without any other statistically significant findings. This did not affect the overall conclusions from the analyses regarding ART regimens.

DISCUSSION

Our real-world cohort was on average gaining weight both pre- and post-ART in our unadjusted analysis. However, we did not observe a difference in the rate of weight or BMI gain after starting an ART regimen containing RAL or DTG compared with a PI- or NNRTI-based regimen. Our study is novel in that we incorporated weights before baseline to assess pre-ART trends. We analysed the rate at which weight is gained (or lost), not the absolute weight change. Our aim was to identify whether the inclusion of INSTIs within the ART regimen was associated with an acceleration in the rate of weight gain as opposed to quantifying absolute weight change.

Observations of absolute weight gain after starting INSTIs in other treatment-naïve cohorts and a pooled analysis of randomized studies have been reported [6,7], although contrasting findings have been reported in switch cohorts [8]. Although ART-naïve individuals may experience gains in absolute weight after starting ART, the cause is likely to be multifactorial. One hypothesis to explain the variability of effect could be that INSTIs sensitize PLWH to gaining weight, the melanocortin-4 receptor pathway being one proposed mechanism [9]. However, absolute gains are probably influenced by lifestyle [10], cultural attitudes to weight [11], the individual's perception of their weight [12], and host genetics/microbiome [13]. This may explain why weight gain is not a universal phenomenon for all individuals on ART and for all regimens [14]. Improved tolerability of modern regimens could also be a factor and is more easily identified when full adverse event data from studies are reported [7]. For individuals who undergo extremely rapid weight gain with ART, host/genetic factors, yet to be elucidated, could amplify or synergize such an effect.

Individuals with a more recent year of ART initiation were heavier at initiation. This probably reflects the fact that PLWH are now diagnosed and started on treatment earlier, with fewer patients presenting with advanced disease and the associated weight loss. However, it may also reflect the

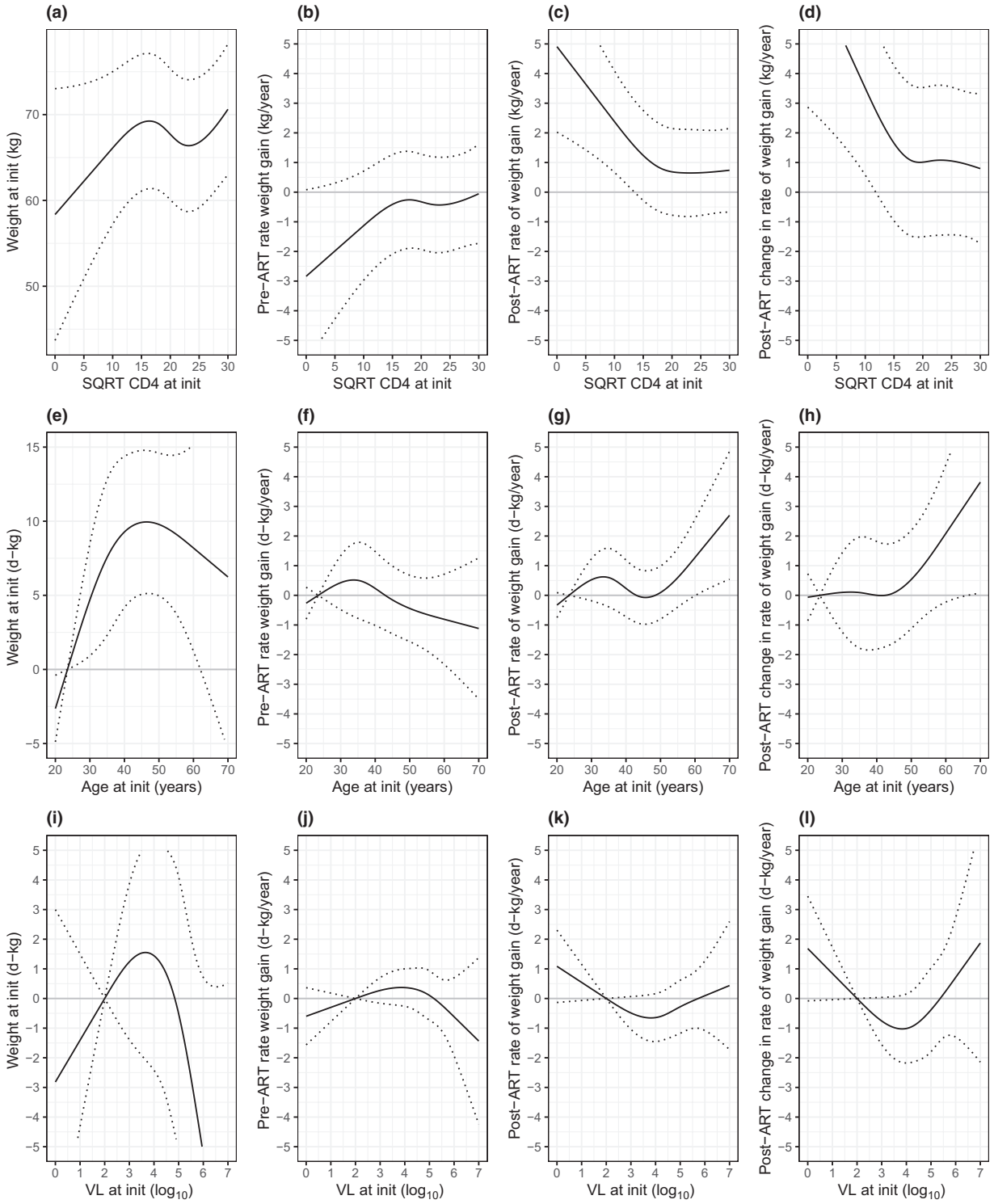

FIGURE 1 Estimates of average weight at antiretroviral therapy (ART) initiation (a), pre-ART rate of weight gain (b), post-ART rate of weight gain (c), and post-ART change in rate of weight gain in relation to CD4 at ART initiation (d). (e–l) These panels show the difference (delta) in the predicted weight/rate of change relative to (a)–(d), in relation to age at ART initiation for (e)–(h), and in relation to viral load at ART initiation for (i)–(l). These estimates are derived from models with adjustment for the demographic and treatment characteristics listed in Table 1. The plots relate to a white male individual starting a raltegravir-based regimen. Dotted lines show pointwise 95% confidence interval. Plot (c) represents a summation of the functions in (b) and (d); (g) is a summation of (f) and (h); and (k) is a summation of (j) and (l). d-, delta; init, initiation; SQRT, square root; VL, viral load.

TABLE 1 Associations between demographic/treatment characteristics and weight/body mass index (BMI) trajectories.

			Weight/BMI at ART initiation		Pre-ART rate of weight/BMI gain (per year)		Change in rate of weight/BMI gain after starting ART (per year)	
		n (%)	ΔEst. (95%CI)	*p*-value	ΔEst. (95% CI)	P-value	ΔEst. (95% CI)	*p*-value
Weight results (kg)								
Gender	Men	346 (89)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	Women	44 (11)	−7.04 (−11.87 to −2.21)	0.005	0.21 (−0.63–1.05)	0.63	0.37 (−1.07–1.82)	0.62
Ethnicity	White	259 (66)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	Black African	42 (11)	3.61 (−1.39–8.61)	0.16	−0.3 (−1.14–0.55)	0.49	0.49 (−0.99–1.97)	0.52
	Other non-white	89 (23)	1.72 (−1.53–4.96)	0.30	−0.46 (−1.04–0.12)	0.12	0.26 (−0.72–1.24)	0.60
ART regimen type	NNRTI	95 (24)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	PI	41 (11)	−0.41 (−5.33–4.51)	0.87	0.36 (−0.57–1.29)	0.46	−0.45 (−1.97–1.07)	0.57
	DTG	58 (15)	2.58 (−3.55–8.71)	0.41	0.69 (−0.51–1.88)	0.26	−1.01 (−2.97–0.95)	0.32
	RAL	196 (50)	3.23 (−1.7–8.15)	0.20	0.93 (−0.06–1.92)	0.07	−0.76 (−2.36–0.84)	0.36
NRTI backbone	TDF + FTC	233 (60)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	ABC + 3TC	157 (40)	−2.05 (−5.23–1.13)	0.21	−0.08 (−0.64–0.48)	0.78	0.32 (−0.64–1.28)	0.52
Year of ART initiation	2007–2010	49 (13)	−0.82 (−6.82–5.18)	0.79	0.41 (−0.7–1.51)	0.48	−0.8 (−2.68–1.07)	0.41
	2011–2013	63 (16)	−0.13 (−5.29–5.03)	0.96	0.45 (−0.63–1.54)	0.42	−0.87 (−2.57–0.84)	0.32
	2014–2016	195 (50)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	2017–2020	83 (21)	2.57 (−0.99–6.13)	0.16	0.72 (0.03–1.41)	0.04	−0.82 (−1.99–0.34)	0.17
BMI results (kg/m^2)								
Gender	Men	346 (89)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	Women	44 (11)	1.29 (−0.23–2.8)	0.10	0.16 (−0.13–0.45)	0.29	0 (−0.48–0.47)	0.99
Ethnicity	White	259 (66)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	Black African	42 (11)	2.54 (0.96–4.13)	0.002	−0.07 (−0.36–0.22)	0.65	0.07 (−0.42–0.57)	0.77
	Other non-white	89 (22)	1.38 (0.34–2.43)	0.01	−0.14 (−0.33–0.05)	0.17	0.1 (−0.22–0.42)	0.56
ART regimen type	NNRTI	95 (24)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	PI	41 (11)	−1.22 (−2.88–0.43)	0.15	0.08 (−0.26–0.41)	0.66	−0.09 (−0.62–0.44)	0.74
	DTG	58 (15)	0.34 (−1.6–2.29)	0.73	0.15 (−0.25–0.56)	0.47	−0.28 (−0.92–0.37)	0.41
	RAL	196 (50)	0.44 (−1.14–2.02)	0.59	0.22 (−0.13–0.56)	0.22	−0.17 (−0.7–0.36)	0.53
NRTI backbone	TDF + FTC	233 (60)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	ABC + 3TC	157 (40)	−0.84 (−1.87–0.18)	0.11	−0.01 (−0.21–0.18)	0.88	0.08 (−0.24–0.4)	0.64

(Continues)

TABLE 1 (Continued)

			Weight/BMI at ART initiation		Pre-ART rate of weight/BMI gain (per year)		Change in rate of weight/BMI gain after starting ART (per year)	
		n (%)	ΔEst. (95%CI)	*p*-value	ΔEst. (95% CI)	P-value	ΔEst. (95% CI)	*p*-value
Year of ART initiation	2007–2010	49 (13)	0.18 (−1.79–2.15)	0.86	0.15 (−0.23–0.54)	0.45	−0.44 (−1.08–0.2)	0.18
	2011–2013	63 (16)	0.07 (−1.63–1.76)	0.94	0.23 (−0.17–0.63)	0.27	−0.49 (−1.08–0.1)	0.11
	2014–2016	195 (50)	0 [Reference]	-	0 [Reference]	-	0 [Reference]	-
	2017–2020	83 (21)	0.37 (−0.73–1.47)	0.52	0.23 (0.01–0.46)	0.046	−0.26 (−0.64–0.12)	0.18

Note: Associations between demographic/treatment characteristics and both average weight and BMI trajectories during the 5 years before and after ART initiation. Adjusted for age, CD4 and VL at switch and the other variables listed in the table. Estimates are expressed relative (ΔEst.) to the average value at any given age (Figure 1).

Abbreviations: 3TC, lamivudine; ABC, abacavir; ART, antiretroviral therapy; CI, confidence interval; DTG, dolutegravir; FTC, emtricitabine; NNRTI, nonnucleoside reverse transcriptase inhibitor; PI, protease inhibitor; RAL, raltegravir; TDF, tenofovir disoproxil fumarate.

increasingly obesogenic environment that equally affects the general population; 64% of adults in England are overweight or obese (36% and 28%, respectively) [15], with a similar prevalence observed in comparable countries [16]. An increased rate of weight gain was observed in those starting treatment with lower CD4, higher VL and older age, probably associated with a return to health. Both findings have been observed in other HIV cohorts [6].

Our study is limited by being a relatively small, single-centre cohort comprised predominantly of white males. This restricts the generalisability of our findings to other groups taking INSTI-containing regimens, particularly black females who are thought to be susceptible to weight gain. Similarly, TAF was excluded due to small numbers ($n = 4$), precluding assessment of the potential effect of TAF as noted in other studies [7,17]. We were also unable to include other INSTIs (cabotegravir, elvitegravir and bictegravir) due to low use.

Our study will not have captured extremely rapid changes in weight that may have occurred during the 6 months after ART initiation if this caused a regimen switch. However, only a small number of individuals were excluded on this basis ($n = 6$) and our primary focus was the rate of weight change across the whole cohort following a longer period of INSTI exposure. This approach allowed for sufficient time to determine if any immediate acceleration of weight gain is maintained.

CONCLUSIONS

Weight change after ART initiation is probably a complex, multifactorial process. These data suggest that RAL- and DTG-containing regimens in treatment-naïve individuals are not associated with an acceleration in weight gain compared with other ART regimens, beyond that expected following viral suppression, within the limitations specified. Research incorporating assessment of general weight gain factors such as diet and exercise would be beneficial, mitigating the under-reporting and recording bias with diet recall, although this is often logistically challenging [18]. Comorbidities also need to be considered; depression was associated with increased rate of weight gain, a condition with high prevalence [19] where both the disease and the treatment can affect weight [20].

Caution should be shown in attributing weight gain as a causal effect of INSTI use in the absence of adequate follow-up and monitoring. More data are needed to evaluate individuals switching away from INSTIs because of weight gain. This should include capturing obesogenic factors; whether the weight gain continues, plateaus or reverses; weight distribution, and whether any clinical or metabolic sequelae manifest.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the assistance of Stephen Ogunnaike with the data extraction.

CONFLICT OF INTEREST

LW received speaker fees, advisory fees and support for conference attendance from Gilead Sciences, MSD, ViiV, Janssen, Mylan and Cipla. LW has also been involved in research sponsored by Gilead Sciences and Janssen. For the remaining authors none were declared.

AUTHOR CONTRIBUTIONS

JEB, SLP, DD and OS developed the research question and study. JEB completed electronic clinical record reviews and data collection, data cleaning and drafted the initial manuscript. OS performed the statistical analyses and contributed to the initial draft manuscript of the article. All authors were involved in the final interpretation of the results and finalized the manuscript.

DATA AVAILABILITY STATEMENT

Research data are not shared.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Burns JE, Stirrup O, Waters L, Dunn D, Gilson R, Pett SL. No overall impact on rate of weight gain with integrase inhibitor-containing regimens in antiretroviral-naïve adults. *HIV Med*. 2021;00:1–7. doi:10.1111/hiv.13186