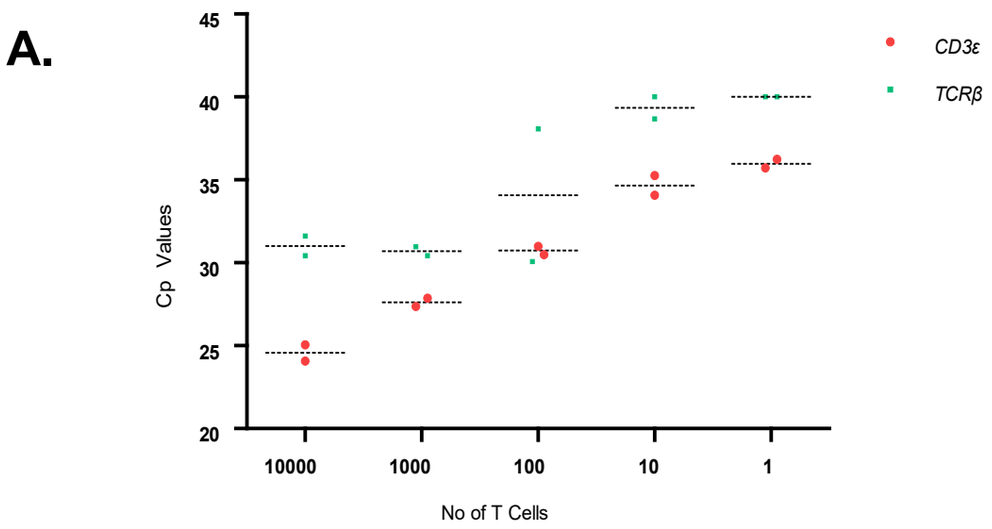
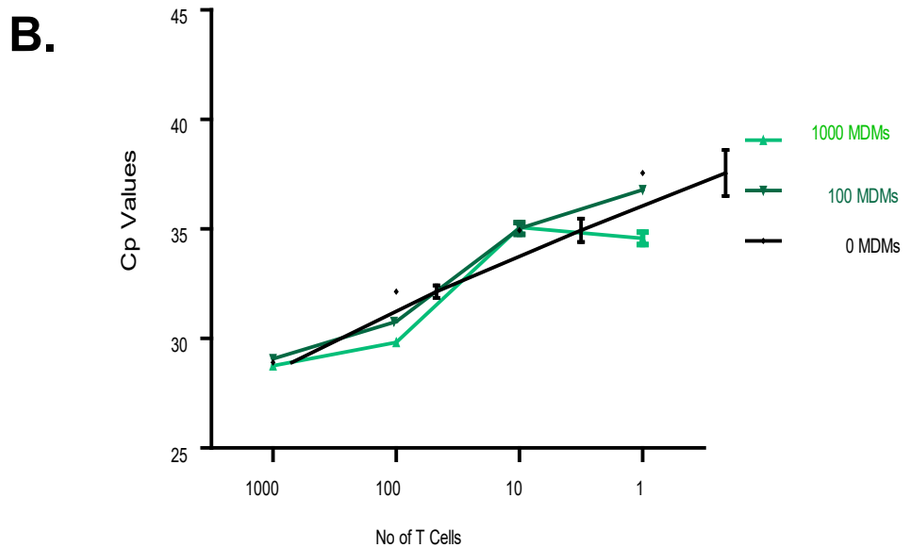
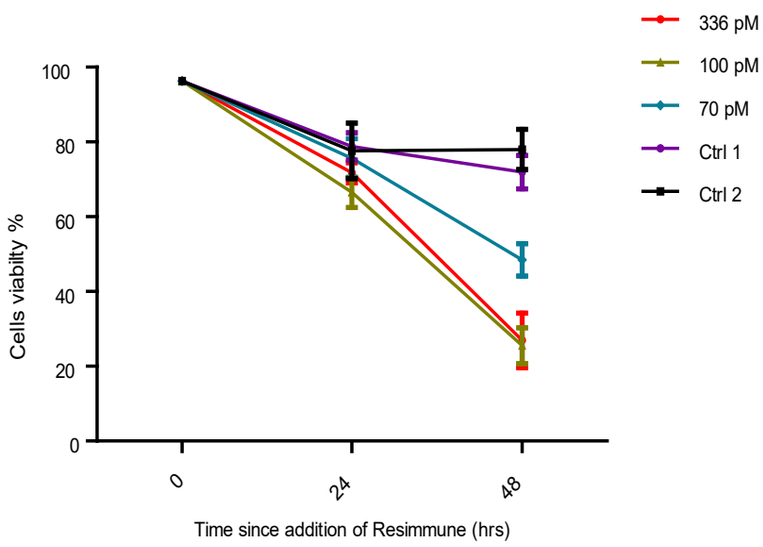
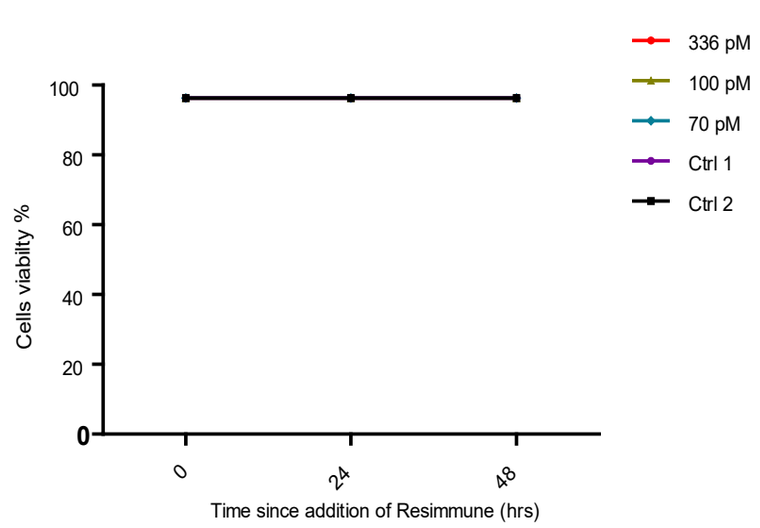
SFigure1.



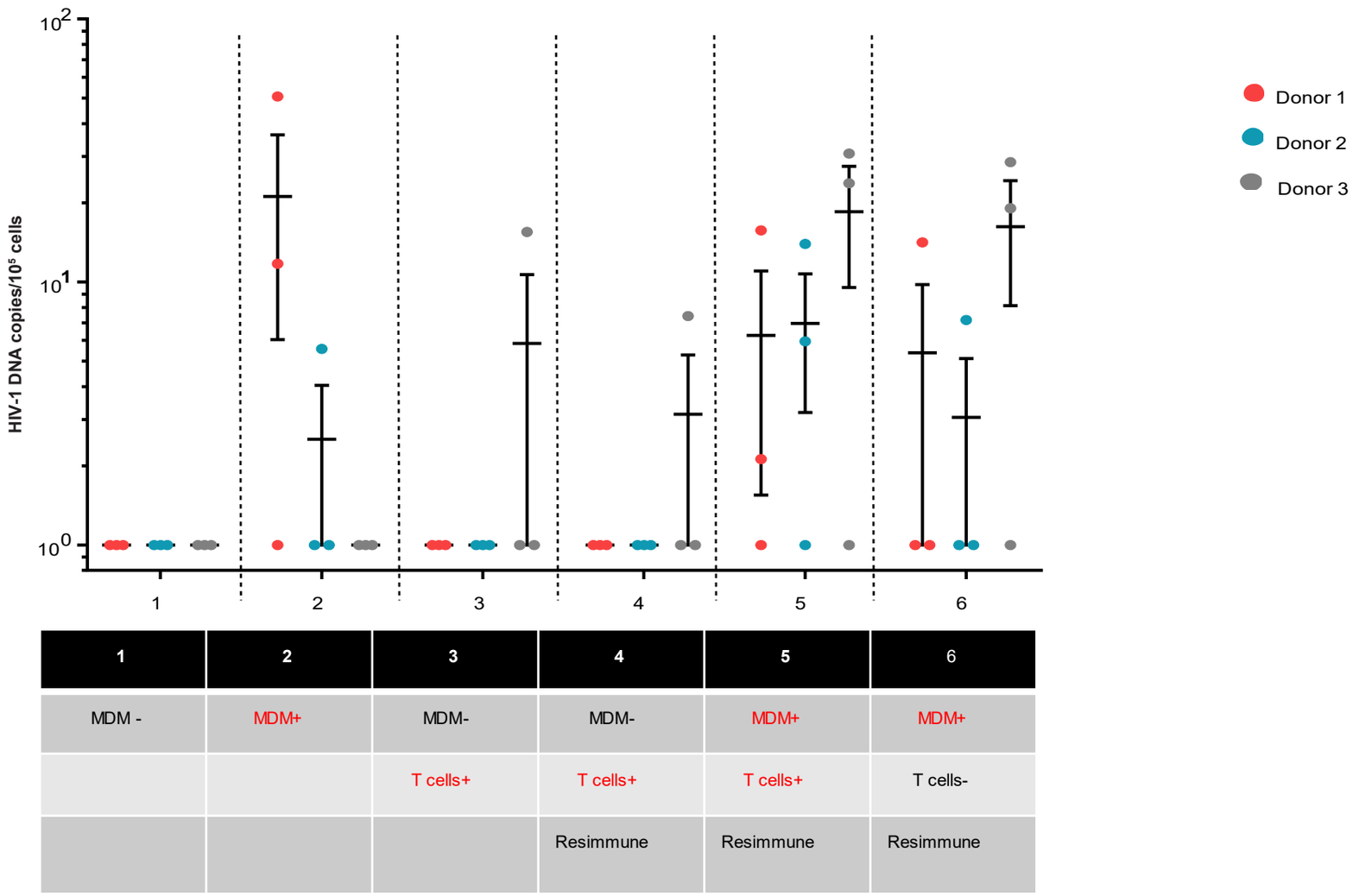






SFig. 1. T cells can be tracked by quantification of *CD3ε* mRNA qPCR and specifically depleted. (A) *CD3ε* mRNA qPCR is an optimal assay for quantifying T cells in low numbers. Two qPCR assays were evaluated for their sensitivity to detect total T cells serially diluted to single cells that were quantified in bulk prior to dilution using an automated cell counter (see methods). The first, an assay for *CD3ε* mRNA, was sensitive to single cells. The second, an assay for recombined *TCRβ*, was sensitive to single cells, but did not appear to have linear characteristics and registered at higher Cp values. Thereafter, we used the *CD3ε* mRNA qPCR assay to detect contaminating total T cells. (B) Detection of T cells contaminating macrophages was feasible using qPCR for *CD3ε* mRNA in mixed populations of cells. Monocytes were isolated from LeukoPaks using MACS column-based CD14+ selection and cultured in vitro in macrophage differentiation media for 7 days. The resulting monocyte derived macrophage (MDMs) populations were serially diluted, and unactivated T cells were mixed in decreasing concentrations to test the sensitivity of qPCR for the detection of contaminating T cells. Single CD4+ T cells were detectable by qPCR in the absence of MDMs. These data show that i) the level of T cell contamination in MDMs using CD14+ selection is between 1:100 – 1:1000 and ii) the level of detection of T cell contamination in MDMs that can be detected is between 1:100 and 1:1000. (C) Resimmune™ successfully depletes T cells in vitro. Based on the reported Resimmune IC50 of 0.1 pM for resting T cells, viability was measured in CD3+ cells isolated from healthy individuals and cultured in serial dilutions of Resimmune™, a high-affinity anti-CD3-diphtheria toxin fusion protein. Negative controls included CD3+ cells in media alone (Ctrl 1, purple line) and CD3+ T cells grown in media and drug buffer without the addition of Resimmune™ (Ctrl 2, black line). Viability was measured by Acridine Orange/Propidium iodide live/dead staining. At maximal concentrations of Resimmune™, CD3+ T cell viability declined by 80%. (D) Human monocyte derived macrophage (MDMs) experienced no change in viability when grown in the presence of the same concentrations of Resimmune™ as was used for T cells.

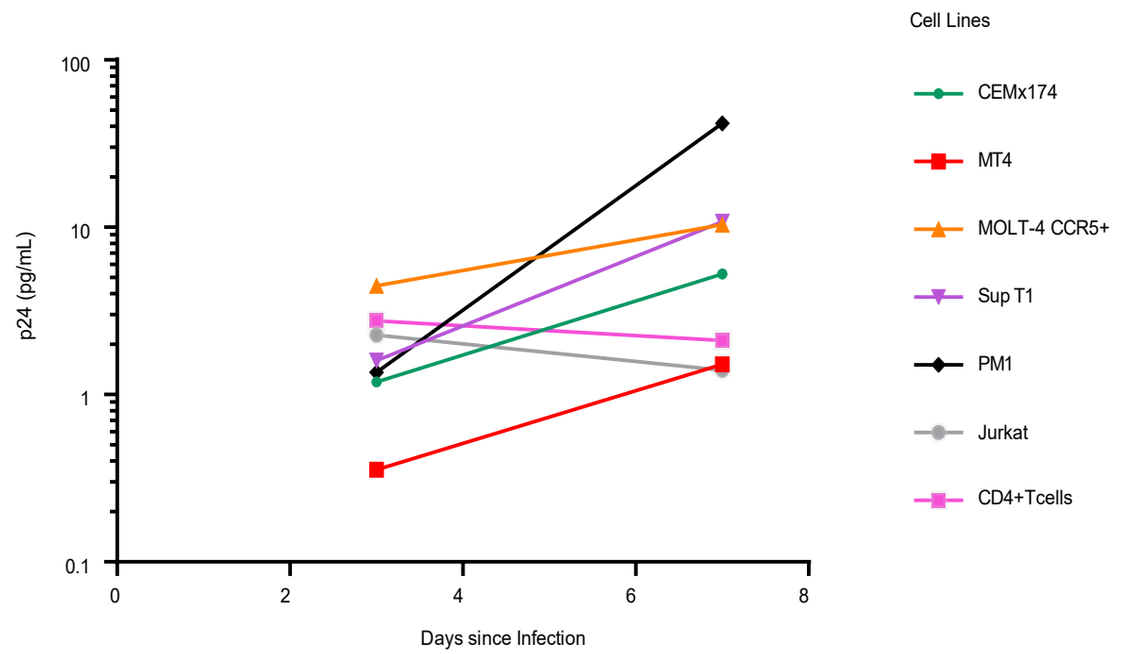
SFigure 2.



1	2	3	4	5	6
MDM -	MDM+	MDM-	MDM-	MDM+	MDM+
		T cells+	T cells+	T cells+	T cells-
			Resimmune	Resimmune	Resimmune

SFig. 2. HIV-1 infection of macrophages can be quantified in culture despite the presence of CD4+ T cells. We used different co-culturing conditions of MDMs and purified CD4+ T cells to model LM VOA, testing whether T cell contamination or phagocytosis might confound our results. After maturation, MDMs were either maintained in culture alone or in co-culture with CD4+ T cells from the same donors in a ratio of 100 MDM: 1 CD4+ T cell (total MDMs=10^5 cells/well), mimicking the lower limit of detection of contaminating T cells in LM cultures that we derived from patients. MDMs and CD4+ T cells were either pre-infected with BaL HIV-1 or uninfected prior to co-culture. In some CD4+ T cell wells, Resimmune™ was added for 48 hours to mimic the culture conditions that we employed in the LM VOA. After pre-infection, MDM and CD4+ T cells were co-cultured together in the presence of antiretrovirals (FTC, TDV, RTG at concentrations that were sufficient to inhibit infection,see methods) to prevent transmission of infectious HIV-1 between cell types. After 30 days of co-culture, all cells were lysed and total DNA was extracted. Results that are depicted are HIV-1 proviral DNA levels that were measured in cell lysates using qPCR after 30 days. Each condition was performed in triplicate wells for every donor and data represent mean ± SEM.The table below the figure indicates the culture or co-culture conditions for each arm of the experiment. Red text in the table is used to underscore whether MDMs were pre-infected, CD4+ T cells were pre-infected, or both cell types were pre-infected prior to co-culture. MDMs from all three donors appear to support HIV-1 infection in some conditions, although only MDMs from donors 1 and 2 support infection on their own. For donors 1 and 2, the addition of HIV-1 infected CD4+ T cells did not appear to alter the measurement of HIV-1 proviral DNA in MDM:CD4+ T cell co-cultures >30 days after co-culture (conditions 3 and 4). For donor 3 there was enhancement of the detection of MDM infection that was independent of whether CD4+ T cells were infected (conditions 5 and 6). MDMs from all donors showed infection when co-cultured with uninfected CD4+ T cells that were Resimmune™ treated (condition 6). *CD3ε* mRNA was detectable in all co-cultures (conditions 4-6) after >30 days, except for donor 1 condition 4. The proviral DNA qPCR assay had a lower level of quantification of 10 cp/10^6 cells: values for levels that were positive but below this threshold are indicated for completeness, although there is greater uncertainty on the amount of proviral DNA in those conditions compared to values above this threshold.

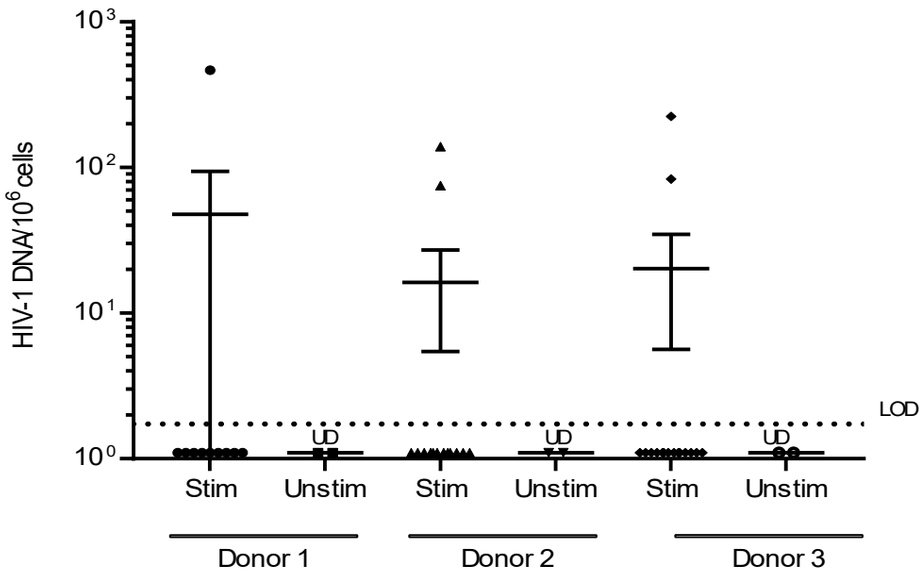
SFigure 3.



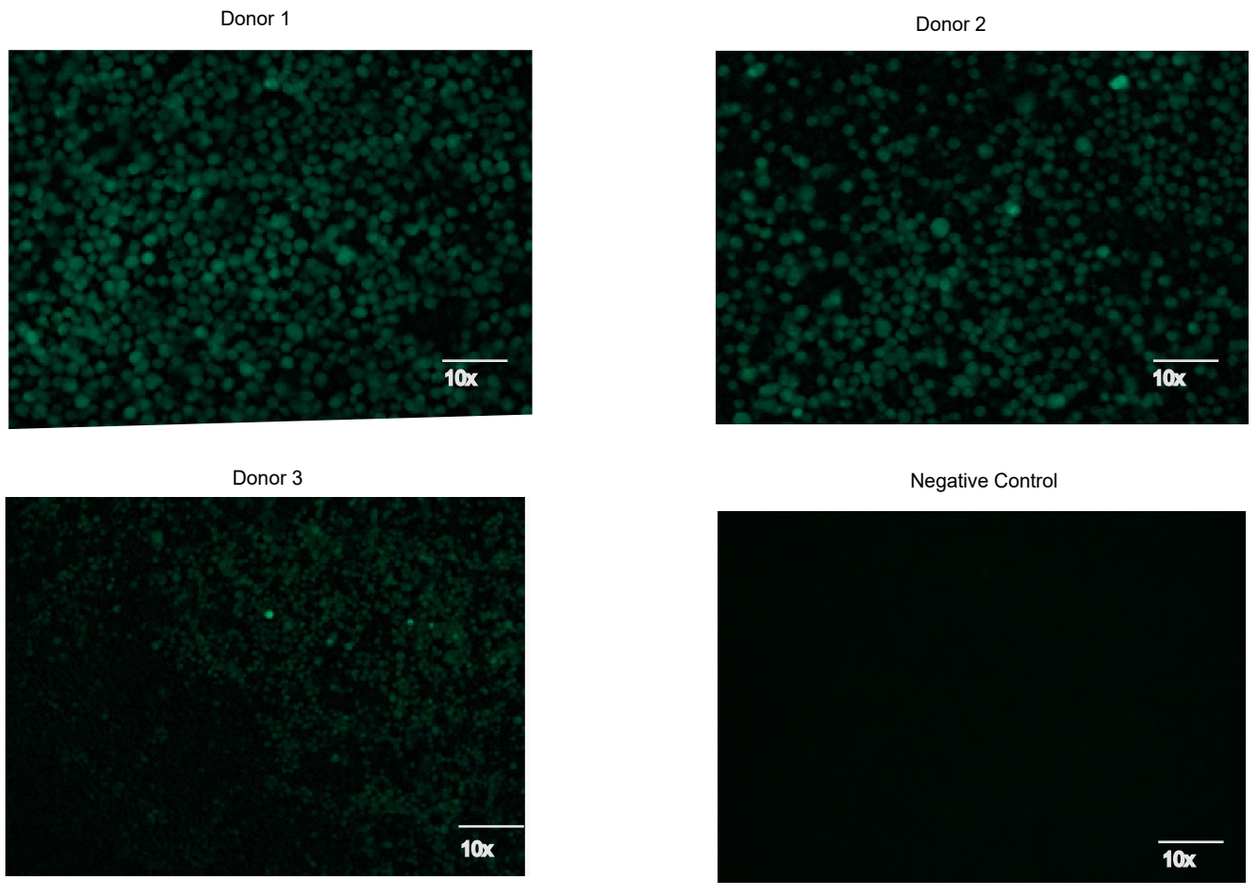
SFig. 3. CEMx174 that are infected with HIV-1 BaL can propagate infection. Activated CD4+ T cells and six cells lines were infected with 1 pg/mL of HIV-1BaL and p24 levels were monitored on days 3 and 7 following infection. Except activated CD4+ T cells and Jurkat cells, all other cells were observed to propagate infection. MOLT-4 CCR5+ cells were cultured in selective media containing G148.

SFigure 4.

A.

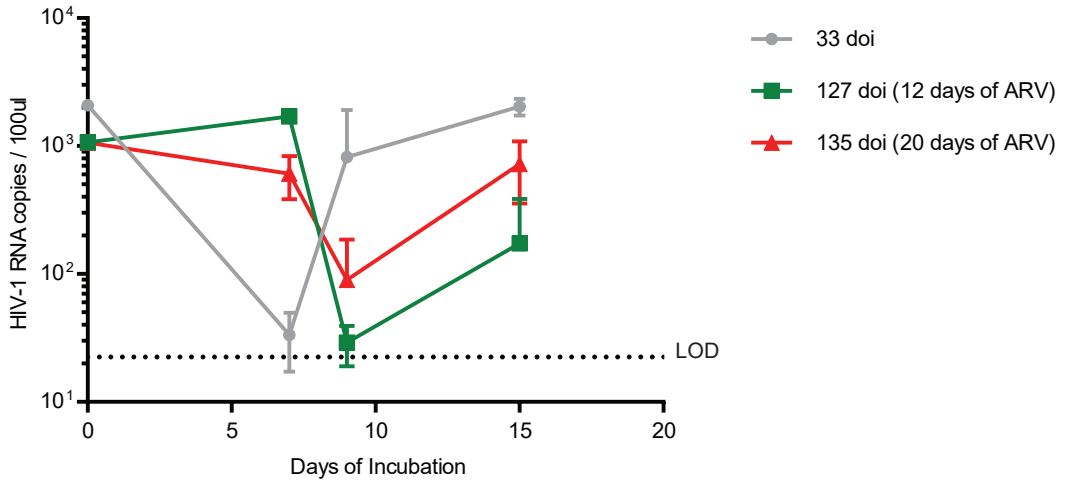


B.



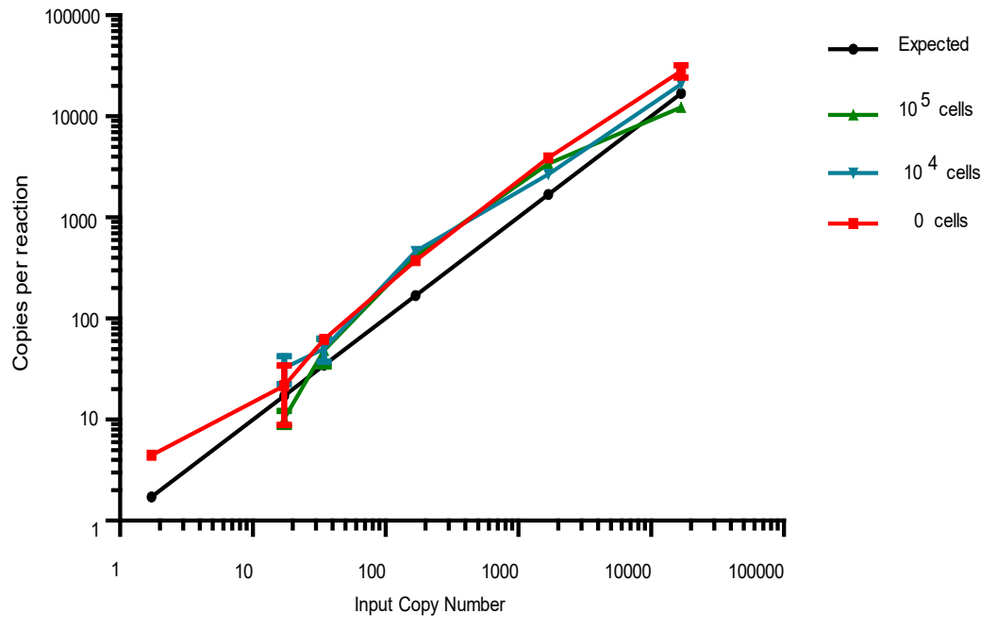
SFig. 4. HIV-1 infected liver macrophages that are stimulated with IFNG and rTat can transmit infectious virus to CEMx174 cells. Liver macrophages from three HIV-1 uninfected persons were infected in vitro with GFP-containing HIV-1. After viral RNA levels in the supernatants declined to levels that were close to or lower than the level of detection for the assay, LM were stimulated with IFNG and rTat over the course of a week. After stimulation, filtered LM supernatants were transferred to uninfected CEMx174 target cells and incubated for 14 days. Supernatants from unstimulated LM were also transferred to separate CEMx174 cells as a negative control. (A) Proviral DNA was detected in CEMx174 target cells following lysis on day 14 after inoculation with filtered LM supernatants. HIV-1 DNA was undetectable in target cells that were inoculated with supernatants from unstimulated LM from the corresponding donors. Error bars indicate mean ± SEM. (B) GFP+ target cells were observed after inoculation of filtered supernatants from all 3 donor LM on day 14. CEMx174 cells infected with supernatants of corresponding unstimulated donor macrophages (negative control) were negative for GFP+ cells. UD: undetectable. LOD: limit of detection

SFigure.5.



SFigure 5. Liver macrophages that are HIV-1 infected in vitro can propagate infection in CEMx174 cells. Supernatants from liver macrophages that were infected with a GFP-containing R5-tropic HIV-1 virus were transferred at the indicated times to uninfected CEMx174 reporter cells. HIV-1 RNA levels were measured in supernatants of CEMx174 reporter cells over the course of 15 days of incubation to assess whether infected liver macrophages released infectious virus that has the capacity to propagate. CEMx174 cells inoculated with supernatants that were transferred from LM all showed evidence of propagation, irrespective of whether they derived from LM that were recently infected (gray line, 33 doi) or had sustained infection ([blue line (127 doi, after 12 days of ARV) and green line (135 doi, after 20 days of ARV)]. All CEMx174 wells were found to be GFP+ on day 15 after transfer of LM supernatants, LOD: Limit of detection. Error bars indicate mean ± SD.

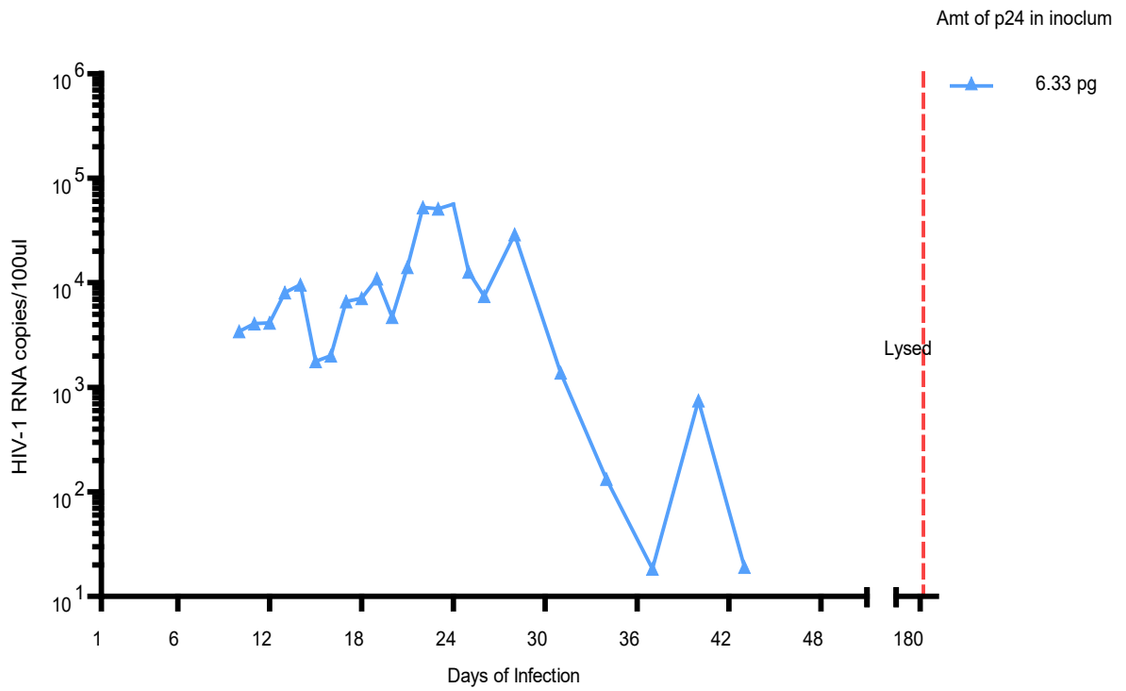
SFigure 6.



		0 cells	10^5 cells	10^4 cells
R square	1.000	0.9864	0.9690	0.9986

SFig. 6. Sensitive detection of HIV-1 proviral DNA amidst uninfected MDM. HIV-1 proviral DNA qPCR was evaluated using the 8E5 cell line that contains a single copy of HIV-1 proviral DNA in each cell. The black line indicates the expected copy number per reaction based on the input number of 8E5 cells (ATCC® CRL-8993™). qPCR was sensitive and accurate to detect a single copy of 8E5 cells that was serially diluted without the addition of MDM (red line). The addition of 10^5 (green) and 10^4 (blue) MDM to the 8E5 cell line did not appreciably change the sensitivity or accuracy of the qPCR assay

SFigure 7.



SFig. 7. Viral kinetics of HIV-1 BaL strain following infection of primary human liver macrophages. Primary human liver macrophages from an HIV-1 uninfected donor were infected with the HIV-1 BaL strain (p24 conc. = 6.33pg). HIV-1 RNA was measured in supernatants at regular intervals by qPCR. Media was changed every 2-3 days during the course of the experiment. Despite decline in HIV-1 RNA in supernatant to undetectable levels by 50 doi, intracellular HIV-1 RNA (435 copies) was detectable upon cell lysis on 181 doi.

STable. 1. IUPM measured using quantitative viral outgrowth assay on circulating resting memory CD4+ T cells.

ID	Time post transplantation	Infectious Units per Million cells (IUPM)
LT01	2 weeks	1.65
	34 months	1.69
LT02	26 months	16.25

STable 2. Baseline characteristics of study subjects

ID	Date	Clinical setting prior to obtaining liver tissue	Reason for transplant/ Cause of Death	Liver fibrosis	CD4+ T cell count	Interval from removal to processing	Coinfection		ART duration (months)
							HCV	HBV	
LT01	July, 2013	Explant during transplantation	Decompensated liver disease due to HCV	cirrhosis	607 cells/uL (47%) (4 days preTx)	< 1 hour	+	-	140
LT02	March, 2014	Explant during transplantation	HCC	cirrhosis	116 cells/uL (11.4%) 5 months preTx	< 1 hour	+	-	8
N7	May, 2014	Post-mortem	HIV (listed on death certificate)	Cirrhosis, portal hypertension	53 cells/uL 20 days prior to tissue	3.5 hours	-	-	-
N9	April, 2015	Post-mortem	HIV/Cancer (listed on death certificate)	NA	NA	17 hours	-	+	NA
LT06	March, 2016	Explant during transplantation	Decompensated liver disease due to HCV	cirrhosis	1183 cells/uL (35.4%) 3 months preTx	< 1 hour	+	-	64
LT07	April, 2016	Explant during transplantation	Decompensated liver disease due to HBV	Cirrhosis	426 cells/uL (39.2%) 3 months preTx	< 1 hour	-	+	> 113

LT08	May, 2016	Explant during transplantation	Decompensated liver disease due to cryptogenic cirrhosis	Cirrhotic changes, ascites, portal hypertension, splenomegaly on MRI, not on biopsy report. No fibrosis staging on bx.	154 cells/uL (33.4%) 1 day preTx	< 1 hour	-	-	> 60
LT09	June, 2016	Explant during transplantation	Decompensated liver disease, prior liver tx with chronic allograft dysfunction from hepatic artery stenosis	cirrhosis	180 cells/uL (22%) 5 months preTx	< 1 hour	-	-	115
LT10	July, 2016	Explant during transplantation	HCC	Cirrhosis	155 cells/uL (14.3%) 4 months preTx	< 1 hour	-	+	15

Supplementary Information

Determining the decay half-life of infected liver macrophages (LMs) during ARV. Because media was changed at various times not all of which correspond to points at which the viral load was measured we need to account for effects of media changes when determining the half-life of infected LMs from the rate of decay of HIV-RNA. Further, because infected cells can die between media changes, we need to account for the precise dynamics of infected cells and virus during the intervals between media changes.

Viral and infected cell dynamics during ARV phase

Under the assumption that ARV prevents all de novo infection of LMs, the standard viral dynamics model from Perelson et al. (1) can be simplified to

$$\frac{dI}{dt} = -\delta I \qquad (1)$$

$$\frac{dV}{dt} = pI$$

where I and V represent the concentration of infected LMs and HIV, respectively, δ is the per capita death rate of infected cells and *p* the rate of production of virus per infected cell. For simplicity, we assume there is no loss of virus by degradation in the culture system. From model (1), we also have $\frac{dV}{dI} = -\frac{p}{\delta}$. On integrating this, we have $V(t) = V(0) - \frac{p}{\delta}[I(t) - I(0)]$. Thus, it is clear that $\Delta V \propto \Delta \mathrm{I}$.

Let $t_0, t_1, t_2 \ldots . t_i$, where $t_i,\ i = 1, 2, 3, \ldots .$, be the time points when media changes were done and caused V in the well after media changes (or, V_{MC}) to be very small compared to $V_{t_i^-}$, where $V_{t_i^-}$ is the concentration of HIV just before the media change. We assume that $V_{MC} \ll V_{t_i^-}$ in the analysis.

Similarly, let $T_0, T_1, T_2 \ldots . T_j$, where $T_j, j = 1, 2, 3, \ldots .,$ be the time points when viremia was measured with corresponding viral load denoted as V_{Tj}. Whenever viremia was measured, the supernatant was removed and thus V in the well was reduced to V_{MC}. We further assume that $V_{MC} \ll V_{Tj}$ in the analysis.

Case (1): Derivation of viral concentration at time t during the ARV phase with no media change

This is the simplest case. In this case, we assume that $t_i = T_i$ for all values of *i*. Additionally, V_{ti} represents the virus concentration at time point t_i. We are interested in determining the virus concentration at any time *t* between $t_0 = T_0$ and $t_1 = T_1$ given that no media change occurs in between.

From the first equation of the model (1), we have $I(t) = I_0 e^{-\delta(t-t_0)}$. Substituting *I*(*t*) into the second equation of model (1), and solving we have

$V(t) - V_{MC} = \frac{pI_0}{\delta}(1 - e^{-\delta(t-t_0)})$, for $t_0 < t \leq t_1$. Here, I_0 is the infected cell population at t_0.

Under the assumption $V_{MC} \ll V_{ti}$ for all values of *i*, we have $V(t) - V_{MC} \sim V(t)$. Therefore,

$$\log(V_{T1}) = \log(V_{t1}) = \log(pI_0) + \log(\frac{1 - e^{-\delta(t_1 - t_0)}}{\delta})$$

Assuming $\delta(t_1 - t_0) < 1$ and using a Taylor series expansion of the exponential term,

$$\log(V_{T1}) = \log(V_{t1}) = \log(pI_0) + \log(\frac{2\delta(t_1 - t_0) - \delta^2(t_1 - t_0)^2}{2\delta})$$

or

$$\log(V_{T1}) = \log(pI_0) + \log((t_1 - t_0)[1 - \frac{\delta(t_1 - t_0)}{2}])$$

or

$$\log(V_{T1}) = \log(pI_0) + \log(t_1 - t_0) + \log(1 - \frac{\delta(t_1 - t_0)}{2})$$

Using $\log(1 - x) = -x, for\ x < 1$, we have

$$\log(V_{T1}) - \log(t_1 - t_0) = \log(pI_0) - \frac{\delta(t_1 - t_0)}{2}$$

or

$$\log\left(\frac{V_{T1}}{t_1 - t_0}\right) = \log(pI_0) - \delta\frac{(t_1 - t_0)}{2}$$

Denoting $V'_{T1} = \frac{V_{T1}}{t_1 - t_0}$ and $t_{d1} = \frac{t_1 - t_0}{2}$, we have

$$\log(V'_{T1}) = \log(pI_0) - \delta t_{d1}$$

Similarly, over the next time interval ($t_1 < t \leq t_2$), we have

$$\log\left(\frac{V_{T2}}{t_2 - t_1}\right) = \log(pI_1) - \delta\frac{(t_2 - t_1)}{2}$$

On substituting $I_1 = I_0 e^{-\delta(t_1 - t_0)}$ in the above equation and denoting $V'_{T2} = \frac{V_{T2}}{t_2 - t_1}$ and $t_{d2} = \frac{t_2 + t_1}{2} - t_0$, we have

$$\log(V'_{T2}) = \log(pI_0) - \delta t_{d2}$$

We can generalize this over any time interval $t_{n-1} < t \leq t_n$ as

$$\log(V'_{Tn}) = \log(pI_0) - \delta t_{dn}$$

where $V'_{Tn} = \frac{V_{Tn}}{t_n - t_{n-1}}$ and $t_{dn} = \frac{t_n + t_{n-1}}{2} - t_0$, where n=1,2,3 … .

If one plots the natural logarithm of V'_{Tn} versus t_{dn}, one should obtain a straight line with slope δ and δ can be estimated by linear regression.

<u>Case (2): Derivation of viral concentration at time t during the ARV phase with media changes</u>

Let's first assume that media changes occurred twice at t_1 and t_2 between viral load measurements at time points T_0 and T_1. Here $t_0 = T_0$ and $t_3 = T_1$. Additionally, V_{ti} represents the virus concentration at time point t_i. We are interested in determining the virus concentration at any time t between T_0 and T_1 given that media changes occurred twice in between measurements at time points t_1 and t_2. Also, whenever either viremia was measured or media change occurred, V in the well was reduced to V_{MC}.

As before, we have $I(t) = I(0)e^{-\delta(t-t_0)}$ and $V(t) - V_{MC} = \frac{pI(0)}{\delta}(1 - e^{-\delta(t-t_0)})$ when $t_0 < t \leq t_1$. Under the assumption $V_{MC} \ll V_{ti}$ for all values of *i*, we have $V(t) - V_{MC} \sim V(t)$. Therefore, $V(t) = \frac{pI(0)}{\delta}(1 - e^{-\delta(t-t_0)})$ when $t_0 < t \leq t_1$. Similarly, $V(t) = \frac{pI(1)}{\delta}(1 - e^{-\delta(t-t_1)})$ when $t_1 < t \leq t_2$ and $I(1) = I(0)e^{-\delta(t_1-t_0)}$. Here, $I(1)$ is the infected cell population at t_1. Lastly, $V(t) = \frac{pI(2)}{\delta}(1 - e^{-\delta(t-t_2)})$ when $t_2 < t \leq t_3$ and $I(2) = I(1)e^{-\delta(t_2-t_1)} = I(0)e^{-\delta(t_2-t_0)}$. Here, $I(2)$ is the infected cell population at t_2.

Therefore,

$$V(t_3) = V_{T1} = \frac{pI(0)e^{-\delta(t_2-t_0)}}{\delta}(1 - e^{-\delta(t_3-t_2)})$$

Here, $t_2 - t_0$ is the time difference between the time of the last media change and the time of last measurement, while $t_3 - t_2$ is the time difference between the time of current measurement and the time of last media change.

Taking log of both sides

$$\log(V_{T1}) = \log(pI(0)) - \delta(t_2 - t_0) + \log(\frac{1 - e^{-\delta(t_3-t_2)}}{\delta})$$

Assuming $\delta(t_3 - t_2) < 1$ and using a Taylor series, we have

$$\log(V_{T1}) = \log(pI(0)) - \delta(t_2 - t_0) + \log(\frac{2\delta(t_3 - t_2) - \delta^2(t_3 - t_2)^2}{2\delta})$$

or

$$\log(V_{T1}) = \log(pI(0)) - \delta(t_2 - t_0) + \log((t_3 - t_2)[1 - \frac{\delta(t_3 - t_2)}{2}])$$

or

$$\log(V_{T1}) = \log(pI(0)) - \delta(t_2 - t_0) + \log(t_3 - t_2) + \log(1 - \frac{\delta(t_3 - t_2)}{2})$$

Using $\log(1 - x) = -x, for\ x < 1$, we have

$$\log(V_{T1}) - \log(t_3 - t_2) = \log(pI(0)) - \delta(t_2 - t_0) - \frac{\delta(t_3 - t_2)}{2}$$

or

$$\log\left(\frac{V_{T1}}{t_3 - t_2}\right) = \log(pI(0)) - \delta\left(\frac{t_2 + t_3}{2} - t_0\right)$$

As in the previous section, let $V'_{T1} = \frac{V_{T1}}{t_3 - t_2}$ and $t_{d1} = (\frac{t_2 + t_3}{2} - t_0)$, so that

$$\log(V'_{T1}) = \log(pI_0) - \delta t_{d1}.$$

Similarly, if we assume that another media change occurred once at t_4 between measurements at two time points given by $T_1 = t_3$ and $T_2 = t_5$, then

$$\frac{V_{T2}}{t_5 - t_4} = \log(pI(3)) - \delta\left(\frac{t_5 + t_4}{2} - t_3\right)$$

On substituting $I(3) = I_0 e^{-\delta(t_3 - t_0)}$ in the above equation, we have

$$\log(V'_{T2}) = \log(pI_0) - \delta t_{d2}$$

with $V'_{T2} = \frac{V_{T2}}{t_5 - t_4}$ and $t_{d2} = (\frac{t_5 + t_4}{2} - t_0)$.

In general, if two measurements (numbered $T_x = t_n$ and $T_{(x+1)} = t_m$) were made at t_n and t_m while the last media change occurred at t_l, where $t_n < t_l < t_m$, then

$$\log\left(V'_{T(x+1)}\right) = \log(pI_0) - \delta t_{d(x+1)},$$

where $V'_{T(x+1)} = \frac{V_{T(x+1)}}{t_m - t_l}$ and $t_{d(x+1)} = (\frac{t_m + t_l}{2} - t_0)$.

Therefore, in this case as in the previous case, δ can be estimated by linear regression to determine the slope of a plot of the natural logarithm of $V'_{T(x+1)}$versus $t_{d(x+1)}$.

Numerical Procedure

To estimate δ, we performed the linear regression after accounting for media changes in between two measurements, as per the procedure described in Case(1) and Case(2). During the linear regression, all viral load data points that were below the lower limit of detection (LLOD=230 copies/mL) were assigned a value of 230 copies/mL; however only the first viral load data point below the LLOD was used in the process of estimating δ. Measurements below the limit of detection used to compute the trend line are plotted in black, while measurements above the LLOD are plotted in blue in figures (see Figure 4 in the main text).

We further employed another procedure, which is maximum-likelihood procedure that allows for 'censored data' in order to estimate δ (2). In this procedure, HIV viral loads below LLOD were treated as censored data. Furthermore, the logarithm of the viral load was assumed to decay according to a straight line, such that $\log\left(V'_{T(x+1)}\right) = -\delta t_{d(x+1)} + q + \epsilon_{(x+1)}$, where $\epsilon_{(x+1)} \approx N(0, \sigma^2)$ is the error between the theoretical model and the experimental data. Maximum likelihood estimates for this linear regression model are then obtained by searching for parameters (δ, q, σ^2) that maximize the likelihood function as following,

$$\prod_{V'_{T(x+1)} \in U} \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{1}{2\sigma^2}\left[V'_{T(x+1)} - f\left(\theta, t_{d(x+1)}\right)\right]^2} \cdot \prod_{V'_{T(x+1)} \in C} \int_{-\infty}^{5.44} \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{1}{2\sigma^2}\left[u - f\left(\theta, t_{d(x+1)}\right)\right]^2} du$$

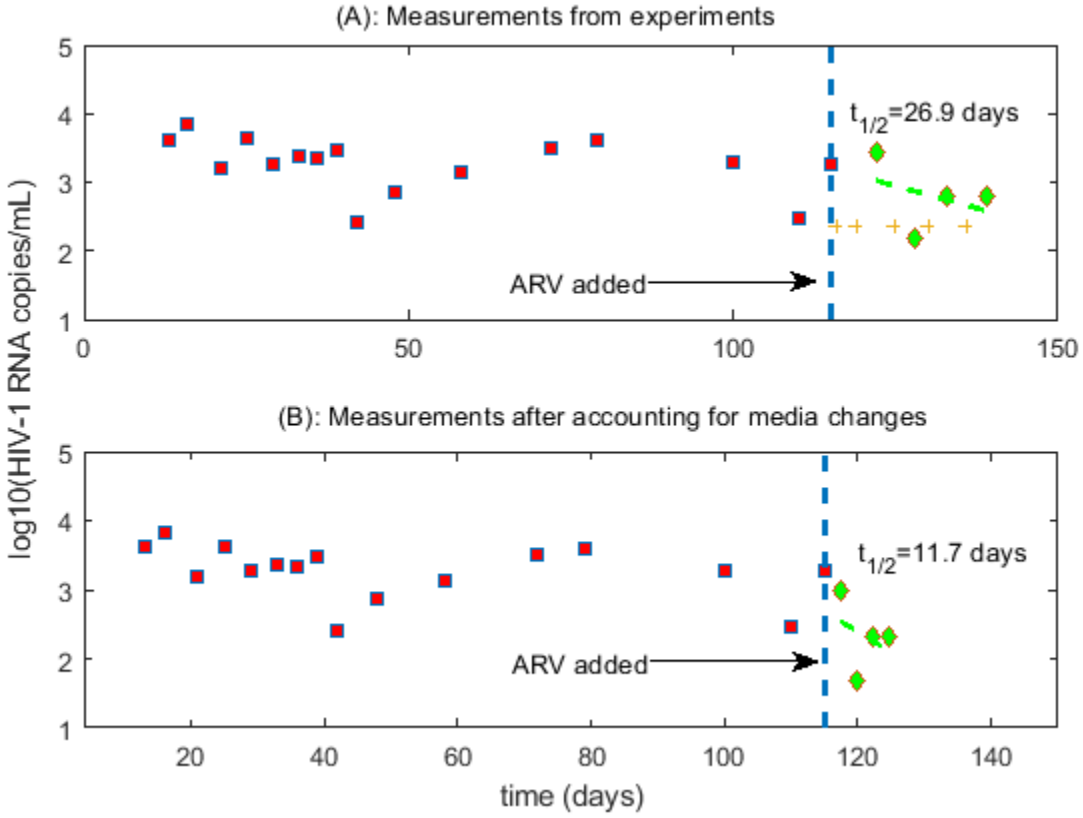
where U and C are uncensored and censored data points, respectively, the upper limit of the integral 5.44 is the natural log of 230, while the linear regression model is $V'_{T(x+1)} = f\left(\theta, t_{d(x+1)}\right) + \epsilon_{(x+1)}$. For more details on this procedure, see ref. (2).

It should be noted that results obtained from the above-mentioned two procedures were similar.

Results

STable 1: *An example, Donor 2 - Well 6: ARV was initiated at day 115. Log represents the natural logarithm.*

Measured in the experiment			**After accounting for media changes**	
Day (media changed)	Day ($T_{(x+1)}$)	log($V_{T(x+1)}$)	Day ($t_{d(x+1)}$)	log($V'_{T(x+1)}$)
$t_0 = 115$	$T_0 = 115$	7.52	$t_{d0} = 115$	7.52
$t_1 = 116$				
$t_2 = 119$				
$t_3 = 122$	$T_1 = 122$	7.95	$t_{d1} = 120.5$	6.85
$t_4 = 125$				
$t_5 = 128$	$T_2 = 128$	5.0	$t_{d2} = 126.5$	3.90
$t_6 = 130$				
$t_7 = 133$	$T_3 = 133$	6.45	$t_{d3} = 131.5$	5.35
$t_8 = 136$				
$t_9 = 139$	$T_4 = 139$	6.43	$t_{d4} = 137.5$	5.33



SFigure 1: *Illustration of the analysis of data obtained after the start of ARV that takes media changes into account for Donor 2- well 6 (and this process is also shown in* ***STable 1****): (A) plotted as measured in experiments, (B) plotted after accounting for media changes; time plotted is the scaled time t_d and the plotted viral load is the scaled viral load V'. Individual points are plotted pre-ARV (red), during ARV (green) and media changes (cyan, + marker).*

STable 2: *The half-life of infected cells after accounting for media changes post-ARV.*

Donor number and well number	Half-life of infected cells ($t_{1/2} = \frac{log(2)}{\delta}$) in days
Donor 2 and well 3	Indeterminate
Donor 2 and well 4	2.3
Donor 2 and well 6	11.7
Donor 2 and well 10	4.8
Donor 3 and well 3	33.4
Donor 3 and well 4	4.1
Donor 3 and well 8	3.2
Donor 3 and well 9	3.6
Mean (Median)	9.0 (4.1)

References

1. **Perelson AS, Neumann AU, Markowitz M, Leonard JM, Ho DD.** 1996. HIV-1 dynamics in vivo: virion clearance rate, infected cell life-span, and viral generation time. Science **271:**1582-1586.
2. **Di Mascio M, Dornadula G, Zhang H, Sullivan J, Xu Y, Kulkosky J, Pomerantz RJ, Perelson AS.** 2003. In a subset of subjects on highly active antiretroviral therapy, human immunodeficiency virus type 1 RNA in plasma decays from 50 to <5 copies per milliliter, with a half-life of 6 months. J Virol. **77:**2271-2275.