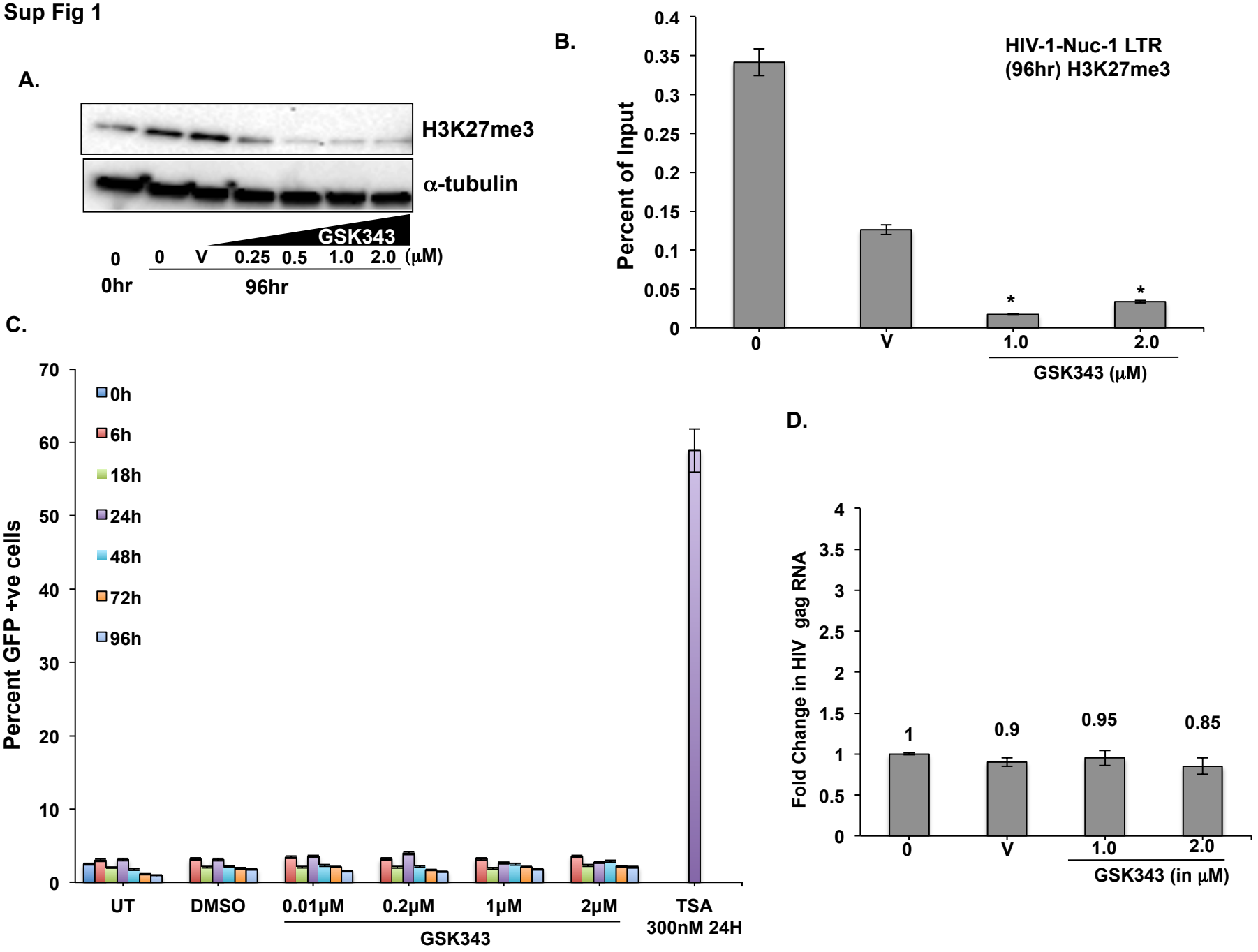
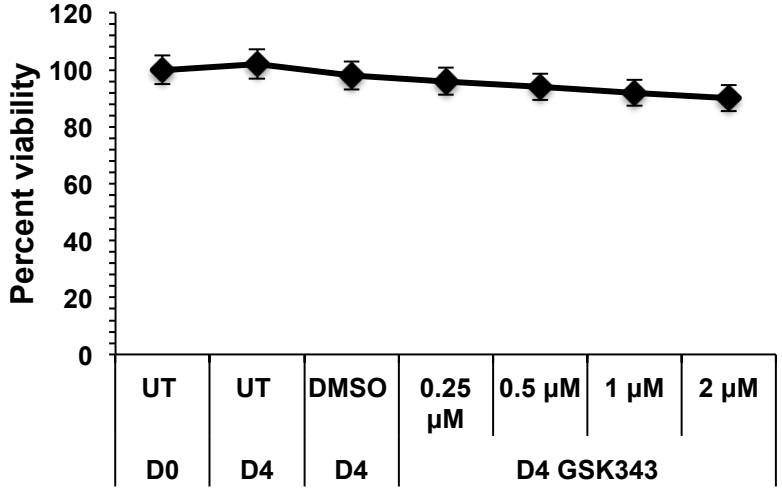
Sup Fig 1

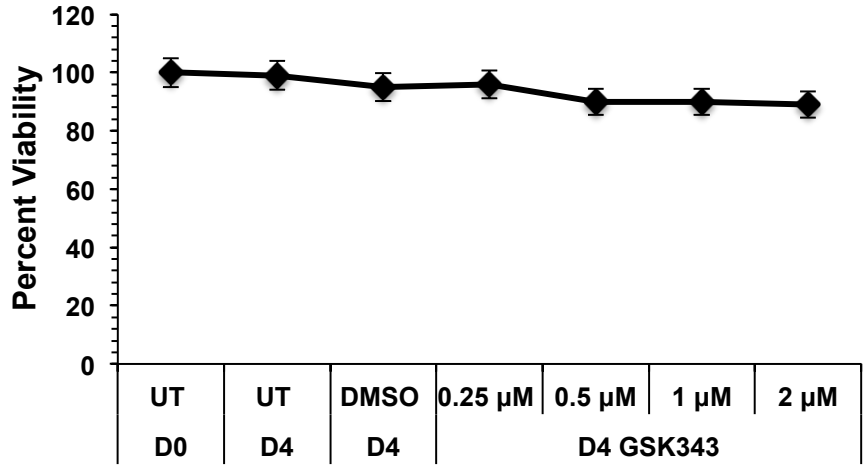

A.
H3K27me3
α-tubulin
GSK343
0 0 V 0.25 0.5 1.0 2.0 (μM)
0hr
96hr
B.
HIV-1-Nuc-1 LTR
(96hr) H3K27me3
Percent of Input
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
*
*
0
V
1.0
2.0
GSK343 (μM)
C.
Percent GFP +ve cells
70
60
50
40
30
20
10
0
0h
6h
18h
24h
48h
72h
96h
UT
DMSO
0.01μM
0.2μM
1μM
2μM
TSA
300nM 24H
GSK343
D.
Fold Change in HIV gag RNA
4
3.5
3
2.5
2
1.5
1
0.5
0
1
0.9
0.95
0.85
0
V
1.0
2.0
GSK343 (in μM)

Sup Fig 2

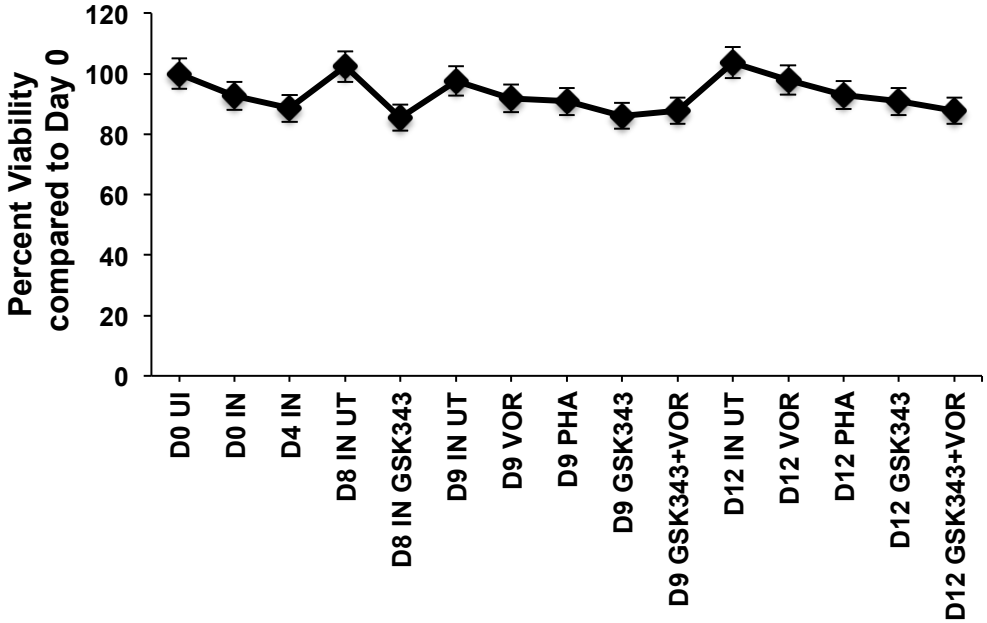
A.


Percent viability
120
100
80
60
40
20
0
UT
UT
DMSO
0.25 µM
0.5 µM
1 µM
2 µM
D0
D4
D4
D4 GSK343

B.


Percent Viability
120
100
80
60
40
20
0
UT
UT
DMSO
0.25 µM
0.5 µM
1 µM
2 µM
D0
D4
D4
D4 GSK343

C.


Percent Viability compared to Day 0
120
100
80
60
40
20
0
D0 UI
D0 IN
D4 IN
D8 IN UT
D8 IN GSK343
D9 IN UT
D9 VOR
D9 PHA
D9 GSK343
D9 GSK343+VOR
D12 IN UT
D12 VOR
D12 PHA
D12 GSK343
D12 GSK343+VOR

Sup Fig 1.EZH2 inhibitor GSK343 leads to a reduction of trimethylated H3K27 in Jurkat J-89 model of latency without proviral activation: **A.** A western blot showing global reduction in H3K27me3 in whole cell lysate at different concentrations of GSK343 as indicated for 96 hrs. Histone H3 and α-tubulin are shown as loading controls. (V: vehicle control, 0.02% DMSO) **B.** ChIP at HIV LTR Nuc-1 promoter for H3K27me3 normalized to HA as an unrelated isotype control and total Histone H3 occupancy at 96 h. Results were compared with cells treated with vehicle control DMSO using Student paired t-test. Error bars represent SEM; n = 3. *$p < 0.05$. **C.** Flow cytometry measuring proviral activation (% GFP-positive) in untreated (UT), DMSO (V) and GSK343 treated at the indicated concentrations for 0-96 hrs. TSA (300 nM) treatment for 24h serves as a positive control. **D.** The relative fold change of cell-associated *gag* RNA expression upon GSK343 exposure, normalized to GAPDH, as measured by quantitative RT-PCR at 96hr. Untreated (0), DMSO vehicle control (V). Mean and SEM of three independent experiments.

Sup Fig 2. Viability following GSK343 exposure by MTT Assay: A) 2D10s as described in Fig 2A; **B)** PBMCs as in Fig 6; **C)** Resting T- cell latency model as described in Fig 7.

Target	Primers (F and R) and probe (P) sequences- (5'-3')
Nuc-0	**F**- ACA CAC AAG GCT ACT TCC CTG A **R**-TCT ACC TTA TCT GGC TCA ACT GGT
Nuc-1	**F**-CTG GGA GCT CTC TGG CTA ACT A **R**- TTA CCA GAG TCA CAC AAC AGA CG
Gag (ChIP)	**F**-AGA TAA AAG ACA CCA AGG A **R**- GGC CTG ATG TAC CAT TTG C
GAPDH (ChIP)	**F**-TGA GCA GAC CGG TGT CAC TA **R**-AGG ACT TTG GGA ACG ACT GA
SAT2	GPH110003C(+)01A **(QIAGEN)**
MyoD1	GPH110002C(+)01A **(QIAGEN)**
Gag (RT-PCR)	**F** -ACA TCA AGC AGC CAT GCA AAT **R**-TCT GGC CTG GTG CAA TAG G **P- /FAM/**CTA TCC CAT TCT GCA GCT TCC TCA TTG ATG**/BHQ-1/**
GAPDH (RT- PCR)	**F** –GCA CCA CCA ACT GCT TAG CAC C **R-**TCT TCT GGG TGG CAG TGAT G **P- /HEX/** TCG TGG AA GGA CTC ATG ACC ACA GTC C**/BHQ-1/**
GFP (RT-PCR)	**F**- GGA GCG CAC CAT CTT CA **R**- AGG GTG TCG CCC TCG AA **P- /FAM/** CTA CAA GAC CCG CGC CGA GGT G **/BHQ-1/**