Supporting Information

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Fig. S1. Effect of mAb AP3 to $\alpha V\beta$ 3-integrin on R8102 infection. mAb AP3 does not block interaction of $\alpha V\beta$ 3-integrin with its ligands. The indicated cells lines were exposed to indicated concentrations of mAb Ap3 (full symbols) or control IgGs (void symbols) for 1 h, infected with R8102 (3 pfu/cell) in the same medium, and overlaid with mAb-containing medium until harvesting. In *A*–*D*, the extent of infection was quantified from the Lac-Z gene engineered in the viral genome under the immediate-early α 27 promoter. Cells in 96 wells were fixed at 6–8 h after infection. Extent of β -Galactosidase activity reflects the amount of infection. Each point represents triplicates' average; 100% infection is the value obtained with no antibody. Bars show SD.

Table S1.	Percentage cell viabi	itv in cultures exc	oosed to inhibitors	determined by	Alamar Blue
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Inhibitor	$CHO\text{-}N1\alpha\pm\alphav\beta3$	$\text{CHO-N1}\delta \pm \alpha v\beta 3$	$J\text{-}N1\delta \pm \alpha \nu\beta 3$	293T	HT29 $\pm \alpha v\beta 3$	SW480	1143
Nystatin (100 µM)	94	96	98	99	95	97	95
Filipin (3 μM)	90	95	98	100	93	98	94
Dynasore (75 µM)	91	93	97	98	95	94	95
BFLA (30 nM)	98	97	97	100	97	94	96

For all inhibitors, for each concentration and cell line, toxicity was measured simultaneously with the effect of the inhibitors on virus infection by adding 10% Alamar Blue (Invitrogen) to replicate specimens in DMEM lacking phenol red from 0 time until harvesting and optical reading at 570 and 600 nm. Figures in the table represent the percent of viable cells at the highest inhibitor concentration (reported in parentheses). BFLA, bafilomycin A.

Inhibition of infection	CHO-N1δ	CHON1δ- αVβ3	CHO-N1α	CHO-N1α- αVβ3	J-N1α	J-N1α- αVβ3	293	293-αVβ3	HT	ΗΤ-αVβ3	SW 480	1143
mAb L230		+		+			±	+	±	+	++	_
Nystatin	_	+	-	+	-	+	+	++	_	++	+	_
Filipin	_	+	_	+	±	+	+	++	+	++	+	_
Cav1Y14A	_	_										
Dynasore	_	+	-	+	-	+	+	++	+	++	++	±
DYN K44A	_	+	-	+								
BFLA	+	+	+	+	-	+	+	++	+	+	+	-

Table S2. αVβ3-integrin modifies HSV entry pathway (summary of main modifications induced by αVβ3-integrin)

Inhibition 0–20% (–); inhibition 20–40% (±); inhibition >40% (+); inhibition >80% (++).