

**Cell Host & Microbe, Volume 24**

## **Supplemental Information**

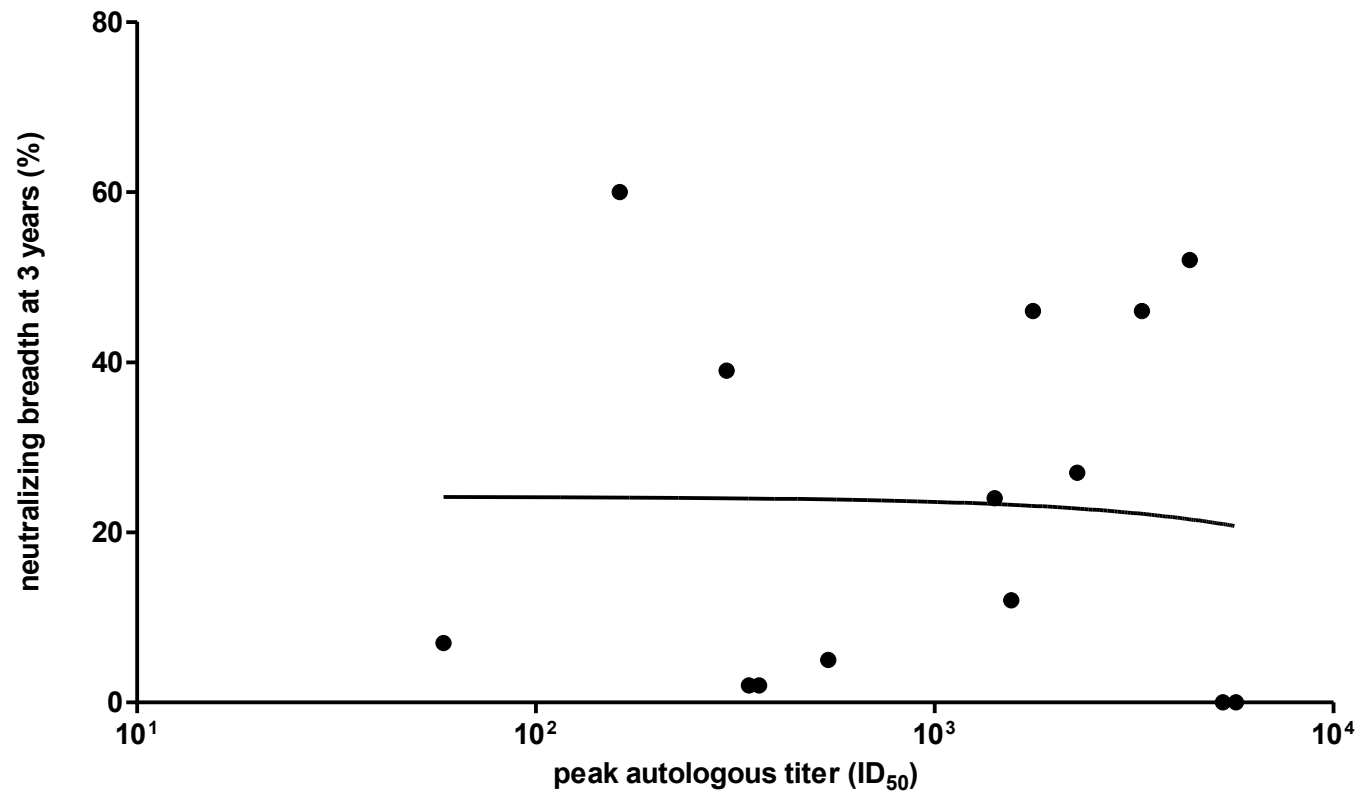
### **HIV Superinfection Drives *De Novo***

### **Antibody Responses and Not Neutralization Breadth**

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<b>PID</b>	<b>Timing of superinfection (weeks post infection)</b>	<b>VL at 52 weeks (copies/ml)</b>	<b>CD4 count at superinfection (cells/<math>\mu</math>l)</b>	<b>PI-SI env distance</b>
CAP237	9 (6-11)	11900	522	11.92%
CAP256	13 (11-14)	178000	555	12.53%
CAP281	42 (40-44)	<400	1199	13.99%
CAP334	(42-47)	14900	(458-543)	nd
CAP377	33 (30-37)	337496	462	15.18%



Plasma neutralization breadth

166

R

K

169

K

I

Q

T

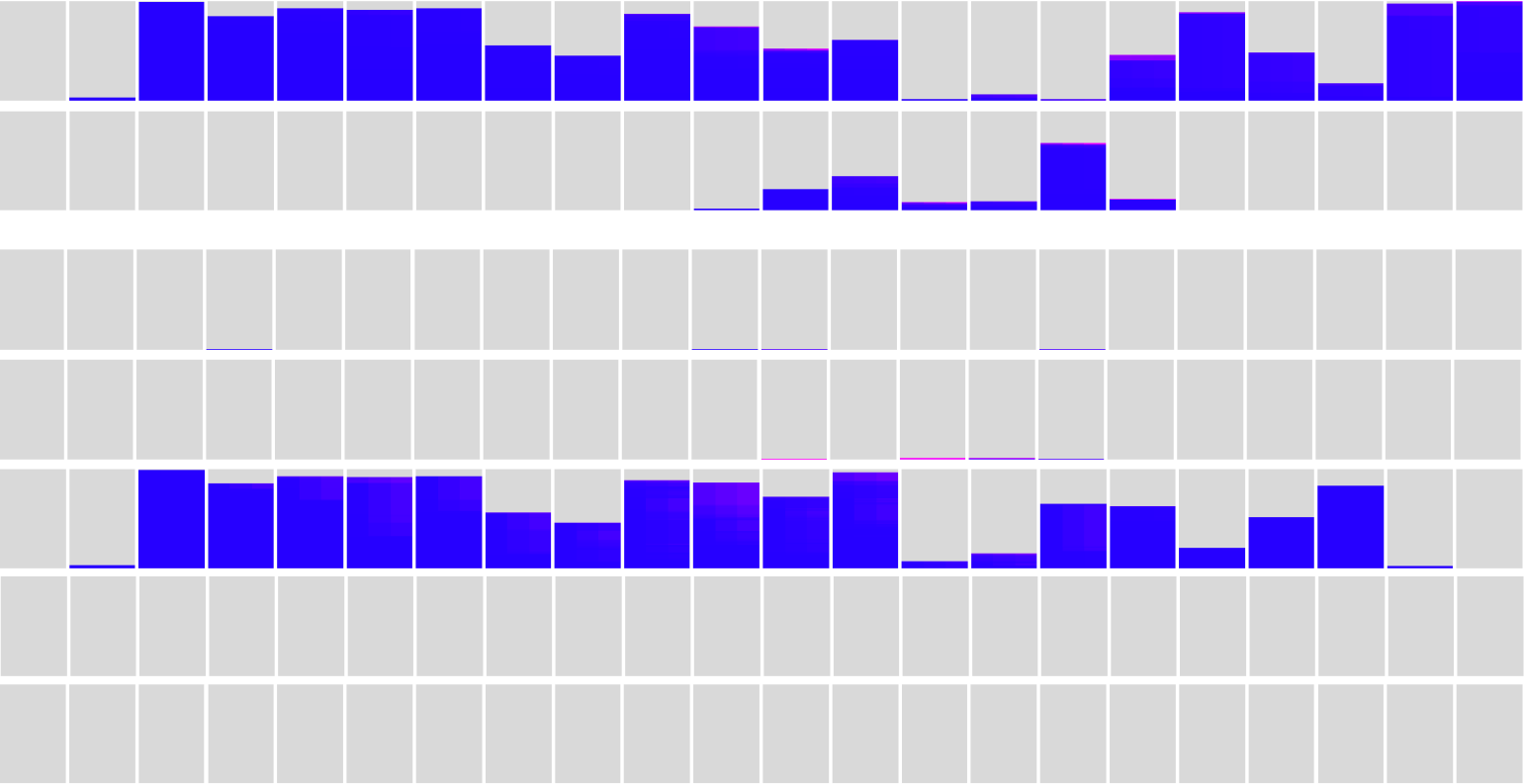
R

15 17 23 27 30 34 38 42 48 53 59 69 82 94 106 119 134 145 159 176 193 206

time post infection (weeks)

100%  
50%  
0%

0.5  
0.75  
1.0  
PI



**Figure S1. Identification of superinfection in five participants, Related to Figure 1a.** The estimated timing of superinfection, with the confidence interval in parenthesis, is summarized together with the viral load (VL) at 1 year post primary infection, CD4 count at the visit closest to the estimated time of superinfection. PI-SI distance; the DNA distance in *env* between the transmitted/founders of primary infection and superinfection for each participant. nd; not done. PID; anonymized participant ID numbers.

**Figure S2. Potency does not lead to breadth, Related to Figure 1b.** No significant correlation ( $P = 0.9559$ ,  $R^2 = 0.0002$ ) between the potency of the autologous neutralizing antibody response and the later development of neutralization breadth in singly infected participants ( $n=14$ ). Breadth was measured at 3 years post infection and potency was estimated as the mean of the 3 highest titers observed against an acute/early Env clone.

**Figure S3. Frequency of key genotypes in the primary infecting lineage over time, Related to Figure 4.** Only residues that were assigned to the primary infecting virus lineage with posterior probabilities  $>0.5$  are included, where more confident assignments are bluer.