**Broadly neutralizing antibody responses in the longitudinal primary HIV-1 infection SPARTAC cohort**

Running head: Neutralizing antibodies in SPARTAC cohort

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**Supplemental Tables S1-S6.**

**Table S1: Neutralization of 6-virus panel and clinical data for control arm of SPARTAC cohort.** Neutralization breadth was measured on a cross-clade 6-virus indicator panel and this data was used to calculate a neutralization score (Neut Score, see methods). The ID50 values are colour coded based on potency. Data also reported includes weeks post recruitment, geometric mean ID50, sex, transmission route (HSW, MSM, MSW), location (UK, Australia (AU), Italy (IT), Brazil (BR), South Africa (SA)), HIV-1 clade, viral load (RNA copies/mL) and CD4 count (per mL) at recruitment, setpoint and the time neutralization was measured.



**Table S2: Multivariate analysis.** A) Correlation of neutralization score versus week post infection (WPI), time in years to initiation of anti-retroviral therapy (ART) and logarithmic viral load at neutralization measurement and at recruitment to trial. Reported are Estimate, Standard (Std.) error, p- and *r2-*vales for univariate and multivariate analysis. B) Correlation of neutralization score versus WPI and logarithmic viral load at neutralization measurement. Reported are Estimate, Standard (Std.) error, p- and *r2*-vales for univariate and multivariate analysis.

A

B

**Table S3: Epitope mapping for glycan-dependant bnAb epitopes.** Fold changes in ID50 for mutant viruses compared to wild-type virus are reported. + indicates neutralization of one virus decreased 3-5 fold, ++ indicates 2 viurses decreased 3-5-fold, +++ indicates 5-fold decrease for at least 2 viruses with glycan site deletion.



**Table S4: MPER peptide competition for neutralization.** Fold-decrease in plasma neutralization (ID50) when competed with MPER peptide. + indicates a decrease in plasma neutralization potency of 3-fold when competed with soluble MPER peptide. The viruses used in this assay were selected based upon the neutralization sensitivity in plasma.

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**Table S5: RSC3 binding to determine CD4 binding site specificity.** Reported are area under the curve (AUC) of serum binding titres to RSC3, RSC3Δ371I and RSC3Δ371I P363N and the AUC-ratio of RSC3 and RSC3Δ371I P363N. AUCs smaller than the AUC of VRC01 to RSC3Δ371I P363N were set to 50 for simplicity. Binding intensities of sera at 1:50 serum dilution to RSC3 were related to 2G12 binding at 20 µg/mL. RSC3 (VRC01-like): If the ratio between the area under the curve (AUC) for RSC3 /RSC3Δ371I P363N is 2-3 (+/-), 3-8 (+) and >8 (++). RSC3 (no-differential): If the ratio between AUC of RSC3/RSC3Δ371I/P363N) is 1.8 and the strength is dependent on percentage of 2G12-binding (20 µg/mL) at 1:50 serum-dilution: ++ is 50%, + is <50%, but 25%, +/- is <25%.



**Table S6: RSC3 competition for neutralization.** Reported arefold-decreases in ID50 when plasma are competed with RSC3 and RSC3Δ371I/P363N, as well as the ratio between ID50 fold changes for each RSC3 and RSC3Δ371I/P363N. RSC3 competition (VRC01-like): + if the neutralization is decreased by 3 fold for RSC3 but not RSC3Δ371I/P363N. RSC3 competition (non-differential): + if the neutralization is decreased by 3 fold for both RCS3 and RSC3Δ371I/P363N.

