BACKGROUND
The HIV Prevention Trials Network (HPTN) 052 was a multi-
national, clinical trial that demonstrated reduced HIV transmission and health benefits with early antiretroviral treatment (ART) [1, 2].

METHODS
STUDY COHORT
HPTN 052 enrolled participants from Botswana, Kenya, Malawi, South Africa (Zambia, Brazil, India, and South Africa), and Zimbabwe. Other participants were on a PI-based regimen or other EFV-based regimens (4.3%). e In the 3 months prior to ART initiation.

RESISTANCE AT BASELINE AND VIROLOGIC FAILURE
Overall, 4.7% of participants had resistance at baseline and 35.5% had new resistance at failure (Figure 2). Among those with new resistance at virologic failure, 62.7% had non-nucleoside reverse-transcribe enzyme inhibitor (NRTI) resistance and 24.3% had non-nucleoside reverse-transcribe enzyme inhibitor (NRTI) resistance, and 16.0% had multiclass resistance (NRTI and NNRTI). The most common NNRTI and NRTI resistance mutations detected were K103N and M184V (70.1% and 90.3%, respectively).

METHODS
Laboratory methods for genotyping were performed using the ViroSeq HIV-1 Genotyping System v2.8. Resistance was assessed using the Resistance Calculator Program (Frontier Science Foundation) using the Stanford v7.0 algorithm. HIV subtype was determined by phylogenetic analysis.

STATISTICAL METHODS
Virologic failure was defined as two consecutive viral loads >1,000 copies/mL ≥24 weeks after ART initiation. Factors associated with HIV drug resistance were analyzed using Chi-square,anova, t-tests, and logistical regression models, using SAS software.

RESULTS
STUDY COHORT
In HPTN 052, 249 participants had virologic failure. Paired baseline/failure resistance results were obtained for 211 participants (128 in the early ART arm; 83 in the delayed ART arm). Figure 1. Participants with virologic genotyping results.

Figure 2. HIV drug resistance in participants with virologic failure.

CONCLUSIONS
New resistance at failure was less frequent in the early ART arm than the delayed ART arm (30.5% vs. 43.4%, p<0.06), and was less frequent in the delayed ART arm in those who started ART before vs. after May 2011 (54.5% vs. 39.3%, p=0.032, Table 2).

The authors thank the HPTN 052 study team and participants for providing the samples and data used in this study. We also thank the laboratory staff who helped with sample management and testing.

REFERENCES

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Factors associated with new resistance at virologic failure

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New resistance at failure was less frequent among participants with higher baseline CD4 cell counts (p=0.047) and lower baseline viral loads (p=0.0001), and was higher among those receiving EFV/3TC/ZDV compared to those receiving other ART regimens (p=0.0074).

In a multivariable model, new resistance at failure was associated with baseline viral load (p<0.0008) and drug regimen (p<0.024).

New drug resistance at virologic failure was less frequent among participants with early ART initiation, but this difference was not statistically significant.

New drug resistance at virologic failure was more frequent among those receiving EFV/3TC/ZDV than among those receiving other ART regimens.

Lower baseline (pre-ART) viral load was the main factor associated with acquisition of HIV drug resistance.

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Most participants had HIV subtype C (76.8%) or B (11.8%). The majority (74.9%) of the participants were on an ART regimen containing efavirenz (EFV), lamivudine (3TC), and zidovudine (ZDV). Other participants were on a PI-based regimen or other EFV-based regimens.

CONCLUSIONS
• New drug resistance at virologic failure was less frequent among participants with early ART initiation, but this difference was not statistically significant.
• New drug resistance at virologic failure was more frequent among those receiving EFV/3TC/ZDV than among those receiving other ART regimens.
• Lower baseline (pre-ART) viral load was the main factor associated with acquisition of HIV drug resistance.