HIV-1 Subtypes and Virological Response to HAART in Europe

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BACKGROUND
Antiretroviral (AR) regimens may vary in ability to suppress viral load (VL) in people infected with different HIV subtypes, e.g. due to differences in resistance development. AR drugs have predominantly been developed in Western Europe and the USA on the basis of subtype B, and is the most prevalent strain. Resistance patterns have also been defined according to subtype C, however, subtype B, which is widespread in Africa, Asia and much of Eastern Europe are increasingly spreading worldwide through travel and migration.

OBJECTIVE
To compare virological response to highly active antiretroviral therapy (HAART) in patients infected with different HIV subtypes in Europe.

METHODS
Analysis was carried out on HIV-1 infected patients in the EuroSIDA study who met the following inclusion requirements:

- Started HAART defined as at least three antiretroviral drugs (including at least one protease inhibitor (PI), non-nucleoside reverse transcriptase inhibitor (NNRTI), abacavir (ABC) or tenofovir (TFV), with no prior PI/NNRTI/ABC/TFV experience.
- Subtype determined before starting HAART.
- Unsuppressed viral load defined as ≥500 copies/mL, measured within six months before starting HAART.
- Virological response to HAART (whether or not first viral load measured at 6 months from start of HAART was suppressed).

RESULTS (1)

- 684 (6%) of the 11 229 patients in EuroSIDA met the inclusion criteria, of which 79 (12%) were infected with subtype A, 542 (81%) with B, 28 (4%) with C and 5 (0.5%) with any other subtype.
- 97 (14%) of the subtypes were determined by phylogenetic analysis, 89% of patients, 4% of subtype B. The remaining subtypes were determined from phylogenetic follow-up forms, sample analysis and phylogenetic paper forms.
- Median dates of starting HAART were July 1998 and May 1999 for B and non-B respectively, p=0.001 (Table 1).
- Baseline viral loads were similar between B and non B infected patients (4.7 and 4.6 log10 copies/mL, p=0.289), as were baseline CD4 counts (502 and 434 cells/µL, p=0.545).

- Baseline VL and CD4 counts were similar between B and non B infected patients (4.7 and 4.6 log10 copies/mL, p=0.289), as were baseline CD4 counts (502 and 434 cells/µL, p=0.545).
- 25% of B and 32% of non-B patients had RT (reverse transcriptase) genetic sequences available with which to study the pattern of resistance mutations, within one year before starting HAART.

- Levels of NRTI resistance, defined as at least one US FDA (Drugs for HIV resistance) mutation, were similar between groups, p=0.35.

- The prevalence of different subtypes was found to differ significantly between regions, p=0.001 (Figure 1). However, there may be some bias in patient selection for subtype testing and so results cannot be assumed to reflect the underlying prevalence in the groups.
- HAART regimens also did not differ significantly between groups, p=0.082 (Figure 2). Over 50% started regimens containing one PI, and 57% of B and 44% of non-B were AR-naive at baseline, p=0.15.