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Antiretroviral Drug Resistance Report

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The following report summarizes apparent antiretroviral drug resistance. It is based upon an analysis* of protease and reverse transcriptase gene sequences of the HIV-1 virus amplified from the supplied specimen (see laboratory numbers above). Additional information detailing the HIV-1 subtype and a quality assessment of the sequence data is also included. The report utilises the following abbreviations: B,D,H,V,N = Standard IUPAC codes for ambiguous nucleotides, PR = Protease, PI = Protease Inhibitor, RT = Reverse Transcriptase, RTI = Reverse Transcriptase Inhibitor, TAMs = Thymidine Analogue-associated Mutations.

* Analysis performed using the Stanford Genotypic Resistance Interpretation Algorithm Version 6.0.7

SUMMARY DATA

Sequence includes PR: codons: 1 - 99

Sequence includes RT: codons: 1 - 342

There are no insertions or deletions

Subtype and % similarity to closest reference isolate:

1. PR: B (94.3%)
2. RT: B (94.2%)

SEQUENCE QUALITY ASSESSMENT

Gene	QA Problem	Codons
PR	Stop Codons, Frame Shifts:	None
PR	B,D,H,V,N:	None
PR	Unusual Residues:	None

Gene	QA Problem	Codons
RT	Stop Codons, Frame Shifts:	None
RT	B,D,H,V,N:	None
RT	Unusual Residues:	None

DRUG RESISTANCE INTERPRETATION

PI Major Resistance Mutations: None

PI Minor Resistance Mutations: None

Other Mutations: I15V, E35D, M36I, R41K, R57KR, L63P, E65D, I93L

Protease Inhibitors

atazanavir/r (ATV/r)	Susceptible
darunavir/r (DRV/r)	Susceptible
fosamprenavir/r (FPV/r)	Susceptible
indinavir/r (IDV/r)	Susceptible
lopinavir/r (LPV/r)	Susceptible
nelfinavir (NFV)	Susceptible
saquinavir/r (SQV/r)	Susceptible
tipranavir/r (TPV/r)	Susceptible

PR Comments

- This sequence has 0 major TPV/r-resistance mutations, 1 minor TPV/r-resistance mutations (M36I), and 0 mutations associated with increased TPV/r responsiveness. RESIST study (Baxter J et al J Virology 2006 and Scherer J et al EACS 2007).
- M36I is weakly associated with PI resistance in subtype B viruses when present with other mutations. However, M36I is the consensus amino acid in most non-B subtypes.
- L63P is a common polymorphism that becomes even more common in persons receiving PIs.
- I93L is a common polymorphism. It is the consensus residue in most subtypes. In subtype B, it is weakly associated with PI treatment.

NRTI Resistance Mutations: None

NNRTI Resistance Mutations: **K103N, V106I**

Other Mutations: E6D, E28EG, I135T, I202V, R211K, F214L, V276IV, K277KR, L283I, A288T, I293V, P313S, V317A, E328D

Nucleoside RTI

lamivudine (3TC)	Susceptible
abacavir (ABC)	Susceptible
zidovudine (AZT)	Susceptible
stavudine (D4T)	Susceptible
didanosine (DDI)	Susceptible
emtricitabine (FTC)	Susceptible
tenofovir (TDF)	Susceptible

Non-Nucleoside RTI

delavirdine (DLV)	High-level resistance
efavirenz (EFV)	High-level resistance
etravirine (ETR)	Potential low-level resistance
nevirapine (NVP)	High-level resistance

RT Comments

- K103N causes high-level resistance to NVP, DLV, and EFV. By itself it has no effect on ETR susceptibility. However, it has a synergistic effect with L100I and possibly K101P on ETR susceptibility.
- V106I is a common polymorphism that was associated with decreased ETR response in the DUE study. However, it does not decrease NNRTI susceptibility.
- The following 1 of the 13 etravirine DUET study mutations were present: V106I (Katlama C et al, IAS 2007).