HIV-1 Genotypic Antiretroviral Resistance Test

Stanford University Medical Center Stanford Hospital and Clinics Clinical Laboratories 300 Pasteur Drive, Stanford, CA 94305 Dr. R. Sibley & Dr. S. Geaghan (877) 717-3733

Cliffical Laboratories				(877) 717-3733				
Last Name Clinic MR Number Collection Date Date Entered	Patient UNKNOWN 123456 07/17/2010 07/17/2010		First Name Physician Accession Number Received Date File Name	Unknown Unknown 123456 (49138) 07/17/2010				
Sequence includes PR codons: 1 - 99 Sequence includes RT codons: 1 - 299 There are no insertions or deletions Subtype: B No. previous patient sequences: PR:0 RT:0								
PI Major Resistance I PI Minor Resistance I Other Mutations P atazanavir/r (ATV/r) darunavir/r (DRV/r) fosamprenavir/r (FPV indinavir/r (IDV/r) lopinavir/r (LPV/r) nelfinavir (NFV) saquinavir/r (SQV/r) tipranavir/r (TPV/r)	Mutations V82A Mutations None I64V, T rotease Inhibit Low-level resist Susceptible //r) Potential low-le Intermediate res Low-level resist Intermediate res Potential low-le Susceptible	V I72M ors tance vel resistance sistance tance sistance vel resistance						
NRTI Resistance MutationsD67N, K70RNNRTI Resistance MutationsV90IV, K103Other MutationsV60I, K102RT286A, E297		K70R, M184IMV, 7, K103N, K238N K102R, D123E, T13 4, E297K	R, M184IMV, K219Q 3N, K238N R, D123E, T139KR, S162NS, K166KR, I178M, G196E, D237DN, A272S, 7K					
lamivudine (3TC) abacavir (ABC) zidovudine (AZT) stavudine (D4T) didanosine (DDI) emtricitabine (FTC) tenofovir (TDF)	Nucleoside K11High-level resistanceLow-level resistanceIntermediate resistanceIntermediate resistancePotential low-level resistanceHigh-level resistancePotential low-level resistancePotential low-level resistance		N delavirdine (DLV) efavirenz (EFV) etravirine (ETR) nevirapine (NVP)	High-level resistance High-level resistance Potential low-level resistance High-level resistance				

PR Comments

+ V82A reduces susceptibility to IDV/r and LPV/r. With other mutations it is associated with reduced susceptibility to NFV, ATV/r, SQV/r, and FPV/r.

RT Comments

- + K70R causes low-level AZT, d4T, and possibly TDF resistance.
- + K219Q/E decrease AZT and probably d4T susceptibility when present with K70R or T215Y/F but have little if any effect on the remaining NRTIs.
- + M184V/I cause high-level in vitro resistance to 3TC and FTC and low-level in vitro resistance to ddI and ABC. M184V/I increase susceptibility to AZT, TDF, and d4T.
- + 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.
- + V90I is a common polymorphism that was weakly associated with decreased ETR response in the DUET studies. However, it has minimal if any effect on NNRTI susceptibility.
- + D67N contributes some degree of resistance to each of the NRTIs except 3TC and FTC. It usually occurs with mutations at positions 70 or 215.
- + K238T is an NNRTI-selected mutations that usually occur in combination with K103N in which case it causes high-level resistance to NVP, EFV, and DLV. Its effect on ETR is not known. K238N is a rarer NNRTI-selected mutation at this position. Its phenotypic effect is not known.
- + The following 1 of the 13 etravirine DUET study mutations were present: V90I (Katlama C et al, IAS 2007).

PR Mutation Scores

	ATV/r	DRV/r	FPV/r	IDV/r	LPV/r	NFV	SQV/r	TPV/r
V82AV	15	0	10	35	25	35	10	6
Total:	15	0	10	35	25	35	10	6

RT Mutation Scores

	3TC	ABC	AZT	D4T	DDI	FTC	TDF	DLV	EFV	ETR	NVP
D67N	0	8	15	12	8	0	5	0	0	0	0
K70R	0	0	18	10	0	0	8	0	0	0	0
K103N	0	0	0	0	0	0	0	60	60	5	60
M184IMV	60	12	0	0	5	60	0	0	0	0	0
K219Q	0	0	15	10	0	0	0	0	0	0	0
K238N	0	0	0	0	0	0	0	30	15	5	30
Total:	60	20	48	32	13	60	13	90	75	10	90

The Genotypic Antiretroviral Resistance Test reports mutations in HIV-1 protease and RT. Mutations are defined as differences from the wildtype consensus B reference sequence. The interpretation is based on published data in the scientific and medical literature linking mutations and combinations of mutations to phenotypic and clinical drug resistance. The report should be used in conjunction with a patient's clinical history (including past treatments) and with a solid understanding of the principles of antiretroviral treatment (http://www.aidsinfo.nih.gov/guidelines/). A more detailed description of the test interpretation, which includes the consensus B protease and RT sequence, all of the mutation scores, all of the mutation comments, and updates can be found on the Stanford HIV Drug Resistance Database http://hivdb.stanford.edu/pages/asi/releaseNotes/.

Laboratory test performed by Stanford Virology Laboratory at Stanford Hospital, 300 Pasteur Dr., Stanford, CA 94305. This test was developed and its performance characteristics determined by the Stanford Clinical Micro/Viro lab. It has not been cleared or approved by the U.S. Food and Drug Administration. Such approval is not required for tests validated by the performing laboratory.