Genotypic ARV Resistance Report

Patient Information

Our Ref. ID: VCI-20949

Name:	♂ ชินโชติ ชัยรุจิขจร	**Your Ref. ID:**	**P16-00792**
Hospital/Site:	PRIBTA	**Study/Visit:**	
Risk Factor:	No Information	**Collection Date:**	19-Nov-2021
Clinical Staging:	No Information	**CDC Staging:** No Information	**Genotyping Date:** 26-Nov-2021

Lab Information

Current CD4:	No Information	**Current Antiretroviral:**	Unknown
Current VL:	No Information		

Summary Data

Subtype and % similarity to closest reference isolate: CRF01_AE (97.4%)

Sequence includes RT: condons: 20 - 267

Resistance Report (RT)

RT TAMs: -

RT NRTIs: -

RT NNRTIs: -

RT Other: V35T, T39K, E42EK, K43E, V60I, S68G, K122E, D123S, K173L, Q174K, V179I, G196E, T200I, Q207A, R211S, K238R, V245E

Antiretroviral	High-level resistance	Intermediate resistance	Low-level resistance	Potential low-level resistance	Susceptible
NRTI					
zidovudine (AZT)					
tenofovir (TDF)					
stavudine (D4T)					
lamivudine (3TC)					
emtricitabine (FTC)					
didanosine (DDI)					
abacavir (ABC)					
NNRTI					
rilpivirine (RPV)					
nevirapine (NVP)					
etravirine (ETR)					
efavirenz (EFV)					
doravirine (DOR)					

ConsensusR#20949RT.txt

Remark: 1. Although the mutation is not found, it does not mean that one is fully susceptible to the treatment since the resistant virus may be minor population which cannot be detected by the assay (detectable limit = viral load 1,000 copies/ml).
2. The accumulation of TAMs (M41L, D67N, K70R, L210W, T215Y/F, K219Q/E) increases resistance to tenofovir. Mutations M41L and L210W, contribute more than others
3. References: Stanford dBase system (http://hivdb.stanford.edu/)

Reported by: _____ Date: 26/11/2021

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RT Comments

Other

- K238R is a common polymorphism that does not reduce NNRTI susceptibility.
- V179I is a polymorphic mutation that is frequently selected in patients receiving ETR and RPV. But It has little, if any, direct effect on NNRTI susceptibility.
- S68G is a polymorphic mutation that is often selected in combination with K65R. It partially restores the replication defect associated with K65R.

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