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VIROLOGICAL RESPONSE TO ANTIRETROVIRAL THERAPY IN THE SETTING OF THE K65R MUTATION

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AB Nevins¹✉, SY Rhee¹, WJ Fessel², M Horberg², A Scarsella³, SY Lee³, RW Shafer¹, AR Zolopa¹

¹Stanford University, Stanford, CA, USA ²Kaiser-Permanente Medical Care Program, San Francisco, CA, USA ³Pacific Oaks Medical Center, Los Angeles, CA, USA

BACKGROUND: The impact of the K65R reverse transcriptase (RT) mutation on virological response to salvage therapy has not been fully defined. We performed a retrospective analysis of virological response to subsequent therapy in antiretroviral-experienced patients with K65R drawn from a large clinical cohort.

METHODS: We identified all K65R mutations in virus samples from three California clinical programs from the period 7/1/97 to 10/1/04. From 9060 isolates (6147 patients) we found 169 (144 patients) with K65R. Complete treatment histories were available for 98 patients, including 39 who had ≥ 1 plasma HIV-1 RNA measurement following a change in therapy based on the genotype.

RESULTS: Baseline characteristics. Patients were highly treatment-experienced. Previous treatment included tenofovir (TDF, 25/39 patients), didanosine (ddI, 28/39), abacavir (18/39), and lamivudine (36/39). Nineteen patients (49%) had used both TDF and ddI. Mutations M184V, Q151M, and L74V were present in 22 (56%), 7 (18%, three of whom also had M184V), and 2 (5%) of patients, respectively. At least one TAM was present in 17 patients (44%); five patients had ≥ 3 TAM's, three of whom also had M184V and two with Q151M. Five patients had no other known resistance mutations.

RESPONSE TO THERAPY: Thirty-nine patients changed therapy based on their genotype results; 36 (92%) were changed to a protease inhibitor-based regimen. Eleven new regimens included TDF. The median baseline viral load was 15 849 copies/ml. Overall we observed decreases in viral loads following a change in antiretroviral therapy: median changes observed: -1.3 log at 34 days, -1.7 log at 85 days, and -1.95 log at 157 days. Fourteen patients (36%) achieved plasma viral loads of <50 copies/ml. The

response observed was independent of previous antiretroviral use (including TDF and the combination of TDF/ddI), or any component of the new regimen. The response was independent of the presence of other resistance mutations.

CONCLUSIONS: Among this cohort of highly treatment-experienced patients with the K65R mutation, response to subsequent antiretroviral therapy was robust in spite of the presence of multiple resistance mutations in many of these cases. This response appeared independent of specific prior RT inhibitor use or specific components of the new regimen.

PRESENTING AUTHOR: AB Nevins

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