

Comparing the Binding Affinities and Dynamics of the Wild-type and Mutant (N88D, L90M) of Subtype C HIV-1 Protease

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Acquired Immunodeficiency Syndrome (AIDS) is responsible for millions of deaths worldwide each year. One of the major targets in anti-HIV therapeutics is protease inhibition. The Human Immunodeficiency Virus (HIV) protease has a crucial role in the reproductive process of the virus, and its inhibition would prevent the maturation and the spread of the virus to neighboring cells. The emergence of drug resistant mutants has become a dire problem in the treatment of HIV patients. The mechanism by which non-active site mutations cause resistance is not well understood. This study investigates and offers an explanation of how the non-active site mutations N88D and L90M reduce the drug efficacy of the protease inhibitor saquinavir.