Investigation of the Recovery Stroke and ATP Hydrolysis and Changes Caused Due to the Cardiomyopathic Point Mutations in Human Cardiac Beta Myosin

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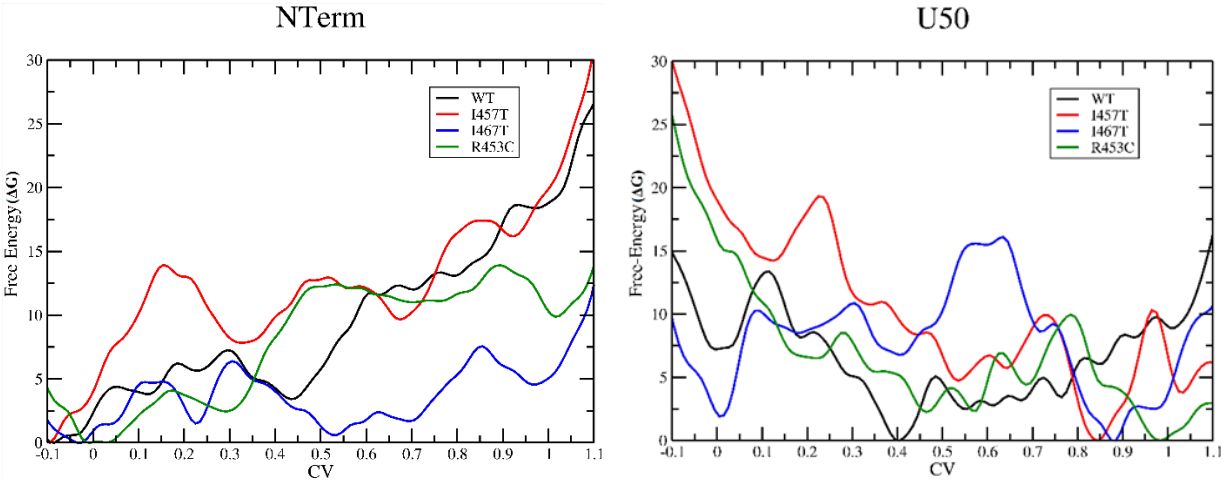
Supplemental Information:



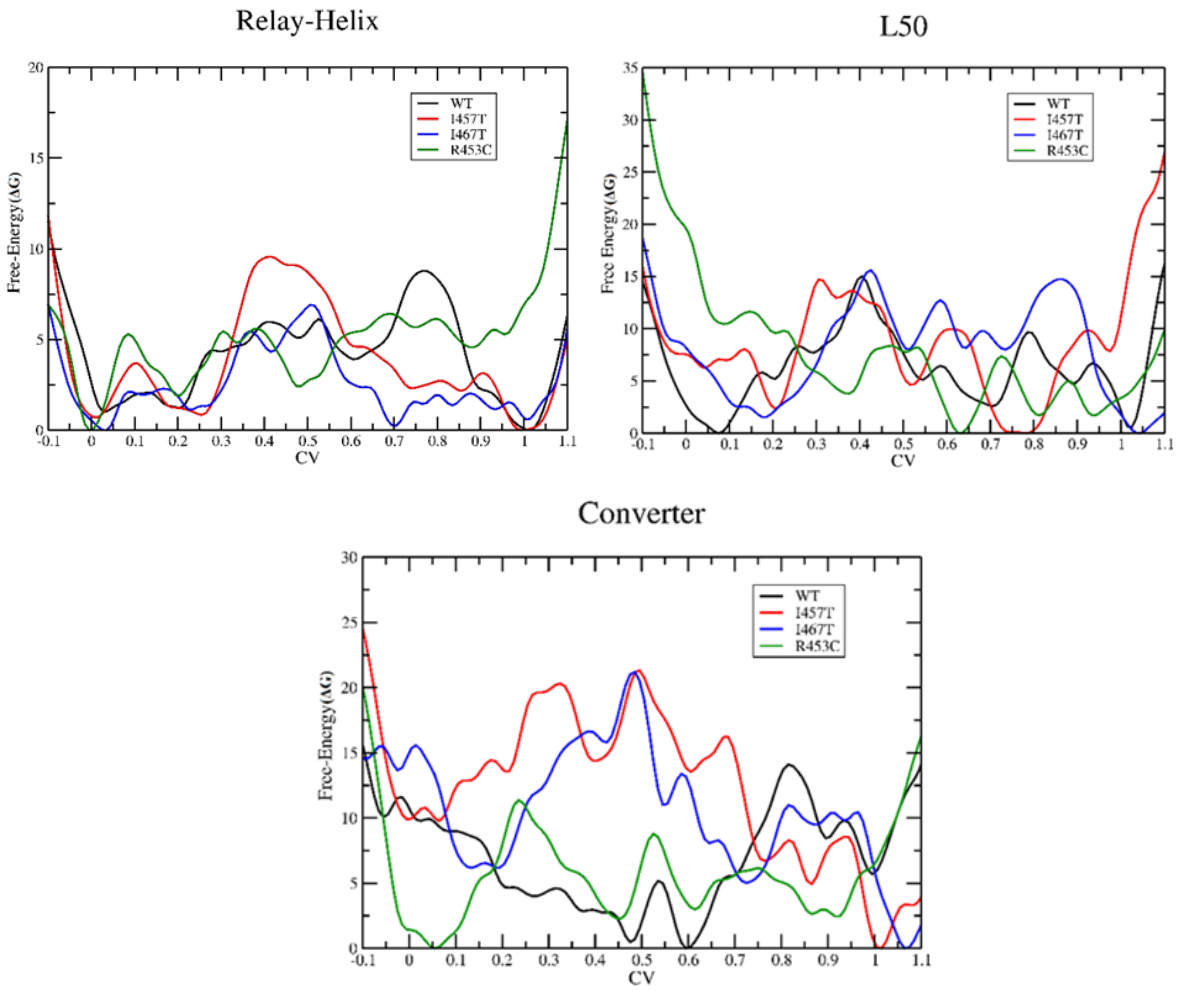


Figure S1: Converged FES profiles (in kcal/mol) for the (Top Left) Nterminal (residues 1 to 129), (Top Right) U50 (residues 130 to 460), (Middle Left) Relay-Helix (residues 461 to 505), (Middle Right) L50 (residues 506 to 710) and (Bottom) Converter (residues 711 to 782) domains for the recovery stroke.

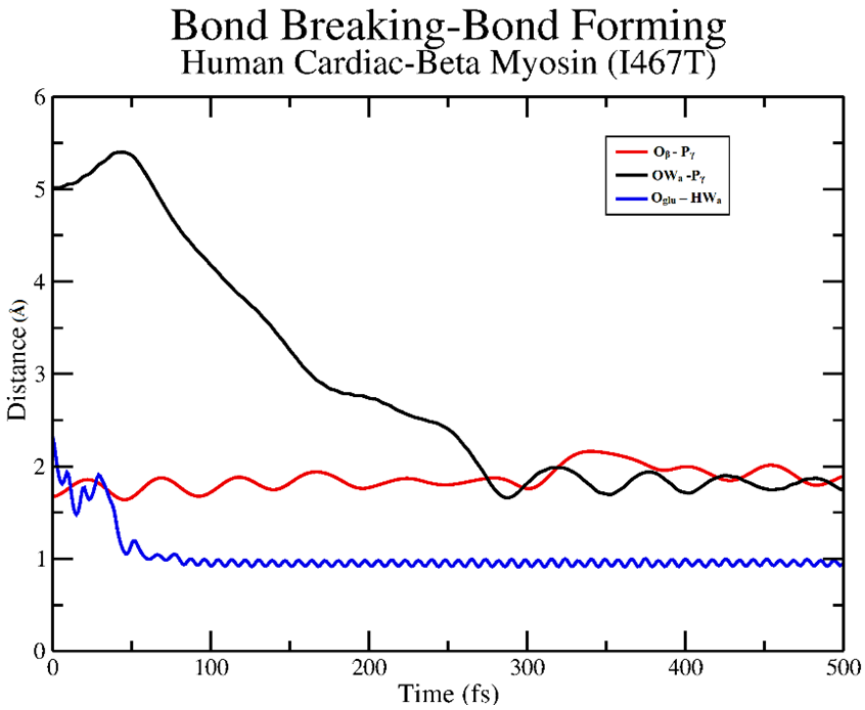


Figure S2. Plot of atomic distances for the gamma phosphate (Pγ), beta oxygen (Oβ) bond of ATP along with the deprotonation of the attacking water (HWa) by GLU466 (Oglu) and subsequent attack of the newly formed hydroxide (OWa) to the gamma phosphate from a representative trajectory for the mutated myosin (I467T).