THE Co-N BOND CLEAVAGE IN THE ADENOSYNCOBALAMIN COFACTOR IN ADVANCE TO GLUTAMATE MUTASE AND METHYLMALONYL-CoA MUTASE PROCESSES

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Abstract. The *in vivo* experiments show that the adenosylcobalamin cofactor in glutamate mutase and methylmalonyl-CoA mutase processes lose its dimethylbenzimidazole axial ligand before starting the enzymatic processes. CASSCF geometry optimization of the vitamin B12 active forms plus substrates joint models have been performed. These joint models include the adenosylcobalamin cofactor, the carboxyl negative ion model of the studied processes' active substrates and the histidine molecule. Partial electronic density is transferred from HOMO substrate molecular orbitals to the LUMO antibonding molecular orbitals, which consist of corrin ring and dimethylbenzimidazole ligand common molecular orbitals during the MCSCF molecular orbital mixing process. As a result, the Co-N axial bond is permanently elongated during the CASSCF geometry optimization until its complete rupture and until the removal of the dimethylbenzimidazole ligand from the central cobalt atom and the corrin ring is complete. The Co-N bond cleavage in the adenosylcobalamin cofactors in the studied processes is running as no energy barrier process under the influence of their active substrates and histidine molecule.