ABSTRACT

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has undergone genetic diversifications since the start of the coronavirus disease 2019 (COVID-19) pandemic, resulting in the appearance of novel variants. But the genetic markers underlying the prevalent variations still lack a precise characterization. A thorough analysis of the structural changes that distinguish each SARS-CoV-2 variation, concentrating on mutation profile of different SARS-CoV-2 variants and binding affinity of furin protease to the S-protein are needed, the S-protein binds to the angiotensin-converting enzyme 2 (ACE2), which is mostly expressed in the kidneys, testes, and lungs, and facilitates the SARS-CoV-2 virus invasion to the host cell. Proteases must cleave the S-protein for the binding process to take place. The distinctive polybasic "PRRAR" cleavage site on the SARS-CoV-2 S-protein was identified by the furin enzyme, evaluating the values of the binding of furin protease to different SARS-CoV-2 variants was observed to see how stable the complex, therefore knowing how severe SARS-CoV-2 invades the immune system, and varying conformational changes that are known to alter the viral infectivity and/or antigenicity. In this study, the binding of furin protease to different variants of SARS-CoV-2 was observed using multiple structural bioinformatics techniques by constructing a structural model of S-proteins, for the docking process of furin protease to different variants of SARS-CoV-2, for understanding the stability of the docking complex and knowing the varying binding affinity of the variants and for observation of deformability, (B-factor atom index and Eigenvalues). Conclusively, among the seven different variants that are analyzed, the Omicron variant showed the most stable binding to furin protease with a value of -69.7 kcal/mol compared to the original variant Wuhan -64.9, having enhanced rate of transmission and infectivity.