**Streszczenie rozprawy doktorskiej w języku angielskim:**

The aim of the doctoral thesis was to investigate the mechanism of infection by the SARS-CoV-2 coronavirus. The SARS-CoV-2 virus, causing the disease known as COVID-19, emerged in Wuhan, China, and became a threat to human health and life. With the announcement of the pandemic, the SARS-CoV-2 coronavirus sparked significant interest among scientists worldwide. A crucial aspect for public health was and still is understanding how the virus adapts to new conditions and evolves. The World Health Organization identified the most prevalent variants of the SARS-CoV-2 coronavirus, namely: alpha, beta, gamma, delta, and omicron. With the virus's evolution, the number of observed mutations in the receptor-binding domain (RBD) increased compared to the originally discovered wild type variant. The alpha variant has only one mutation; beta and gamma have three, while the omicron variant has fifteen. The delta variant appears to be a result of independent evolution, as it has two mutations in sequence locations different from previously observed cases. The virus evolution process and the occurrence of mutations can lead to significant changes in virion affinity for host cells, thereby increasing infectivity or altering the previously understood mechanism of virus-host complex formation.

In this doctoral thesis, I investigated the binding affinity of the human coronavirus SARS-CoV-2 to the ACE2 receptor in selected species (dogs, mice, bats, pigs, civets, and humans) using the UNRES force field. The second goal was to examine changes in the viral infection mechanism for the human ACE2 enzyme and the most prevalent SARS-CoV-2 variants, namely alpha, beta, gamma, delta, micron, and the first recognized variant (wild type). Based on the obtained results, I identified a transition state in which the virus-host complex is formed located on the extracellular side of the host's ACE2 receptor. I described segments of both ACE2 and RBD crucial for the mutual protein matching and the formation of a stable complex. I discussed interactions potentially responsible for the development of infection in the studied organisms, elaborated on energy dependencies, and differences in ACE2 structure leading to variable susceptibility to COVID-19 among animals. I also discussed differences in the interaction of individual virus variants with human ACE2, indicating amino acid residues with the greatest contribution to the formation of a stable RBD-ACE2 connection.

The results obtained indicate that the proper conformational matching of proteins is the first stage of infection, which can be simplified as a series of conformational changes leading to the release of progeny virions.