СЕКЦІЯ 4. МІКРОБІОЛОГІЯ ТА ВІРУСОЛОГІЯ

УДК 578

DETERMINATION OF VARIATIONS IN NEURAMINIDASE (NA), HEMAGGLUTININ (HA) AND NUCLEOPROTEIN (NP) GENES OF AVIAN INFLUENZA STRAINS H1N1 AND H7N9

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Avian influenza is an infectious disease caused by influenza virus type A. In many cases, avian influenza can cause severe diseases in birds, as well as irreversible processes. Hemagglutinin (HA), neuraminidase (NA) and nucleoprotein (NP) are important viral proteins identified in H1N1 and H7N9 strains of avian influenza. Studying the variability of genes such as hemagglutinin (HA), neuraminidase (NA), and nucleoprotein (NP) is critical to understanding the mechanisms of pathogenesis and adaptation of these viruses. Hemagglutinin, one of the main antigens of the virus, plays a key role in binding the virus to host cells and initiating infection. Its variability may affect the viruses ability to evade the immune response and adapt to new hosts. Neuraminidase, a second important antigen, promotes the release of viral particles from infected cells and thus plays an important role in the spread of infection. Variation in this gene may also influence sensitivity to antiviral drugs, making it an important target for study in the context of epidemiological risks and treatment resistance [1]. The nucleoprotein responsible for assembling viral particles and interacting with cellular machinery is also subject to mutations, which can affect the stability of the virus's genetic material. Studying variation in this gene can help understand how changes affect viral replication and pathogenicity [2]. In light of the above, analysis of the variability of the hemagglutinin, neuraminidase and nucleoprotein genes of the H1N1 and H7N9 strains is a relevant area of research that contributes to the understanding of the mechanisms of pathogenesis of these viruses. These proteins play critical roles in viral replication, virulence, and host range. Analysis of the amino acid sequences of the H1N1 and H7N9 viruses reveals key molecular differences that may influence their pathogenic properties and susceptibility to drugs. H1N1, known as the swine flu virus, exhibits high variability in its proteins, including hemagglutinin (HA) and neuraminidase (NA), which affects its ability to undergo antigenic drift. Structural changes in these proteins can lead to a deterioration of the immune response in the population and complicate the task of vaccination. H7N9, on the other hand, is a strain typically found in birds, with a potential threat to humans. Its amino acid profiles, especially in proteins responsible for cell adhesion and entry, highlight evolutionary adaptations to mammals, facilitating its zoonotic transmission. Comparative analyzes consistently show expressed positions in proteins based on virulence and antiviral drug resistance. This

is important for the development of external therapy and vaccination. PCR was used followed by sequencing and mutational analysis and comparison of the neuraminidase, hemagglutinin and nucleoprotein gene sequences. The amino acid sequence data of the H1N1 and H7N9 strains for the study were taken from the NCBI database. The research methods were the MEGA 5 (Molecular Evolutionary Genetic Analysis) program. Phylogenetic and amino acid sequence variation analyzes were performed. Research have identified several variations in the NA gene of avian influenza A(H1N1) strains. Amino acid substitutions in positions 225, 226 and 228 are associated with increased virulence and transmissibility of the virus. Variations in the NA gene have been associated with reduced susceptibility to antiviral drugs such as Oseltamivir and Zanamivir. Amino acid substitutions at positions 119 and 150 correlate with loss of resistance to these drugs. The NP protein is a conserved protein found in all influenza A viruses, including avian influenza H1N1 and H7N9. Variations in the NP gene were determined to affect virus replication and host range. A single amino acid substitution at position 100 was associated with increased replication and virulence of the virus in mammals. Hemagglutinin H7N9, compared to H1N1, shows changes in interstitial regions that affect its binding to animal and human cell receptors. This may be due to its pathogenicity and transmission ability. H7N9 neuraminidase has unique amino acid substitutions that may improve its activity under conditions common in avian and human infections, whereas H1N1 has a more stable profile consistent with its adaptation to human populations. nucleoprotein, both strains have differences in amino acid sequences that may affect viral replication and assembly. These adaptations highlight differences in virulence and transmission ability between strains. Thus, the amino acid profiles of these proteins reflect not only their functions, but also the evolutionary paths that the H7N9 and H1N1 viruses have taken. Variations of these genes are associated with increased pathogenicity and transmission of avian influenza viruses to humans. Therefore, understanding the genetic variation in these genes is important for the prevention and control of future infectious outbreaks. Our research allows us to better understand the evolution and spread of viruses, provides new data for the creation of vaccines and the development of methods for controlling avian influenza.

References

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