Journal for Research in Applied Sciences and Biotechnology

www.jrasb.com

ISSN: 2583-4053

Volume-3 Issue-4 || August 2024 || PP. 170-176

https://doi.org/10.55544/jrasb.3.4.20

An Overview about the Influenza a Virus

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www.jrasb.com || Vol. 3 No. 4 (2024): August Issue

Received: 21-08-2024	**Revised:** 26-08-2024	**Accepted:** 10-09-2024

ABSTRACT

As influenza A can cause pandemics and epidemics with high rates of morbidity and mortality, it continues to be a major worldwide health concern. It is critical to comprehend its development, implications, and upcoming difficulties. This review explores the historical background, molecular causes, and modern management approaches for influenza A. Influenza A presents difficulties due to its rapid mutation rates and capacity to infect a wide variety of hosts. Even if vaccination is the main preventive measure, existing tactics have drawbacks that call for the creation of novel solutions. The overview covers the molecular mechanisms underpinning influenza A's pathogenicity, as well as the virus's historical history and effects on human health. It also looks at modern methods of treating influenza A, such as antiviral medication and immunization. To lessen the threat posed by influenza A, the review looks ahead, highlighting future research challenges and initiatives. It emphasizes the significance of enhancing global surveillance efforts, developing novel treatment options, and improving vaccine responses.

Keywords- Influenza A, Evolution, Impact, Pathogenesis, Epidemiology, Prevention, mutation, global health.

I. INTRODUCTION

Generally, Influenza is a highly contagious disease that represents one of the most serious health and economic threats to humans and animals worldwide. In order to understand the epidemiology of influenza, it is critical to recognize that influenza A viruses infect a wide variety of species.[4] Rarely, there are global influenza A pandemics linked to the erratic appearance of novel influenza viruses. Subtypes of viruses There have been three pandemics this century. [6] the influenza virus is classified as an Orthomyxoviridae family. It is an encapsulated virus with a negative sense single-stranded RNA genome that is divided into segments. The eight segments of influenza A viruses are hemagglutinin (HA), neuraminidase (NA), matrix 1 (M1), matrix 2 (M2), nucleoprotein (NP), non-structural protein 1 (NSP1), non-structural protein 2 (NS2; also known as nuclear export protein, NEP), polymerase acidic protein (PA), polymerase basic protein 1 (PB1), polymerase basic protein 2 (PB2), and polymerase basic protein 1 –F2 (PB1–F2).[1] It has long been recognized that influenza A viruses (IAVs) may infect a variety of host species, including people and animals. IAVs are zoonotic diseases that may infect a wide variety of species, including humans, pigs, and birds. IAVs must adapt to their new host to spread between species, and their rapid mutation rates and ease of reassortment make this process easier. [2] Influenza A viruses are pathogens of major concern in veterinary and public health. They are highly variable, owing to their segmented genome and lack of proofreading during replication. Influenza A viruses can change into strains that cause moderate to severe illnesses when they infect domestic poultry, pigs, or humans.

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ISSN: 2583-4053

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While all 16 hemagglutinin (HA) subtypes of influenza A viruses can replicate in domestic ducks or quail, only a limited number can infect chickens.[3] Placed in its ecological niche _ the dabbling ducks _influenza A virus is a benign disease. However, it is a very adaptable virus, and it has been able to infect and adapt to a wide range of hosts [5]

II. MATERIAL AND METHODS

A comprehensive review of existing literature on Influenza A virus was conducted using databases such as PubMed, Scopus, and WHO reports, covering publications up to October 2023. The search focused on the virus's molecular pathogenicity mechanisms, historical outbreak data, mutation rates, host range, current management strategies, and vaccination efficacy. Relevant studies were selected based on their contribution to understanding the evolution, impact, and control measures of Influenza A. Data were synthesized and analyzed to identify gaps in current knowledge and to propose future research directions aimed at improving prevention and treatment approaches.

III. RESEARCH RESULT

The article gives a thorough description of the influenza A virus, going into its molecular processes, history, and current control measures.[15] It draws attention to the virus's quick pace of mutation, which enables it to infect other animals and pose constant threats to public health.[18] The essay questions the efficacy of the present vaccination campaigns and highlights the necessity for creative remedies to properly contain the virus.[11] To lessen the effects of this rapidly spreading infection, the study further emphasizes the significance of international surveillance and urges better immunization responses and public health readiness.[13]

Evolution of influenza A: Ancient Origins to Modern Challenges:

Hippocrates reported a possible influenza-like sickness condition known as the "fever of Perinthus" or "cough of Perinthus" in 412 BC in the "Book of Epidemics." Although some academics argue that this is most likely the earliest historical account of influenza A. [9] The influenza virus is the causative agent of acute respiratory illnesses in mammals and several other domestic poultry. influenza viruses continued to increase recurring seasonal endemics and potentially fatal pandemics even after they were first discovered in 1918. Consequently, there is an urgent need for efficient antivirals and vaccinations against influenza, which causes between 290 and 650 thousand fatalities annually. Depending on how aggressive the viruses are, fever, coughing, headaches, fatigue, sore throat, runny nose, and body pains are among the symptoms of an influenza infection.[8] For at least the last several centuries, the influenza virus has been the source of repeated epidemics of acute febrile illness every one to four years. Although there were reports of influenza–like sickness in 1173–743, the first confirmed pandemic wasn't recorded until 1694.4 Between 1918 and 1919, the globe saw the worst epidemic on record, with an estimated 21 million people dying globally. Among the bloodiest incidents in recorded human history was this one. then, there were three further pandemics in the 20th century: the H2N2 pandemic in 1957, the H3N2 pandemic in 1968, and the influenza A (H1N1) virus (pH1N1) In the most recent incident, an influenza strain was discovered in Mexico and subsequently in the United States of America. This strain had a mix of gene segments not before documented in the swine or human influenza virus strains. Six In August 2010, with the participation of several nations, the epidemic was deemed to be finished.[7]

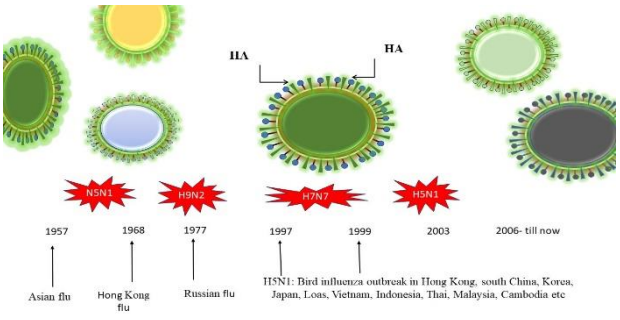

Figure 1 - displays the evolution of influenza A virus.

Impact of Influenza A: From Symptoms to Societal Burden:

An acute respiratory disease with high transmissibility that has affected people from prehistoric times and has roots comparable to modern influenza epidemics is revealed by the historical record. It is commonly known that seasonal and pandemic influenza viruses are seasonal and have the capacity to spread globally. Epidemiological investigations have demonstrated infections that are clearly visible in a variety of groups and environments, such as convalescent homes, cruise ships, medical facilities, and school residences. Depending on the virus and individual, the components of infections might be random, epidemic, pandemic, or periodic. Type A or type B influenza viruses are responsible for most human illnesses.[11] Sneezing, fever, myalgia, rhinorrhea, and sore throat are the symptoms of an IAV infection. Viral shedding peaks at days 2-3 after infection while symptoms peak 3-5 days after infection. The upper respiratory tract, which includes the nasal, tracheal, and frequently bronchial epithelium, is the only site of infection for human IAVs. In more extreme situations, an infection of the lung and lower respiratory tract (LRT) develops, frequently with serious aftereffects that necessitate hospitalization. The emergence of viral pneumonia and subsequent bacterial infections may result in acute respiratory distress syndrome (ARDS), acute lung injury (ALI), and ultimately, mortality (32). Obese individuals are more

ISSN: 2583-4053

Volume-3 Issue-4 || August 2024 || PP. 170-176

https://doi.org/10.55544/jrasb.3.4.20

likely than non-obese people to experience severe sequelae and infection progression to the LRT, which impairs infection resolution and recovery. [10] However, death is a very uncommon consequence of influenza infection and only makes up a small portion of the population's overall health burden from influenza [2, 9, 10]. Furthermore, even while influenza A(H3N2) outbreaks often lead to a rise in unnecessary hospital admissions and fatalities.[12] It is widely known that influenza outbreaks can lead to increased adult morbidity and death. 2. There is insufficient population-based evidence to quantify the amount of excess morbidity (beyond average rates of acute respiratory viral illness) that occurs among children during influenza epidemics, despite the fact that several surveys have shown high influenza attack rates among children during epidemics3–7.[13] during the 2009 H1N1 pandemic, it was expected that a higher proportion of pregnant women would be affected during the 2009 H1N1 pandemic than in previous influenza seasons. Early evidence during the 2009 H1N1 pandemic showed that pregnant women were disproportionally represented among hospitalized, intensive care unit (ICU)- admitted cases and deaths due to influenza.[14] also numerous surveys have demonstrated high influenza attack rates among children during epidemics3-7; however, there is little population-based data to estimate how much excess morbidity (over usual rates of acute respiratory viral disease) occurs among children during influenza epidemics. Because children, in contrast to adults, are particularly susceptible and exposed to several other acute respiratory virus infections, an epidemic of influenza may show less of an incremental impact upon childhood morbidity rates than has been the case among adults. Among children, those with high-risk conditions are at greatest risk of complications from acute influenza.[18]

Insights into Influenza Virus Pathogenesis and Epidemiology:

Numerous strategies have been developed by pathogenic viruses to impede the RIG-I signaling system and reduce the host's innate immune response. Non-structural protein 1 (NS1) of influenza is one instance. IAVs are versatile virulence factors that bind a variety of host proteins during infection, including TRIM25NS1. Their structural makeup consists of an unstructured C-terminal tail and an RNA-binding domain (RBD, a.a. 1–73), followed by a short linker to the effector domain (ED, a.a. 85–202). The conformational flexibility of the RBD and ED architecture, the interaction with dsRNA and host proteins, and the self-association of NS1 have all been clarified by the several structures of individual domains and two structures of full-length NS1.[15] other from that Influenza A and B viruses belong to the Orthomyxoviridae family and have an irregular shape and pleomorphism, with a diameter of 80-120 nm. They have eight segments of negative sense RNA. They consist of two glycoprotein molecules, neuraminidase (NA) and haemagglutinin (HA), inserted inside a lipid membrane. The rod-shaped polypeptide molecules of HA produce triangular trimers that emerge as spikes from the surface of the virus. During viral maturation, HA cleaves into HA1 and HA2, which is required for virus infectivity. Enzymes in the respiratory system, where the virus replicates, usually speed it up. Four NA molecules combine to create a tetramer with a contrasting morphology, forming a square, box-shaped head that rests at the end of a long, thin stalk. Its extremity is enmeshed in the lipid layer of the viral membrane.[16] In order to understand the epidemiology of influenza, it is critical to recognize that influenza A viruses infect a wide variety of species. Moreover, the viruses exhibit only partial restriction of their host range such that viruses from one species can occasionally transmit to infect another species (Webster et al., 1992; Webby and Webster, 2001). Historically, only a limited number of subtypes of influenza viruses have been associated with widespread infection of mammals (Webster et al., 1992; Alexander and Brown, 2000). However, viruses of all 16 hemagglutinin (HA) and nine neuraminidase (NA) subtypes have been recovered from wild waterfowl and seabirds (Webster et al., 1992; Webby and Webster, 2001). As such, waterfowl provide a vast global reservoir of influenza viruses in nature from which novel viruses can emerge to infect mammalian species (Webster et al., 1992; Webby and Webster, 2001). Undoubtedly, the most prominent examples of direct transmission of avian viruses to mammalian species are the recent infections of humans and cats with the highly pathogenic avian H5N1 viruses (de Jong et al., 1997; Claas et al., 1998b; Kuiken et al., 2004; Webster et al., 2005). Yet, while these examples clearly demonstrate that cross species transmission of viruses can occur, it has long been recognized that barriers exist that limit transmission of influenza viruses among species. [20]

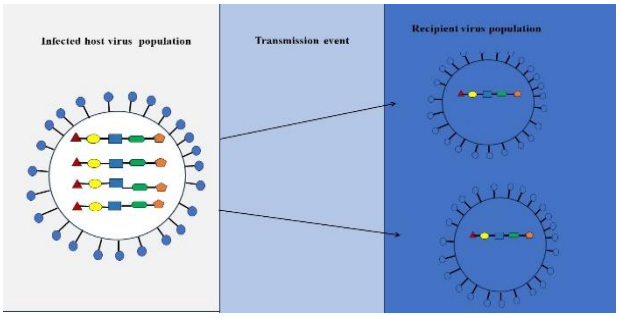

Figure 2 - displays transmission of influenza A virus

In 2013, zoonotic infections with LPAIV H7N9 were first reported in China. The human pathogenic H7N9 is derived from multiple reassortments between AIV H7N9 (NA), H7N7 (HA), and H9N2 (internal proteins coding gene segments) in domestic ducks and chickens. The virus is still maintained in poultry leading only to sporadic human infections. In February 2017, the Chinese province Guangdong reported the first human infection with the mutated trypsin independent HPAIV H7N9.

Journal for Research in Applied Sciences and Biotechnology

www.jrasb.com

ISSN: 2583-4053

Volume-3 Issue-4 || August 2024 || PP. 170-176

https://doi.org/10.55544/jrasb.3.4.20

Recent studies revealed that the newly emerging HPAIV H7N9 viruses acquired the internal protein-coding genes from co-circulating H9N2 strains. Despite the higher viral polymerase activity, increased replication efficiency and pathogenicity in human, no clear impact on viral transmissibility or virulence was noticed for HPAIV H7N9[28].

Table 1: Confirmed human infections/fatalities with LPAIV- or HPAIV H7N9 per wave.

Wave Number	Period	Phenotype	Cases	Fatalities	Fatality Rate
1	February 2013–September 2013	LPAIV	134	45	0.34
2	October 2013–September 2014	LPAIV	306	131	0.43
3	October 2014–September 2015	LPAIV	219	102	0.47
4	October 2015–September 2016	LPAIV	1114	47	0.41
5	October 2016–September 2017	LPAIV/HPAIV	848	295	0.35
6	Since October 2017	LPAIV/HPAIV	4	3	0.75

Current approaches to control influenza A:

Influenza virus vaccines are the cornerstone of public-health efforts to reduce the burden of seasonal influenza and to respond to the unpredictable emergence of pandemic influenza. Current strategies for generating seasonal influenza vaccines and for influenza vaccine pandemic preparedness, however, are far from optimal. Seasonal influenza vaccines are strain-specific and not designed to provide broad protection against the continual evolution of influenza viruses. [17] Antiviral medications and immunization are the mainstays of the management of influenza illness. However, creating a novel antiviral strategy is severely hampered by the ongoing influenza mutations that impart host immune evasions and medication resistance. Currently, three drug classes have been approved to block the influenza virus's life cycle: polymerase acidic (PA) endonuclease inhibitors (Baloxavir Marboxil), neuraminidase inhibitors (NAI; Peramivir, Zanamivir, Oseltamivir, and Laninamivir), and M2 proton channel antagonists (amantadine, rimantadine). The latter has evolved into a new class of antivirals over time. To lessen the effects of IAV, further options such RNA-based therapies and medication repositioning are also covered.[19]

The most economical method of preventing influenza is vaccination. Both influenza A and influenza B can be prevented by vaccination, and it still has benefits even in cases where there is a poor match between the vaccine and the strain that is circulating. Seasonal chemoprophylaxis is a costly but effective treatment that is best used when there is an impending influenza exposure. For example, it can be used for two weeks while controlling an epidemic in an institution or for ten days if a patient is cohabiting with someone who has the flu. Antiviral therapy should only be administered to patients who exhibit signs of infection, and it should start 48 hours after symptoms appear. Only the influenza A virus can be defeated by M2 channel inhibitors, which are linked to the fast growth of influenza A virus and are exclusively effective against it. [21]

IV. ADVANCEMENTS AND CHALLENGES IN INFLUENZA RESEARCH AND PREVENTION

It has been a century since the 1918 Spanish flu pandemic, which was brought on by the influenza A(H1N1) virus (Boxes 1 and 2). Subsequently, A(H2N2), A(H3N2), and A(H1N1) pdm09 viruses have been responsible for three further pandemics: the 1957 Asian flu, the 1968 Hong Kong flu, and the 2009 swine-origin flu, respectively1. At the moment, influenza B viruses from the Yamagata and Victorian lineages, together with A(H1N1) pdm09 and A(H3N2) viruses, create epidemics associated with seasonal influenza; however, A(H1N1) and A(H2N2) viruses have vanished.[23] Smorodintseff et al. presented the first influenza challenge trial that was thoroughly documented in 1937. 72 volunteers were infected by the authors using a human influenza virus that was kept alive by passing it through ferrets and mice (Smorodintseff et al., 1937). It was found that only about 20% of the population experienced moderate illness. After being found to be safe, the model was used for many years to study immunological reactions to influenza and evaluate treatment and prevention strategies.[22] Even though influenza vaccination has greatly reduced influenza-related morbidity and mortality worldwide, new technologies and approaches are urgently needed to enhance influenza vaccine responses in order to produce broader and more durable protective immunity, especially in those who are susceptible to vaccine failure. Particularly in developed countries, the proportion of the population that should react adversely to immunizations is growing. While there are now strategies in place to improve vaccine responses for some people at risk—like the elderly.[24] There are currently few therapy options available to treat influenza virus infections. Only 2 classes of agents have licensed products, neuraminidase (NA) inhibitors and M2 inhibitors, and only NA inhibitors are active against currently circulating seasonal viruses. While observational studies show a benefit of NA inhibitor treatment for hospitalized patients with

ISSN: 2583-4053

Volume-3 Issue-4 || August 2024 || PP. 170-176

https://doi.org/10.55544/jrasb.3.4.20

influenza A. [25] At the moment, three different kinds of vaccine development platforms are being used worldwide: recombinant-HA vaccine, live-attenuated influenza vaccine, and inactivated influenza vaccine. There are benefits and drawbacks to these vaccinations. Immunological and cellular effectors, such as cytotoxic CD8+ T-lymphocytes that kill infected cells and stop them from spreading throughout the body and antibodies made by B lymphocytes that can bind specifically to viral antigens, can be activated by vaccines. Given that defense against the fu is just momentary.[26] re-clinical and clinical research on influenza vaccines has concentrated on novel immunogens that mimic evolutionarily conserved, functionally constrained portions of the virus or that improve the recruitment of cell-mediated or mucosal immune responses, as well as novel delivery platforms such as virus-like particles, viral vectors, nucleic acids, peptide epitopes, and nanoparticles. These novel strategies might greatly increase the efficacy of influenza vaccinations in the future.[27]

V. CONCLUSION

In general, further study of influenza A is essential for public health preparedness. Influenza A is still a serious concern despite advances, with recurrent epidemics and occasional pandemics causing a great deal of damage. To successfully control influenza A, future efforts must concentrate enhancing vaccination responses, investigating novel treatments, and bolstering international surveillance. By improving our knowledge and readiness, we can lessen its effects and safeguard world health. Working together, researchers, medical experts, and legislators can effectively handle the complex issues that influenza A presents. The early identification and control of new influenza strains depend heavily on enhanced public awareness and international collaboration. Furthermore, obtaining a wider and more durable protection against influenza A necessitates investing in cutting-edge technology and distribution tactics for vaccine. Through adoption of these strategies and promotion of a proactive approach to influenza management, we can mitigate the global burden of influenza-associated disease and mortality. We can ensure the developing a strong defense against future outbreaks via persistent efforts and coordinated action.

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