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Molecular Characterization of an Avian-origin Reassortant H7N1 Influenza Virus

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Recently, sporadic cases of human infection by genetic reassortants of H7Nx influenza A viruses have been reported; such viruses have also been continuously isolated from avian species. In this study, A/wild bird/South Korea/sw-anu/2023, a novel reassortant of the H7N1 avian influenza virus, was analyzed using full-genome sequencing and molecular characterization. Phylogenetic analysis showed that A/wild bird/South Korea/sw-anu/2023 belonged to the Eurasian lineage of H7Nx viruses. The polymerase basic (PB)2, PB1, polymerase acidic (PA), and nucleoprotein (NP) genes of these viruses were found to be closely related to those of avian influenza viruses isolated from wild birds, while the hemagglutinin (HA), neuraminidase (NA), matrix (M), and nonstructural (NS) genes were similar to those of avian influenza viruses isolated from domestic ducks. In addition, A/wild bird/South Korea/sw-anu/2023 also had a high binding preference for avian-specific glycans in the solid-phase direct binding assay. These results suggest the presence of a new generation of H7N1 avian influenza viruses in wild birds and highlight the reassortment of avian influenza viruses found along the East Asian–Australasian flyway. Overall, H7Nx viruses circulate worldwide, and mutated H7N1 avian viruses may infect humans, which emphasizes the requirement for continued surveillance of the H7N1 avian influenza virus in wild birds and poultry.

Key words : Avian influenza A virus, eurasian lineage, H7N1, virus reassortment, wild birds

Introduction

Avian influenza viruses (AIVs) belong to *Orthomyxoviridae* family; they are single-stranded negative-sense viruses with eight-segment genomes. AIVs can be antigenically classified into several subtypes on the basis of two surface glycoproteins: hemagglutinin (HA; H1-H18) and neuraminidase (NA; N1-N11) [19]. All the AIV subtypes, except H17, H18, N10, and N11, have been detected in wild aquatic birds, especially those belonging to the orders *Anseriformes* and *Charadriiformes*. Therefore, these wild birds are thought to be AIV reservoirs for various animal influenza infections that circulate via bird migration.

AIVs can be divided into the following categories on the basis of their pathogenic properties in chickens: highly pathogenic avian influenza (HPAI) and low-pathogenic avian influenza (LPAI) viruses. Among the several subtypes, some H7 AIV strains can be highly pathogenic in chicken flocks and infect humans sometimes as well [3]. Low-pathogenic H7 subtype viruses may evolve into HPAI viruses via glycosylation and amino acid mutations in poultry [4]. In 1999, H7N1 HPAI outbreaks were caused by low-pathogenic H7N1 viruses circulating in northern Italy, which showed additional glycosylation, an unusual multibasic cleavage site (MBCS) in the HA protein, and NA stalk deletion [2]. Since 2003, reports of H7Nx outbreaks, including H7N3 [17], H7N7 [5], and H7N9 [10] subtype outbreaks, in humans have increased. In particular, after the first outbreak in 2013, H7N9 infection continued to have severe clinical impact, such as pneumonia and acute respiratory distress syndrome, in humans in China; however, these viruses caused mild or no symptoms in chicken flocks. Therefore, owing to continuous H7 virus evolution, we have retained focus on the threat posed by possible H7 subtype AIV-related pandemics to humans and the poultry industry. In this study, we isolated and genetically characterized A/wild bird/South Korea/sw-anu/2023, a novel H7N1 AIV obtained from wild bird feces collected in South Korea during the 2022-2023 winter season.

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Materials and Methods

AIV isolation

During the 2022-2023 winter season, approximately 250 fecal samples of migratory birds were collected from migratory bird sites in Gyeongsang Province (GPS: 35.34.11.60 [N], 128.24.07.7[E]), South Korea. All the samples were placed in universal viral transport medium (BD Diagnostics, USA) and transported to the laboratory for analysis. For viral isolation, the samples were inoculated into 10-day-old embryonated specific-pathogen-free (SPF) eggs and incubated for 72 hr at 37℃ under humid conditions. The presence of AIV in the allantoic fluid was confirmed using a hemagglutination (HA) assay performed using 0.5% chicken red blood cells in phosphate-buffered solution (PBS, pH 7.4). One out of the 250 fecal samples were finally found positive for the intact AIV. Stock viral titers were determined using 50% tissue culture infectious dose (TCID50) and 50% egg infectious dose (EID50) titrations.

Phylogenetic and genetic analyses of the AIV

For phylogenetic and genetic analyses of A/wild bird/South Korea/sw-anu/2023, viral RNA was extracted from the allantoic fluid of HA activity–positive eggs by using the QIAamp Viral RNA Mini Kit (Qiagen) in accordance with the manufacturer's instructions. RNA was reverse-transcribed to cDNA by using the PrimeScript™ first-strand cDNA Synthesis Kit (Takara, Japan) according to the manufacturer's protocol, along with the Unit 12 primer (AGCAAAAGCAGG). The eight viral segments were amplified by polymerase chain reaction (PCR) performed using Premix Taq (Takara) and previously described primers. Nucleotide sequences were obtained by direct sequencing performed using an ABI 3730XL DNA analyzer (Applied Cosmo Genetech, South Korea). The sequences were manipulated in BioEdit and assembled using CLC Sequence Viewer 6.7. Multiple alignments of the full coding nucleotide sequences were performed using the MUSCLE algorithm.

Receptor-binding preference

To determine the receptor-binding preference of A/wild bird/South Korea/sw-anu/2023, a solid-phase direct-binding assay was performed a2,3′SL (Neu5Aca 2-3Galβ 1-4Glcβ-PAA-Biotin) and α2,6′SL (Neu5Aca 2-6Galβ 1-4Glcβ-PAA-Biotin). In brief, each virus (64 hemagglutinating units) was added to fetuin (Sigma, USA)-coated 96-well microtiter plates and incubated at 4℃ overnight. After removal of the virus, each biotinylated glycan was incubated at 4℃ for 2 hr and washed four times with ice-cold PBS. Subsequently, horseradish peroxidase (HRP)-conjugated streptavidin (1000-fold dilution with PBS) was added to each well of the plates, and the mixture was incubated at 4℃ for 1 hr. Next, the mixture was incubated with 0.05 ml 3,3′,5,5′-tetramethylbenzidine (TMB) substrate, following which the reaction was stopped with 0.05 ml of 50 mM Hydrogen chloride (HCl). Optical density was measured at 450 nm by using a Synergy 2 multimode microplate reader. A low-pathogenic H5N2 AIV and human H1N1 influenza virus were used as controls [18].

Replication kinetics

For *in vitro* analysis of viral replication kinetics, Madin-Darby canine kidney (MDCK) cells were infected at a multiplicity of infection (MOI) of 0.01 for each virus. MDCK cells were washed once before infection with the infection medium (MEM containing 2% BSA, penicillin/streptomycin, amino acids, and TPCK-treated trypsin). At various time points post-infection (p.i.), an aliquot of the culture supernatant was removed and virus titers were determined using the Reed-Muench method [14].

Results

Phylogenetic analysis of A/wild bird/South Korea/sw-anu/2023

During the 2022-2023 winter season in South Korea, the avian-origin H7N1 influenza A virus was isolated from wild bird feces collected from Gyeongsang Province of Korea and isolated from 9-11-day-old embryonated SPF eggs. The complete genome lengths of the eight segments containing polymerase basic (PB)2, PB1, polymerase acidic (PA), HA, nucleoprotein (NP), NA, matrix (M), and nonstructural (NS) were 2280, 2274, 2151, 1713, 1497, 1410, 982, and 838 nucleotides, respectively. The full genome sequences of the H7N1 influenza virus isolated have been deposited in GenBank under accession numbers OQ579561 to OQ579568.

Phylogenetic analysis based on the nucleotide sequences was performed with Molecular Evolutionary Genetics Analysis 6 (version 6.06), using the neighbor-joining method with 1000 replicates. Reference strains were derived from influenza virus resources available from the National Center for Biotechnology Information (NCBI). The eight genes of A/wild bird/South Korea/sw-anu/2023 were found to belong to Eurasian lineages of the H7 AIV. The HA segment of A/wild bird/South Korea/sw-anu/2023 was genetically closest to that

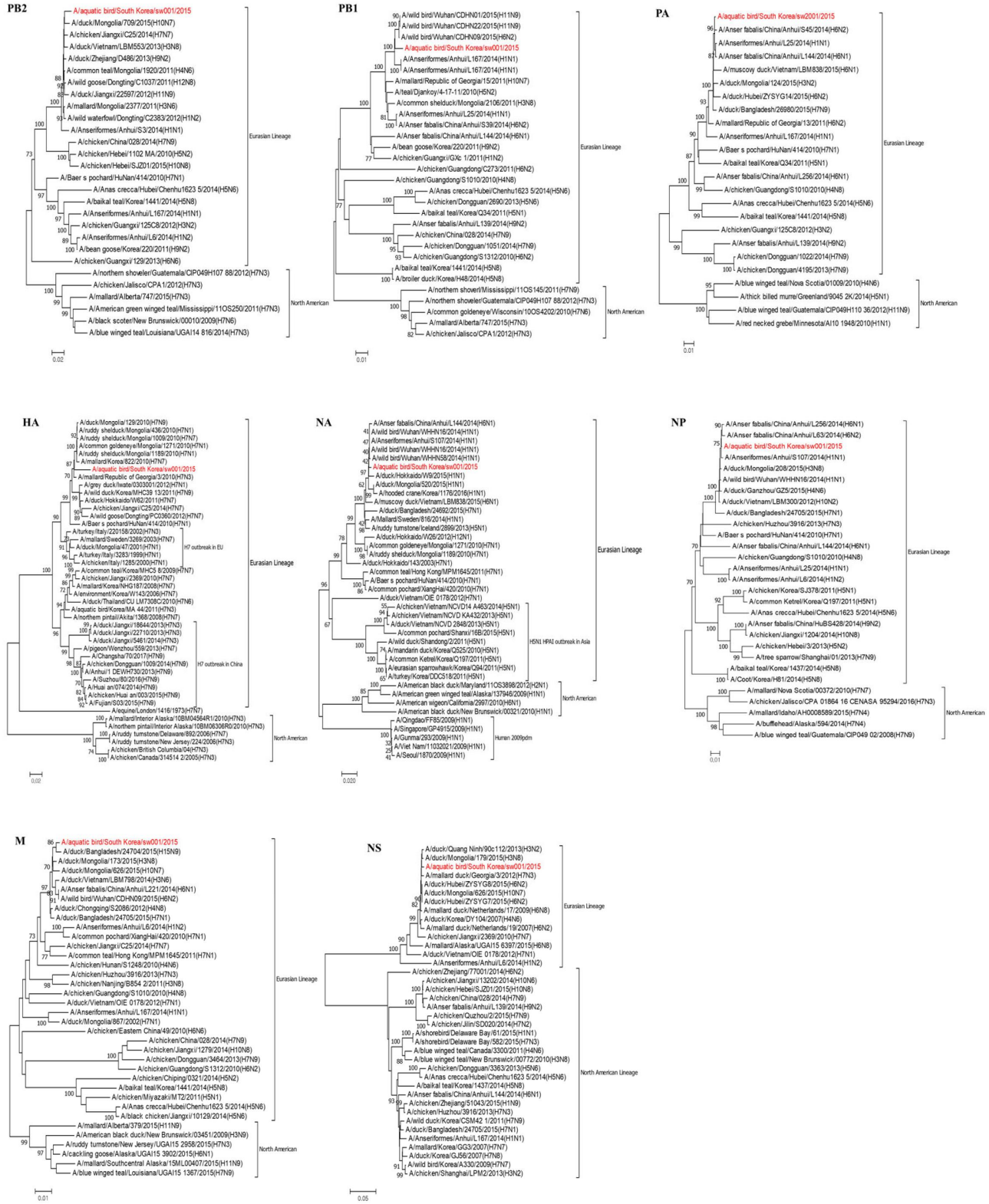

Fig. 1. Phylogenetic analysis of A/wild bird/South Korea/sw-anu/2023 virus. Phylogenetic analysis based on nucleotide sequences was constructed with neighbor joining method with 1000 replicates in Molecular Evolutionary Genetics Analysis 6 (MEGA6, version 6.06). Reference strains were derived from Influenza virus resource in National Center for Biotechnology Information (NCBI).

of the avian H7N9 subtype A/duck/Mongolia/129/2010 (97% identity at the nucleotide level); however, the NA segment might have been derived from the avian H1N1 subtype A/duck/Hokkaido/201/2014 (99% identity at the nucleotide level) (Fig. 1).

Molecular characterization of A/wild bird/South Korea/sw-anu/2023

Molecular characterization of HA revealed that the cleavage site sequence of HA from A/wild bird/South Korea/sw-anu/2023 was PELPKGR↓GLF, which exhibited LPAI properties. A previous study described the importance of amino acids at positions V186N, E190D, and Q226L in HA (H3 numbering) at receptor-binding sites, which may contribute to AIV virulence in the mammalian host [10]. Molecular analysis of the HA gene confirmed that A/wild bird/South Korea/sw-anu/2023 did not contain the major residues for mammalian receptor-binding preference, indicating that it had an a(2,3)-linked sialic acid receptor-binding preference. A/Anhui/1/2013 (H7N9) and A/wild bird/South Korea/sw-anu/2023 contain the S138A substitution in the HA protein, which may have increased the affinity for a(2,6)-linked sialic acid receptors, subsequently leading to mammalian adaptation in mammalian hosts [16]. AIVs typically possess a glutamic acid residue encoded by the PB2 protein at amino acid 627. However, human influenza viruses, including the 2009 pandemic H1N1 and the 2013 human H7N9 influenza virus, possess lysine at this position. Previous studies showed that the PB2-E627K mutation in H7N7 AIV [6] and the adaptation of H7N1 AIV [21] contributed to a highly pathogenic phenotype in mammalian hosts. In our study, A/wild bird/South Korea/sw-anu/2023 did not contain mammalian adaptation-related mutations in the PB2 segment at position 627, indicating that the polymerase complexes were avian-like. To analyze susceptibility to antiviral activity, we examined amino acid substitutions in the NA and M1 of A/wild bird/South Korea/sw-anu/2023. The NA gene of the A/sw001/15(H7N1) AIV has been previously shown to have histidine at position 275, which confers susceptibility to NA inhibitors such as oseltamivir [1]; A/wild bird/South Korea/sw-anu/2023 has been found to have serine at position 31 of M2, which confers susceptibility to ion-channel inhibitors such as amantadine and rimantadine [18]. In addition, NS1-S42 is highly conserved in the human influenza virus, which is related to increased virulence in the mammalian host [9]. The A/wild bird/South Korea/sw-anu/2023 virus isolated had a proline residue at position 42 of the NS1 protein, indicating that it might have low pathogenicity in mammalian hosts.

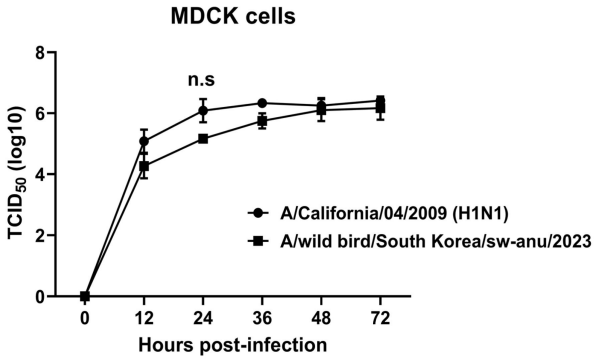

Fig. 2. Growth kinetics of A/wild bird/South Korea/sw-anu/2023 virus in MCDK cells. Replication of the A/aquatic birds/South Korea/sw001/2015 (H7N1) virus was monitored in MDCK. Cells were infected with virus at 0.01 MOI and infected culture supernatants were collected at different time points. Virus titers are shown as means ± standard error (SE) in bar graphs [not significant (n.s)].

Replication of A/wild bird/South Korea/sw-anu/2023

To determine the growth characteristics of A/wild bird/South Korea/sw-anu/2023 in mammalian cells, we measured its growth kinetics in MDCK cells. Similar titers were achieved for both A/California/04/2009 (H1N1) and A/wild bird/South Korea/sw-anu/2023 in the MDCK cells. Compared to A/California/04/2009 (H1N1), A/wild bird/South Korea/sw-anu/2023 had delayed kinetics 24 hr p.i. (Fig. 2b), but there were no significant differences between their replication efficacies.

Receptor-binding preference of A/wild bird/South Korea/sw-anu/2023

The binding affinity of HA for human receptors is an important factor for successful transmission in mammalian species. Therefore, we assessed the receptor-binding preferences of A/wild bird/South Korea/sw-anu/2023. To analyze the characteristics of the glycan-binding specificity of A/wild bird/South Korea/sw-anu/2023, the receptor-binding specificity of the resulting these viruses was determined using an assay measuring dose-dependent direct glycan binding to either a2,3-sialyl linkages (Neu5Aca2-3GalNAca) or a2,6-sialyl linkages (Neu5Gca2-6GalNAca) (Fig. 3). Generally, AIVs preferentially bind to α-2,3 linkage SA the vicinal galactose (a2,3-SAL) while human or swine influenza virus showed preferentially binding affinity to SA in α-2,6 pattern (a2,6-

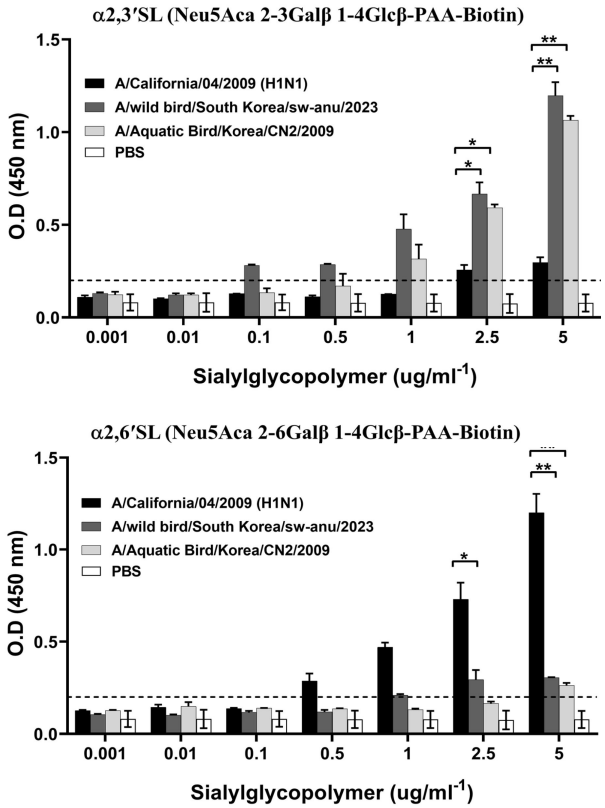

Fig. 3. Receptor binding assay of A/wild bird/South Korea/sw-anu/2023 virus. Receptor binding affinity of inactivated whole viruses to biotinylated α2,3- and α2,6-linked sialic acids glycans. All data are shown as means ± standard error (mean of three replicates) in bar graphs or asterisks indicate statistically significant differences between groups (*$p \leq 0.05$ or **$p \leq 0.01$). The dashed lines indicate the limit of detection.

SAL) [15]. We expected that a2,3-SAL glycan-binding properties would substantially increase the binding of A/wild bird/South Korea/sw-anu/2023 (Fig. 3a). Our results also showed that the a2,6-SAL binding affinity of A/wild bird/South Korea/sw-anu/2023 was not greater than that of A/California/04/2009 (Fig. 3b)

Discussion

In our current study, genomic analysis showed that A/wild bird/South Korea/sw-anu/2023 was a reassortant virus with genes originating from wild birds and domestic ducks. A previous study reported that reassortant H7N1 AIVs containing genes originating from domestic ducks and wild birds were isolated from environmental samples collected from live poultry markets [4]. The PB2, PB1, PA, and NP genes were similar to those of AIVs originating from wild birds, and the HA, NA, M, and NS genes were similar to those of AIVs originating from ducks. Considering the closest reference strains identified, genetic reassortment of A/wild bird/South Korea/sw-anu/2023 might have occurred when wild birds with the reassortant H7N1 AIV subtype came into contact with domestic ducks. Additionally, A/wild bird/South Korea/sw-anu/2023 could replicate in MDCK cells and achieve titers similar to those of the human H1N1 influenza A virus, A/California/04/2009, despite delayed kinetics. A/wild bird/South Korea/sw-anu/2023 also had high binding affinity for avian-specific glycans in the solid-phase direct binding assay.

H7N1 AIVs circulate in various regions such as North America [12], Europe [8], and China [20]. However, LPAI H7N1 viruses sporadically evolved HPAI properties, resulting in an outbreak in a poultry farm in Italy in 1999-2000 [2]. Sutton *et al*. identified an adapted H7N1 AIV capable of airborne transmission [16], indicating that H7N1 viruses can evolve HPAI characteristics from LPAI precursors. Recently, the first case of human infection with a novel avian-origin H7N9 virus was reported in China [22]. Phylogenetic analysis indicated that the novel avian-origin H7N9 virus showed reassortment of its external HA and NA genes from domestic ducks and wild birds and six internal genes from domestic poultry [7]. Thus, low pathogenic avian-origin H7 viruses may become highly pathogenic in humans, potentially leading to a pandemic situation.

Many Asian countries, including China, South Korea, and Japan, share the East Asian–Australasian migratory bird flyway [11]. In these countries, domestic poultry is raised in farms, which might contribute to the reassortment of novel AIVs with genes originating from both domestic poultry and migratory wild birds. The A/wild bird/South Korea/sw-anu/2023 virus isolated in our study exhibited LPAI characteristics; however, recently, LPAI H7N1 may have been acquired via an interspecies transmission event in the mammalian host. Therefore, considering zoonotic of AIVs, it is necessary to conduct more active surveillance of H7N1 AIVs in wild birds and poultry, as well as further study the molecular features involved in the mammalian adaptation of LPAI H7N1 viruses.

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The Conflict of Interest Statement

The authors declare that they have no conflicts of interest with the contents of this article.

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초록 : 조류 유래 재조합 H7N1 인플루엔자 바이러스의 분자적 특성 규명

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최근에 재조합 H7Nx 인플루엔자 바이러스가 산발적으로 인체 감염 사례가 보고되고 있으며 이러한 바이러스는 조류 종으로부터 지속적으로 분리되고있다. 본 연구에서는 조류에서 유래된 H7N1 인플루엔자 바이러스를 분리하여 A/wild bird/South Korea/sw-anu/2023로 명명하였고, 전장유전체 분석과 분자적 특성을 분석하였다. 계통발생학적 분석 결과 A/wild bird/South Korea/sw-anu/2023는 유라시아 혈통에 속하는 H7N1 인플루엔자 바이러스로 확인되었다. A/wild bird/South Korea/sw-anu/2023 바이러스의 polymerase basic 1(PB)2, PB1, polymerase acidic (PA), nucleoprotein (NP) 유전자는 야생 조류에서 분리되었던 조류 인플루엔자 바이러스유전자와 밀접한 관련이 있는 것으로 밝혀졌으며, hemagglutinin (HA), neuraminidase (NA), matrix (M), nonstructural (NS) 유전자는 집오리에서 분리되었던 조류 인플루엔자 바이러스와 유사하였다. 이러한 결과는 동아시아-호주 이동 경로를 따라 이동하는 야생 조류들 사이에서 새롭게 유전자가 재배열된 재조합 H7N1 조류 인플루엔자 바이러스가 순환되고 있음을 시사하고 있다. 따라서, H7Nx 인플루엔자 바이러스는 전 세계적으로 순환하며, 돌연변이된 H7N1 조류 인플루엔자 바이러스는 인간을 감염시킬 수 있으므로 야생 조류 및 가금류에서 H7N1 조류 인플루엔자 바이러스의 지속적인 감시가 필요할 것이다.