THE UNIVERSITY OF BRITISH COLUMBIA

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Date: 10 February 2025

Re: Expert Report – Risk of H5N1 influenza transmission from Ostriches located at Universal Ostrich Farms, Ltd. – Analysis of natural immunity

For the case involving Universal Ostrich Farms Ltd. represented by Mr. Michael Carter of Cleveland & Doan Barristers & Solicitors

PART 1: DESCRIPTION OF SCOPE OF THE QUESTIONS TO BE ADDRESSED

1. I have previously provided an expert report dated January 29, 2025, in response to questions that were put to me by the firm of Cleveland & Doan related to the flock of ostriches (Herd) located at the Universal Ostrich Farms Ltd. (UOF) near Edgewood, B.C. and the risks of transmission of the H5N1 strain of influenza, which is responsible for the current waves of avian flu. I am responding in this follow-up expert report on the concerns raised by Ms. Cathy Furness of the Canadian Food Inspection Agency (CFIA) in her January 30, 2025 affidavit, and new questions that have been asked of me by Cleveland & Doan in their February 8, 2025 letter (Exhibit A). In addition, I am providing results of testing for antibodies against the H5 hemagglutinin and N1 neuraminidase influenza viral proteins present in the yolk of ostrich eggs recovered in the summer of 2024, which is indicative of the ostriches' previous state of natural immunity against the avian H5N1 virus.

2. In particular, in correspondence (Exhibit A) that I received in the e-mail from Mr. Carter on February 8, 2025, I was requested to address the followings questions:

i. What is the likelihood that the ostriches presently are transmissible for H5N1 to each other and wild migratory birds such as ducks?

ii. What is the risk that ostriches may be asymptomatic and still actively replicating, mutating and shedding the virus?

iii. If there is no circulating virus in the ostriches, what is the risk of the following occurring:

a. the ostriches facilitating mutation of the virus;

b. the virus being transmitted to human;

c. the virus serving as precursor to a human flu pandemic;

d. the ostriches becoming infected with more than one subtype of the virus, which may allow HPAI variants to mix with other circulating influenzas creating new combinations; and

e. the ostriches contributing genetic mutations to AI viruses that may increase viral adaptability to mammalian hosts.

3. I understand that having been named as an expert witness by Universal Ostrich Farms Ltd., and having read the Code of Conduct for Expert Witnesses set out in the schedule to the Federal Court Rules, that I am bound to these rules, including in the preparation of this report.

PART 2: COLLECTION OF FACTS IN THE PREPARATION OF THIS EXPERT REPORT

4. My opinion on these matters is informed in part on the following facts that were conveyed by Mr. Carter on January 27th, 2025 as outlined in my January 29, 2025 expert report and new information provided in Mr. Carter's February 8, 2025 letter, which includes:

i. There have been no additional ostrich deaths from H5N1 type symptoms; and.

ii. The ostriches are not showing any signs of illness and appear healthy.

iii. The results from my own testing through my company Kinexus Bioinformatics Corporation (Vancouver, B.C.) for antibodies against the H5 hemagglutinin and N1 neuraminidase influenza viral proteins present in the yolks of 18 ostrich eggs produced at the UOF site in the summer of 2024.

5. In addition to these facts, I have viewed several media interviews[1,2,3] as well as talked with UOF owners Mr. Dave Bilinski and Ms. Karen Espersen, and her daughter Ms. Katie Pasitney, and this has further informed me regarding the history and status of their ostriches.

6. My own training in immunology and virology and personal experience and understanding of these fields affords me the ability to consider and weigh these issues in a knowledgeable way and offer a qualified expert opinion. My history and my *curriculum vitae* were provided in my January 29, 2025, expert report.

PART 3: RESPONSE TO QUESTIONS FROM CLEVELAND & DOAN

7. My January 29, 2025 expert report already provides detailed information about the history of the influenza virus, and its impacts on human and animal health, and the importance of natural immunity and vaccine-induced immunity in controlling its spread in vulnerable populations. A brief description was given of the tests to confirm an active viral infection: namely the Polymerase Chain Reaction (PCR) genetic test; the Rapid Antigen test (RAT), which detects viral proteins; and the live culture test, in which the specimens of the viral are incubated with live cells in culture or fertilized eggs to evaluate cell killing or developmental defects. To monitor a past infection with a virus following recovery, tests to detect the levels of specific antibodies against the pathogen in blood, saliva and other specimens are most commonly used.

8. A brief description of the composition and function of the cells of the immune system is also offered in my January 29, 2025 expert report. In particular, antibodies (also known as immunoglobulins (Ig)) are part of the main defense of the immune system, and these are produced by receptive B lymphocytes. In birds, reptiles and amphibians, the IgY class antibodies are most common, and can be concentrated in the yolk of their eggs. Antibodies lock on to foreign

[1] https://www.ctvnews.ca/vancouver/article/bc-farm-fights-order-to-cull-ostrich-herd-after-2-birds-test-positive-for-avian-flu/

[2] https://www.rebelnews.com/power_hungry_feds_order_culling_of_ostrich_farm?utm_campaign=dh_ostrichupdate_012725&utm

[3] https://www.youtube.com/live/dM5xHTKSzV0

proteins and other structures found in viruses, bacteria and fungi, and interfere with their ability to infect cells and also recruit other immune cells of the innate and adaptive immune systems to attack the pathogens or body cells that are infected with the pathogen. Measurement of the levels of specific antibodies in specimens from an animal can provide information whether it has been previously exposed to a pathogen and has a degree of protective immunity.

9. **What is the risk that the ostriches may be asymptomatic and still actively replicating, mutating and shedding the virus?**

10. Birds can become infectious about a day before they manifest any symptoms, and they are thought to be typically able remain infectious for five to seven days after the appearance of flu symptoms. Since recovery from symptoms may be up to two weeks in rare cases, in principle, it is possible that a bird could be infectious up to this time in limited cases. As the symptoms of influenza largely reflect the body's protective response to the infection, a subsiding of the symptoms usually reflects the complete cleansing of the body of the infection. As the viral load is reduced, the chances of transmission of the virus also decline.

11. Due to immune attack, the viral particles that may be released from a host body are also compromised, since they are likely coated with antibodies and have experienced damage that will render them less active. Thus, towards the end of an infection, along with a reduced viral load, the virus particles are less infectious. It is important to understand that viruses are not technically "living" since they absolutely need a host cell in which to replicate. It is better to consider them as active or functional, if they are replication capable, and inactive or non-functional if they are not.

12. The ability to establish a successful infection in a host is dependent on each step of a chain of independent events occurring in the proper order for a successful infection that will lead to propagation and transmission of a replication competent pathogen like a virus. These include:

    a. Link 1 – Sufficient dose of an infectious virus to be infective in the host, which is dependent the virus encountering a favorable environment (fertile ground) in the host and in part on the pre-existing immunity in the host.

b. Link 2 - The virus has to be cell penetrating, usually by initially binding to a host receptor, and replication competent, and achieve a critical mass before symptoms of the infection are manifested.

c. Link 3 – The virus has to be able to have a portal of escape from the host. The symptoms of an infection, such coughing, sneezing, production of phlegm, post-nasal drip and other secretions are mechanisms by which a virus may leave the body and infect a new host.

d. Link 4 – The mode of transmission is also critical, and may require close proximity or even direct contact. Influenza particle may remain suspended temporarily in an aerosol form in tiny droplets. However, larger droplets (larger than 60 microns) quickly drop to the ground within a meter, and smaller droplets undergo drying out, and reduction in size with evaporation. The influenza virus is about 0.14 microns in size, and more sensitive to damage in the absence of water following evaporation.

e. Link 5 – The virus has to have a portal of entry to gain access to the fertile ground. Note that being very large birds, with their heads held high, ostriches would be much less likely to breath in a virus particle than an animal like a flightless chicken whose head is closer to the ground.

f. Link 6 – The virus has to also find a susceptible host. The virus needs to be well adapted to the physiology of the host, including specific proteins that act as receptors and intracellular proteins that can replicate and package the virus. However, the health status of the host is also critical, and this can be influenced by wide range of factors, including age, diet, nutrition, life-style (such as access to natural conditions, low density, ability to exercise), stress, comorbidities and prior infection that can increase immunity.

13. Critical to the development of an infection, the six links constituting the Chain of Infection must be joined in the order of; Sufficient Dose of an Infectious Pathogen → Existence of a Viable Infectious Pathogen → A Portal of Escape → A Mode of Transmission → A Portal of Entry → A Susceptible Host. Breaking this chain by removing or incapacitating one of the links prevents the

transmission of an infectious disease. Although each link is critical, the most significant one regarding the transmission of an infectious disease is host susceptibility.

14. The clinical importance of this conclusion is more readily appreciated by focusing on host resistance rather than host susceptibility. This allows the probability of an infectious disease occurring to be represented by the following equation.[4]

$$\text{Infection} = \frac{\text{Virulence of the Pathogen x Dose of the Pathogen}}{\text{Host Resistance}}$$

15. This equation illustrates two factors relevant to the transmission of all infectious diseases. First, infection is not an inevitable outcome of exposure to a pathogen but depends on an interrelated series of events constituting the Chain of Infection. Second, host resistance is more pertinent to the development of an infectious disease than are the specific characteristics of a potential pathogen.[5] The significance of this factor is appreciated when the heterogeneous nature of a population is considered.

16. The concept of asymptomatic transmission is that an individual who has no symptoms of an infectious disease can still transmit it. In addition, it has been proposed that an individual at the early stages of an infectious disease may also be able to transmit that disease. The idea of asymptomatic transmission has been a major driver of policies, procedures and mandates associated with infections and has been a feature in many of the public health policies during the COVID-19 pandemic despite little evidence to support this contention. Much has since been learned about respiratory virus transmission from the COVID-19 pandemic, and since the SARS-CoV-2 virus and the influenza virus are RNA viruses of the same size with many shared characteristics, the lessons learned from either of these viruses is very instructive with respect to their transmission.

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[4] Runnels, R.R. (1984) Infection control in the wet finger environment. Salt Lake City: Publishers Press.

[5] Shulman, T.S. (1992) The biologic and clinical basis of infectious diseases. 4th edition. Toronto: W.B. Saunders.

17. The Chain of Infection dictates that for infection to occur, a sufficient dose of viable respiratory virus like influenza must be transmitted through the air from an infectious carrier to a potentially susceptible new host. It is the force associated with the symptoms of coughs and sneezes that expels infectious doses of a respiratory virus from the respiratory tract of the primary host with sufficient velocity to be transmitted as aerosols through the air and be inhaled by a new host where they must overcome that individual's natural defenses (intact mucous membranes) and immunological responses. The Chain of Infection reveals that an individual might harbor a virus and have nonexistent-to-mild non-specific symptoms, but unless the viable virus is expelled in sufficient amounts by sneezing or coughing and overcomes the resistance of a new host, the potential for transmission does not practically exist.

18. In January 2020, before the onset of the COVID-19 pandemic, Dr. Anthony Fauci, the director of the US National Institute of Allergy and Infectious Diseases at that time, supported this concept when he said, "*In all the history of respiratory borne viruses of any type, asymptomatic transmission has never been the driver of outbreaks. The driver of outbreaks is always a symptomatic person.*"[6] Further evidence on the low level of asymptomatic transmission for SARS-CoV-2 was provided by Dr. Maria Van Kerkhove, head of WHO's emerging diseases and zoonosis unit, on June 8, 2020, when she said that, "*from the data we have, it still seems to be rare that an asymptomatic person actually transmits onward to a secondary individual*." She continued to say that "*We have a number of reports from countries who are doing very detailed contact tracing. They're following asymptomatic cases. They're following contacts. And they're not finding secondary transmission onward. It's very rare*."[7]

19. In some species, it is possible that an animal may become infected with a pathogen and exhibit minimal symptoms due to the ability of that species to have adapted to that pathogen in its evolution. For example, *Yersinia pestis*, which is the bacteria responsible for the Black Death in

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[6] U.S. Department of Health and Human Services. (2020) Update on the new coronavirus outbreak first identified in Wuhan, China. YouTube. Retrieved from https://youtube.com/watch?v=w6koHkBCoNQ&t=2642s

[7] Feuer, W., Higgins-Dunn, N. (2020) "Asymptomatic spread of coronavirus is very rare," WHO says. CNBC. https://www.cnbc.com/2020/06/08/asymptomatic-coronavirus-patients-arent-spreading-new-infections-who-says.html

1348, is extremely deadly in humans through the production of a toxin. However, in the rats that transmitted the bacteria to humans at that time, it had minimal effects on these rodents. Rats lack an enzyme found in the blood of humans that is required to activate the toxin produced by *Yersinia pestis*. However, chickens and ostriches have a significant risk of mortality if they contract avian influenza, so they do not as species appear to have evolved the ability to build up a significant viral load for transmission of influenza without manifestation of obvious symptoms. Birds that do not develop symptoms most likely have a prior degree of natural immunity from previous exposure, although genetics may also play a role.

20. **What is the ability of ostriches to facilitate mutations of the influenza virus?**

21. Like other viruses, with each replication cycle of an influenza virus in a host, there is the potential to introduce mutations in the genome structure of the virus and its encoded proteins. This most commonly arises from the error rate in the RNA polymerase enzymes that produce copies of the RNA in the genome, which is itself segregated into 8 separate chromosomes. The vast majority of these introduced mutations are actually deleterious or inconsequential to the production of functional virus particles. In very rare circumstances, the mutation may increase infectivity, or evasion of the immune system, but extremely rarely does it increase the virulence of the virus. An increase in viral virulence is actually deleterious to the longer-term propagation of the strain of the virus, since killing the host limits transmission. A host that is minimally sick from an infection is much more likely to be transmitted other hosts. This is exactly what happened during the COVID-19 pandemic, when the more virulent Wuhan SARS-CoV-2 strain was eventually out-completed by the more infectious and milder Omicron variants.

22. There is no reason to believe that the mutation rate of the influenza virus in an infected ostrich would be any different than in wild ducks or farmed chickens. However, the opportunity that such chance mutations with a gain of function would occur is very dependent on the group number of infected animals in the species and how confined they may be to increase the chances of spread (*e.g.,* cooped up chickens) or mobile to increase the zone of transmission (*e.g.,* wild migrating ducks). The size of the ostrich herd at UOF, even with 390 animals, is relatively small when compared to most flocks of domestic poultry or wild birds.

22. Because the genome of the influenza virus is separated into 8 small chromosomes, it is theoretically feasible that if different strains of the influenza virus happened to co-infect the very same host cells, there could be mixed packaging of the genomes of these viruses to produce hybrid viruses. This might permit cross-over for infectivity to other species. However, the chances of this happening are very low, because an active infection with one strain of the virus will induce the upregulation of innate and adaptive immune systems to fight any other respiratory virus infections, which would make it harder for second, likely weaker strain to flourish in the host.

23. While it is feasible for a pathogenic virus to evolve through accumulation of mutations of its genome to infect a new host species, this is more difficult the greater the evolutionary difference in the host species. This is rare for a virus, because it has a limited number of genes to start with, and they encode proteins that are already highly optimized for the species that they normally infect. These viral proteins have to be well matched for the host's proteins to successfully gain entry into the cells, hijack the cellular machinery that is normally used to replicate the cell into making viral proteins instead, and assembly complete infectious viral particles.

24. While the jumping from one host species to another host species can occur, and this may result on rare occasions in a pandemic in the new host species, once this occurs, further mutations in the virus usually result in increasing the infectivity, reducing the virulence (*i.e.,* decreasing morbidity and mortality) and increasing evasion of the immune system. There are plenty of examples of this, including the viruses that cause common colds and flues that while inconvenient, rarely require hospitalization and resolve with full recovery. Moreover, it should be appreciated that there are hundreds of thousands of different viruses that are believed to infect mammals, but only about a hundred are actually pathogenic in humans. Again, this is because of the difficulty for viruses to cross species, which is called zoonosis if it is able to cross over to humans.

25. **What is the likelihood of ostrich transmission of avian influenza to humans?**

26. The transmission of avian influenza from chickens to humans is well documented. Any persons working with an animal that is infected with this virus will be exposed to the virus and likely mount an immune response usually without the development of symptoms of disease. However,

as mentioned in paragraphs 12 to 18, the chain of infection must be fully supported to allow transmission of an infectious pathogen where it can cause sickness.

27. The development of immunity against a viral or bacterial pathogen through vaccination, which usually does not cause sickness in the recipient, is ample demonstration that low level infection with a pathogen is sufficient to evoke substantial protection against future infections with a more virulent strain of the pathogen. Moreover, exposure to a milder form of a virus, for example such as cow pox from handling cows as documented for milk maids, can actually provide immunity to more lethal strains in human, such as small pox in this case.

28. The question is really whether the avian flu from a domesticated animal is likely to cause illness in a human. There is a greater chance that a highly pathogenic influenza virus that can seriously affect humans will emerge from a mammalian host such as pigs, cattle or minks, which have been also been shown to become infected with the avian H5N1 strain of influenza. The risk of transmission of influenza that causes sickness from a bird host such an ostrich is substantially lower. Ostriches have been a distinct species for over 120 million years, whereas humans and chimpanzees evolved from a common ancestor about 7.5 million years ago,

29. Moreover, it is more likely that a more virulent strain of influenza would emerge by mutation from influenza strains that already infect humans. Typically, about 5 to 10% of adults and 20 to 30% of children in Canada are infected annually and have flu-like symptoms.[8] In the brief 2022-2023 flu season, 97% of the confirmed influenza cases were influenza A, and about 3,500 death were reported from influenza and pneumonia across Canada.[8] More recently, according the latest reports from the US Center for Disease Control (CDC), during this winter season there has been at least 24 million illnesses, 310,000 hospitalizations and 13,000 death in the US from the

[8] Hawthorne, K. (2024) Influenza in Canada: Stat, impacts and resources. Healthing.ca. Retrieved from https://www.healthing.ca/diseases-and-conditions/influenza-flu/influenza-in-canada-stats-impact-and-resources

flu.[9] This would correspond to a case fatality rate of about 0.05% from what is primarily H1N1 and H3N2 influenza strains.

30. Furthermore according the CDC,[10] in the US there have been a total of 67 confirmed human cases of the avian bird flu, and only 1 death in a 65-year-old that had infected chickens in their backyard, but also had pre-existing health complications. In Canada, there is only single case of a 13-year-old girl with a mild asthma history living in BC that became seriously ill with H5N1, and there is no explanation for how this young female contracted the disease.[11] There are no known cases of human-to-human H5N1 transmission.[10] The majority of H5N1 cases in humans appear to be from working on farms with cattle slightly more than with poultry, and the illnesses were usually mild.[12]

31. Can the H5N1 virus serve as a precursor to a pandemic in humans?

32. It is possible that the H5N1 virus could mutate in to a form that causes a pandemic in people. The H1N1 strain that infects people and is responsible for past human pandemic such as the 1918 Spanish Flu, shares the N1 neuraminidase protein. However, the H5 hemagglutinin protein has not previously been associated with major pandemics in humans in the past century, whereas the H1, H2 and H3 forms have.

33. The hemagglutinin protein is important in the binding of the influenza virus receptors on host cells. Avian influenza viruses use α2-3-linked sialic acids as their preferred receptors, but human-transmissible strains prefer α2-6-linked sialic acids, which are found on the surface of human

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[9] Weekly US influenza surveillance report: Key updates for Week 5, ending February 1, 2025. (2025) CDC FluView. Retrieved from https://www.cdc.gov/fluview/surveillance/2025-week-05.html

[10] H5 bird flu: Current situation (2025). Retrieved from https://www.cdc.gov/bird-flu/situation-summary/index.html

[11] Jassem, A.N., Roberts, A., Tyson, J., Zlosnik, J.E.A., Russell, S.L., Caleta, J.M. *et al.* (2024) New England Journal of Medicine. Doi:10.1056/NEJMc2415890. Retrieved from https://www.nejm.org/doi/full/10.1056/NEJMc2415890

[12] Garg, S., Reinhart, K., Couture, A., Kniss, K., Davis, T., *et al.* (2024) New England Journal of Medicine. Doi: 10.1056/NEJoa2414610. Retrieved from https://www.nejm.org/doi/full/10.1056/NEJMoa2414610

cells in the airways and lungs.[13] Deep mutation scanning of H5 hemagglutinin binding to α2-6-linked sialic acids using pseudoviruses has shown that it is possible to improve binding to α2-6-linked sialic acids, the stability of the H5 3D structure, and reduce immunogenicity with pre-existing neutralizing antibodies. However, this was restricted to about a dozen mutations out of of 10,773 possible mutations in H5. Consequently, the ability of the H5N1 to mutate into a likely contagious and virulent influenza strain for human-to-human transmission remains very low. It is much more likely that the next influenza pandemic will emerge from the existing strains that are already optimized through evolution to infect mammals.

34. **Could co-infection of ostriches with two different strains of influenza result in human pathogenic avian influenza variants?**

35. This question has already been addressed in paragraphs 22 and 23. While theoretically possible, this would be very unlikely. A very sick person with a different influenza strain would have to be attending to a very sick ostrich that had H5N1. This is highly unlikely with a small number of staff at the UOF site and the limited number of birds in the flock that would be sick, especially after acquisition of natural immunity against H5N1 virus. That sick person would most likely have an H1N1 or H3N2 influenza strain, and these strains weakly bind to the α2-3-linked sialic acids found in birds that serve as receptors for the virus. Most combinations of the packaging of the 8 different chromosomes of the influenza genome into a viral particle would likely create less virulent forms for both ostriches and humans, since these would have been previously optimized to work on the host cells from the species for which they are derived.

36. **Could the mutation of the H5N1 virus in ostriches increase viral adaptability to humans?**

Ostriches are as evolutionarily distant from humans as are chickens and ducks. There is no reason to expect that the mutation of the H5N1 virus in ostriches would make it easier to infect humans

[13] Dadonaite, B., Ahn, J.J., Ort, J.T., Yu, J., Furey, C., *et al*., (2024) Deep mutational scanning of H5 hemagglutinin to inform influenza virus surveillance. PLoS Biol. 22(11):e3002916. doi: 10.1371/journal.pbio.3002916. Retrieved from https://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.3002916

than the mutations transpiring in chickens or ducks. On the one hand, should the H5N1 virus happen to mutate to increase its binding to humans, it would be less efficient in binding to other ostriches and other domestic or wild fowl. On the other hand, if the virus mutated in the ostrich to increase its binding to other birds, it would less efficiently infect mammals. It is much more likely that further mutation of the H5N1 virus in a cow, pig or mink would further improve its adaptability to humans.

PART 4: RESPONSE TO POINTS IN THE AFFIDAVIT FROM DR. CATHY FURNESS

37. From Dr. Furness's January 30, 2025, affidavit, it appears that her formal training is as a veterinarian and she plays a senior administrative role at the Canadian Food Inspection Agency (CFIA). She does not appear to be trained or experienced in active research related infectious diseases and immunity. I only found six career publications in Google Scholar in which she appeared to coauthor (under the name Mary Catherine Furness) with her latest in 2017.[14]

38. In paragraph 12 of her affidavit, Dr. Furness notes that for avian influenza that "[a]*pproximate half of the over 900 human cases reported around the world since 1997 have been fatal*." No citation is provided for this statement, and this grossly overstates the lethality of avian H5N1 influenza in humans. As pointed out in paragraph 30, of 68 reported cases of avian H5N1 in humans, there was only 1 recorded fatality.[10, 11]

39. In paragraph 16 of her affidavit, Dr. Furness states that *"[a]lthough rare, transmission of HPAI to humans can occur, most commonly when people have had close contact with infected birds*." Actually, most reports of infection of people with avian H5N1 appear to be from cattle.[12]

40. In paragraph 19 of her affidavit, Dr. Furness mentions that "*CFIA's implementation of stamping out aligns with WOAH's standards. Without stamping out, a country cannot be considered free from HPAI until at least 12 months from an infection in poultry, as opposed 28 days where*

[14] https://www.cambridge.org/core/journals/prehospital-and-disaster-medicine/article/an-emergency-exercise-in-the-veterinary-diagnostic-laboratory-preparing-for-a-foreign-animal-disease-outbreak/C8B9D924E65195FC852C5367C1CBE45F

stamping out is implemented." In my read through the World Organization for Animal Health (WOAH) guidelines provided in her Exhibit A, entitled "Chapter 10.4. Infection with High Pathogenicity Avian Influenza Viruses", I observed that the stamping out of infected animals was not a required policy to control outbreaks of HPAI as described in Article 10.4.3. As pointed out by Dr. Furness, a period of at least a year is stipulated if culling is not performed. Article 10.4.6 indicates that recovery of the "free status" is possible after 28 days following stamping out. However, in my review of the status of flocks in Canada where avian influenza has been detected, there are currently 38 infected premises that have active infections and another 485 that have been infected and subjected to stamping out over about a three-year period.[15] Most of these outbreaks have been in B.C. and have affected about 14.5 million domestic birds across Canada. With such a large number of outbreaks, which are presumable spaced out over the intervening years, but primarily in the winter, it is hard to see how Canada has achieved a "free status" even with the stamping out policy enforced by the CFIA.

41. In paragraph 21 of her affidavit, Dr. Furness claims that "[s]*imilar to many birds, ostriches typically do not show clinical signs of infection of AI but can nonetheless continue to replicate, mutate, and shed the virus. It is also possible for ostriches to be infected with more than one subtype of influenza virus.*" This claim is not supported by any citation. As I have mentioned in paragraphs 10 to 19, it is highly unlikely that an asymptomatic ostrich or other bird is shedding functional replication-competent virus. As pointed out in paragraph 22, 23 and 35, while it is theoretically possible for the same cells in the same bird to be co-infected with two different influenza strains, a result in a more virulent and infectious virus that will infect humans is very unlikely.

42. In paragraph 30 of her affidavit, Dr. Furness notes that "[i]*norder to qualify for an exemption, Universal needed to demonstrate that the ostriches: (a)were a distinct epidemiological unit; and (b) possessed rare and valuable poultry genetics*." From this statement, the fact that the ostriches may have been previously been infected, recovered and have fully immunity is deemed irrelevant. Moreover, CIFA apparently disregarded the unique composition of the UOF flock

[15] Status of ongoing avian influenza response by province. (2025) Government of Canada. Retrieved from https://inspection.canada.ca/en/animal-health/terrestrial-animals/diseases/reportable/avian-influenza/latest-bird-flu-situation/status-ongoing-response

which included breeding with rare and endangered ostrich species, *i.e.*, the Somalian Blue and Arabian ostriches. Culling of this flock will result in a loss of the further loss of this genetic pool, which will be difficult to replace.

PART 5: EVIDENCE OF NATURAL IMMUNITY TO AVIAN H5N1 INFLUENZA IN THE UOF HERD

43. In late December 2024 and early January 2025, some 69 of the approximately 450 ostriches on the UOF site succumbed to an infection, which on the balance of probabilities was influenza H5N1 based on two of the deceased animals testing PCR-positive for this virus. All of these deaths were confined to about 200 ostriches that were added to the herd at the UOF site after 2020. None of the ostriches that were at the UOF site prior to 2021 were seriously sick and died. This would be very consistent with herd immunity against H5N1 in the longer established members of the herd.

44. The UOF herd underwent a period of illness around February 2020, which resulted in the deaths of 10 of the approximately 250 ostriches that were on-site. This was diagnosed as a possible bacterial pseudomonas or *E. coli* infection at the time, but the symptoms associated with the illness were also consistent with influenza. H5N1 was already detected in wild birds in Europe in 2020, and the first H5N1 strain was first detected in geese in China in 1996.[16] Therefore, it is feasible that the ostrich herd could have been infected with H5N1 or a highly related influenza strain in 2020. In addition, secondary bacterial infections following initial influenza infection often are accompanied by a pseudomonas infection.[17]

45. Since there have been no deaths of ostriches on the UOF site since January 15, 2025 and all of the birds now appear to be healthy without symptoms, it is very likely that herd immunity to H5N1 has been achieved in the entire herd. The probability of this being true increases as time

[16] Katella, K. (2024) H5N1 Bird Flu: What you need to know. Yale Medicine. Retrieved from https://www.yalemedicine.org/news/h5n1-bird-flu-what-to-know

[17] Morris, D.E., Cleary, D.W., Clarke, S.C. (2017) Secondary bacterial infections associated with influenza pandemics. Front Microbiol. 8:1041. doi: 10.3389/fmicb.2017.01041.

passes without any further evidence of sickness or positive testing for H5N1 by specific Polymerase Chain Reaction (PCR) tests and rapid antigen tests (RAT).

46. For reasons that are opaque to me, the CFIA has forbidden UOF to perform any testing or treatment of the ostriches at their site since imposition of the quarantine order. This would have been an excellent opportunity to obtain valuable scientific information on the extent and durability of natural immunity following exposure to wild birds that carry the H5N1 virus.

47. Following recovery from a viral or bacterial infection, the only way to determine whether herd immunity has been achieved for the pathogen is to perform testing for the presence of specific antibodies or specific T-lymphocytes that recognize and bind to antigens from the pathogen. Such antigens can be proteins that are encoded by the genome of the pathogen or small portions of these proteins. Often this involves the testing of blood or saliva specimens from humans and animals. However, in birds, reptiles and amphibians, this can be achieved by testing egg yolks, which are rich in the IgY class of antibodies. IgY antibodies are particularly stable and can take temperatures up to 100°C and still retain their ability to lock on to target antigens.

48. In my January 30, 2025 expert report, I outlined how my company Kinexus Bioinformatics Corporation developed a serological test for determining in blood samples the levels of antibodies against the SARS-CoV-2 virus for a clinical study that involved the testing of 4,500 participants. In short, about 6,000 short peptide fragments derived from the predicted amino acid sequences of 28 SARS-CoV-2 proteins were tested in SPOT peptide arrays for their ability to bind antibodies that were present in individuals that got COVID-19 as confirmed by symptoms and positive PCR tests for the virus. These clinical studies revealed that about 90% of the tested participants had multiple antibodies that recognized SARS-CoV-2 virus-derived peptides, that their patterns of which viral peptides that the participants made antibodies varied markedly, and that these patterns were stable for at least two years.

49. With the situation of the apparent outbreak of avian influenza on the UOF site and the evident establishment of herd immunity by January 15, 2025, there was a valuable opportunity to see if the SPOT methodology could be applied to identifying those parts of the H5N1 virus that were

immunogenic (*i.e.,* evoke a strong antibody response) and could be diagnostic for specific prior infection with the H5N1 virus and not other strains of influenza viruses. However, the order from the CFIA prevented the UOF from sending me recent samples from the quarantined ostriches for antibody testing.

50. However, the UOF had frozen samples of egg yolks that had been collected from their ostriches in the summer of 2024, which pre-dated the recent infection of the herd in December 2024 and January 2025, and which were stored off-site, outside of the quarantine zone. I reasoned that if the ostriches had a prior exposure to H5N1 virus, then these yolk samples should have evidence of antibodies that recognize and bind to peptides patterned after parts of the H5 hemagglutinin and N1 neuraminidase proteins. Such peptides should be unique in their amino acid sequences from the hemagglutinin and neuraminidase proteins found in other influenza strains known to infect birds.

51. Figures 1 and 2 show the complete amino acid sequences of hemagglutinin and neuraminidase proteins found in H5N1 and other avian influenza viruses. Those regions that were the most unique to the H5 and N1 proteins are highlighted in yellow on the figure. Based on these unique sequences, 74 peptides were synthesized on cellulose membranes as individual spots on the arrays.

52. These H5N1-specific peptide SPOT arrays were then used for testing. Briefly, the frozen egg yolk were thawed and diluted with a phosphate-buffered saline solution and then incubated with the SPOT arrays for 2 hours. Subsequently, the arrays were washed repeatedly to remove any unbound antibodies from the cellulose membrane. The presence of captured antibodies on the arrays was detected with a horse radish peroxidase (HRP)-coupled secondary antibody that recognizes the constant region of chicken IgY antibodies. Enhanced chemiluminescence (ECL) detection based on the appearance of product from the HRP-couple reaction permits the production of a dark spot on the SPOT array after scanning it with a Bio_rad FluroS-Max imager. The darker the peptide spot on the array image, the higher the level of IgY antibody that specifically recognizes that peptide amino acid sequence.

Figure 1. Alignment of the H5, H4, H7, H9 and H10 hemagglutinin proteins of chicken influenza strains. Amino acid sequences were obtain from Uniprot (www.uniprot.org) and aligned using the CLUSTAL O(1.2.4) multiple sequence alignment program available at the Uniprot website. Perfect matches (*i.e.,* identity) with the amino acid type and position are shown with an asterisk; similar amino acid types in the same position (*i.e.,* similarity) are shown with a colon (high similarity) or semi-colon (low similarity). The regions encompassing the peptides used in the Kinexus anti-H5N1 antibody test for the hemagglutinin are highlighted in yellow.

```
sp|P09345|HEMA_I59A0     H5  ME--RIVLLLA----IVSLVKSDQICIGYHANKSTKQVDTIMEKNVTVTHAQDILERTHN      54
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  MEAVSLITILL----VVTVSNADKICIGYQSTNSTETVDTLTENNVPVTHAKELLHTEHN      56
sp|P19695|HEMA_I75A4     H4  MLSITILFLLIAEGSSQNYTGNPVICLGHHAVSNGTMVKTLTDDQVEVVTAQELVESQHL      60
sp|P12581|HEMA_I49A0     H10 MYKVVVIIALL-----GAVRGLDKICLGHHAVANGTIVKTLTNVQEEVTNATETVESTSL      55
sp|P09343|HEMA_I85A3     H7  MNTQILILTLV----AAIHTNADKICLGHHAVSNGTKVNTLTERGVEVVNATETVERRTI      56
                             *    ::  *             **:*:::  .   *.*: :    *. * : :.

sp|P09345|HEMA_I59A0     H5  GKLCSLNGVKPLILRDCSVAGWLLGNPMCDEFLNVPEWSYIVEKDNPINSLCYPGDFNDY      114
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  GMLCATNLGHPLILDTCTIEGLIYGNPSCNLLLGGREWSYIVERPSAVNGLCYPGNVENL      116
sp|P19695|HEMA_I75A4     H4  PELCPS-PLRLVDGQTCDIVNGALGSPGCNHLNG-AEWDVFIERPTAV-DTCYPFDVPDY      117
sp|P12581|HEMA_I49A0     H10 NRLCMK-GRSYKDLGNCHPIGMLIGTPACDLHLT-GTWDTLIERKNAI-AYCYPGTTINE      112
sp|P09343|HEMA_I85A3     H7  PRICTK-GKKAIDLGQCGLLGIITGPPQCDQFLE-FTADLIIERREGN-DVCYPGKFVNE      113
                               :*           *   .   * * *:        . ::*:       ***    :

sp|P09345|HEMA_I59A0     H5  EELKHLLSSTNHFEKIQIIPRSSWSNHDASSGVSSACPYIGRSSFFRNVVWLIKK--DNA      172
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  EELRSLFSSASSYQSIQIFPDTIWNVSY--SGTSKAC----SDSFYGSMRWLAQK--NNA      168
sp|P19695|HEMA_I75A4     H4  QSLRSILANNGKFEFI--VEKFQWNT-VKQNGKSGACKRANENDFFTNLNWLTKS-DGNA      173
sp|P12581|HEMA_I49A0     H10 GALRQKIMESGGISKT--STGFAYGSSINSAGTTKACMRNGGDSFYAEVKWLVSKDKGQN      170
sp|P09343|HEMA_I85A3     H7  EALRQILRESGGINKE--TTGFTYSG-IRTNGVTSACRR-LGSSFYAEMKWLLSNTDNAA      169
                               *:  : . .  .         :.      * : **     ..*: .: ** ..  .

sp|P09345|HEMA_I59A0     H5  YPTIKRSYNNTNQEDLLILWGIHHPNDAAEQTKLYQNPTTYVSVGTSTLNQRSIPEIATR      232
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  YPIQDAQYTNNRGKNIPFMWGINHPPTDTVQTNLYTRTDTTTSVATEDINRTFKPLIGPR      228
sp|P19695|HEMA_I75A4     H4  YPLQNLTKVNNGDYARLYIWGVHHPSTDTEQTNLYENNPGRVTVSTKTSQTSVVPNIGSR      233
sp|P12581|HEMA_I49A0     H10 FPQTTNTYRNTDTAEHLIIWGIHHPSSTQEKNDLYGTQSLSISVGSSTYQNNFVPVVRAR      230
sp|P09343|HEMA_I85A3     H7  FPQMTKSYKNTRNEPALIVWGIHHSGSATEQTKLYGSGNKLITVGSSNYQQSFVPSPGAR      229
                             :*       *.       :**::*      :..**       :*.:.  :    *    *

sp|P09345|HEMA_I59A0     H5  PKVNGQSGRMEFFWTILKPNDAINFESNGNFIAPEYAYKIVKKGDSAIMKSGLAYGNCDT      292
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  PLVNGQQGRIDYYWSVLKPGQTLRVRSNGNLTAPWYGHILSGESHGRILKTDLNSGNCVV      288
sp|P19695|HEMA_I75A4     H4  PWVRGQSGRISFYWTIVEPGDIIVFNTIGNLIAPRGHYKLNSQKKSTILNTAVPIGSCVS      293
sp|P12581|HEMA_I49A0     H10 PQVNGQSGRIDFHWTLVQPGDNITFSHNGGRIAPSRVSKLVGRGL-GIQSEASIDNGCES      289
sp|P09343|HEMA_I85A3     H7  PQVNGQSGRIDFHWLILNPNDTVTFSFNGAFVAPDRVSFFKGKSM-GIQSEVPVDTNCEG      288
                             * *.**.**:.:.* :::*.: : .   *   **     :  .    * .      .*

sp|P09345|HEMA_I59A0     H5  KCQTPVGAINSSMPFHNIHPHTIGECPKYVKSDRLVLATGLRNVPQR----KKRGLFGAI      348
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  QCQTERGGLNTTLPFHNVSKYAFGNCPKYVGVKSLKLAVGLRNVPAR----SSRGLFGAI      344
sp|P19695|HEMA_I75A4     H4  KCHTDRGSITTTKPFQNISRISIGDCPKYVKQGSLKLATGMRNIPEK----ATRGLFGAI      349
sp|P12581|HEMA_I49A0     H10 KCFWRGGSINTKLPFQNLSPRTVGQCPKYVNKKSLMLATGMRNVPEIM---QGRGLFGAI      346
sp|P09343|HEMA_I85A3     H7  ECYHNGGTITSNLPFQNVNSRAVGKCPRYVKQKSLLLATGMKNVPEIPKKREKRGLFGAI      348
                             :*    * :.:. **:*:   :.*.**:**    * **.*::*:*        *******

sp|P09345|HEMA_I59A0     H5  AGFIEGGWQGMVDGWYGYHHSNEQGSGYAADKESTQKAIDGITNKVNSIIDKMNTQFKAV      408
tr|Q4ZJF4|Q4ZJF4_9INFA  H9  AGFIEGGWSGLVAGWYGFQHSNDQGVGMAADRDSTQKAIDKITSKVNNIVDKMNKQYEII      404
sp|P19695|HEMA_I75A4     H4  AGFIENGWQGLIDGWYGFRHQNAEGTGTAADLKSTQAAIDQINGKLNRLIEKTNEKYHQI      409
sp|P12581|HEMA_I49A0     H10 AGFIENGWEGMVDGWYGFRHQNAQGTGQAADYKSTQAAIDQITGKLNRLIEKTNTEFESI      406
sp|P09343|HEMA_I85A3     H7  AGFIENGWEGLVDGWYGFRHQNAQGEGTAADYKSTQSAIDQITGKLNRLIEKTNQQFELI      408
                             *****.**.*:: ****::*.* :* * *** .*** *** *..*:* :::* * ::. :
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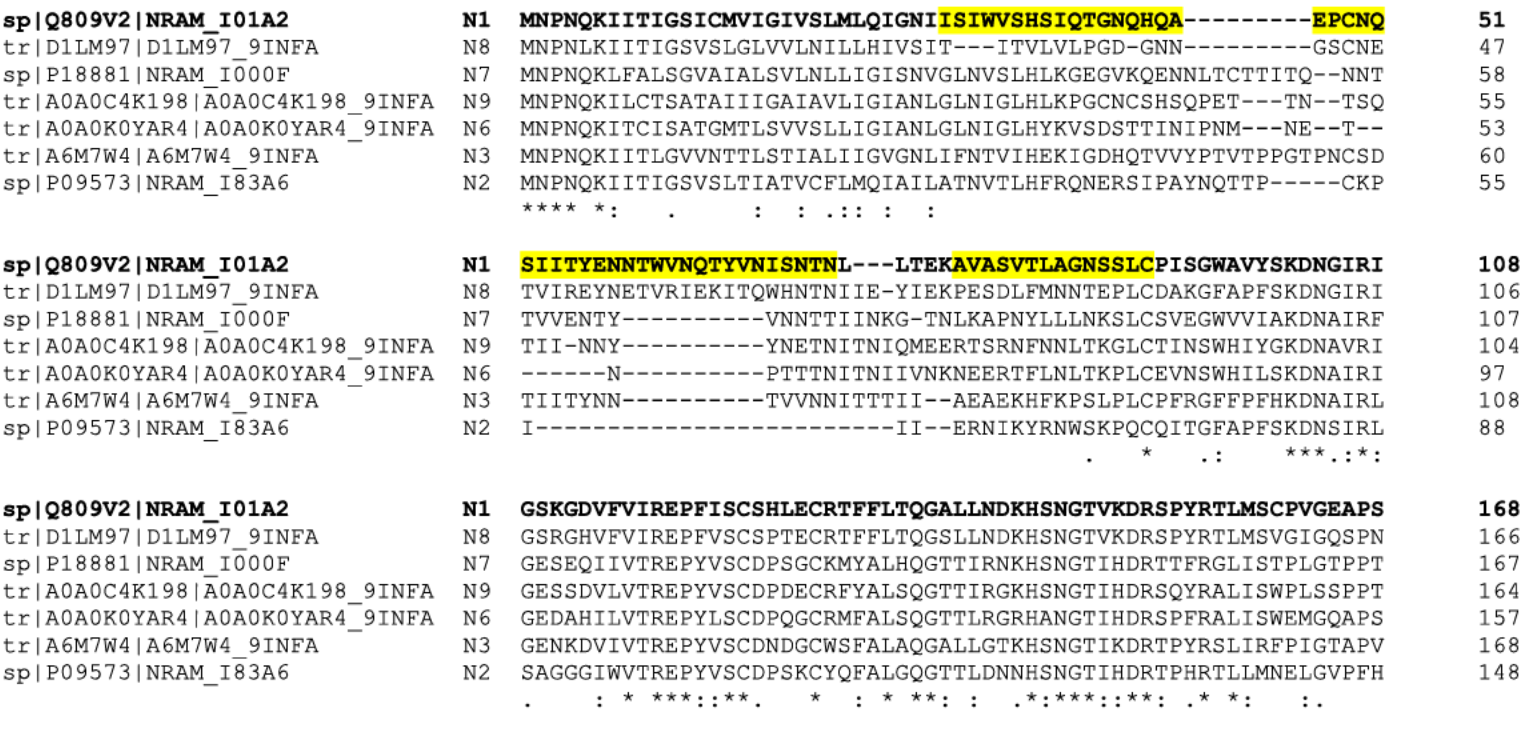
Figure 2. Alignment of the N1, N2, N3, N6, N7, N8 and N9 neuraminidase proteins of chicken influenza strains. Amino acid sequences were obtain from Uniprot (www.uniprot.org) and aligned using the CLUSTAL O(1.2.4) multiple sequence alignment program available at the Uniprot website. Perfect matches (*i.e.,* identity) with the amino acid type and position are shown with an asterisk; similar amino acid types in the same position (*i.e.,* similarity) are shown with a colon (high similarity) or semi-colon (low similarity). The regions encompassing the peptides used in the Kinexus anti-H5N1 antibody test for the neuraminidase are highlighted in yellow.



```
sp|Q809V2|NRAM_I01A2          N1  PYNSRFESVAWSASACHDGTSWLTIGISGPDNGAVAVLKYNGIITDTIKSWRNNILRTQE  228
tr|D1LM97|D1LM97_9INFA        N8  VYQARFEAVAWSATACHDGKKWMTIGVTGPDAKAVAVVHYGGIPTDVINSWAGDILRTQE  226
sp|P18881|NRAM_I000F          N7  VSNSDFICVGWSSTSCHDGVGRMTICIQGNNDNATATVYYNRRLTTTIKTWAKNILRTQE  227
tr|A0A0C4K198|A0A0C4K198_9INFA N9  VYNSRVECIGWSSTSCHDGKSRMSICISGPNNNASAVVWYNRRPVAEINTWAQNILRTQE  224
tr|A0A0K0YAR4|A0A0K0YAR4_9INFA N6  PYNTRVECIGWSSTSCHDGISRMSICISGPNNNASAVVWYRGRPVTEIPSWVGNILRTQE  217
tr|A6M7W4|A6M7W4_9INFA        N3  LGNYKEICVAWSSSSCFDGKEWMHVCMTGNDNDASAQIIYAGKMTDSIKSWRRDILRTQE  228
sp|P09573|NRAM_I83A6          N2  LG-TRQVCIAWSSSSCHDGKAWLHVCVTGDDRNATASFIYNGMLVDSIGSWSQNILRTQE  207
                                      .:.**:::*.**   : : : * :  * * . *    .  * :*  :******

sp|Q809V2|NRAM_I01A2          N1  SECACVNGSCFTVMTDGPSNGQASYKIFKIEKGKVVKSVELN-APNYHYEECSCYPDAGE  287
tr|D1LM97|D1LM97_9INFA        N8  SSCTCIQGECYWVMTDGPANRQAQYRAFKAKQGKIIGQTEIS-FNGGHIEECSCYPNEGK  285
sp|P18881|NRAM_I000F          N7  SECVCYNGTCAVVMTDGPASSQAYTKIMYFHKGLIIKEEPLR-GSARHIEECSCYGHDQK  286
tr|A0A0C4K198|A0A0C4K198_9INFA N9  SECVCHNGVCPVVFTDGSATGPADTRIYYFKEGKILKWESLT-GTAKHIEECSCYGERTG  283
tr|A0A0K0YAR4|A0A0K0YAR4_9INFA N6  SECVCHKGICPVVMTDGPANNKAATKIIYFKEGKIQKIEELQ-GNAQHIEECSCYGAAGM  276
tr|A6M7W4|A6M7W4_9INFA        N3  SECQCIDGTCVVAVTDGPAANSADHRVYWIREGRVIKYENVPKTKIQHLEECSCYVDI-D  287
sp|P09573|NRAM_I83A6          N2  SECVCINGTCTVVMTDGSASGKADIRILFIREGKIVHISPLS-GSAQHIEECSCYPRYPN  266
                                  *.* * .* *  ..*** :   *  :    .:* :     :      * ******

sp|Q809V2|NRAM_I01A2          N1  ITCVCRDNWHGSNRPWVSFNQN-LEYQIGYICSGVFGDNPRPNDG--TGSCGPVSPN--G  342
tr|D1LM97|D1LM97_9INFA        N8  VECVCRDNWTGTNRPVLVISSD-LSYRVGYLCAGLPSDTPRGEDNQFTGSCTSPMGN--Q  342
sp|P18881|NRAM_I000F          N7  VSCVCRDNWQGANRPIIEIDMSTLEHTSRCVCTGVLTDTSRPGDKP-NGDCSNPITGSPG  345
tr|A0A0C4K198|A0A0C4K198_9INFA N9  ITCTCRDNWQGSNRPVIQIDPVAMTHTSQYICSPVLTDNPRPNDPN-IGKCNDPYPGN-N  341
tr|A0A0K0YAR4|A0A0K0YAR4_9INFA N6  IKCVCRDNWKGANRPIITIDPEMMTHTSKYLCSKILTDTSRPNDPT-NGNCDAPITGGSP  335
tr|A6M7W4|A6M7W4_9INFA        N3  VYCVCRDNWKGSNRPWMRINNE-TILETGYVCSKFHSDTPRPADPS-TVSCDSPSNVN-G  344
sp|P09573|NRAM_I83A6          N2  VRCVCRDNWKGSNRPVIDINMADYSIDSSYVCSGLVGDTPRNDDSSSSSNCRDPNNER-G  325
                                  : *.***** *:*** : :.          :*: .  *. *  *     .*

sp|Q809V2|NRAM_I01A2          N1  AYGIKGFSFKYGNGVWIGRTKSTNSRSGFEMIWDPNGWTG-TDSNFSVKQDIVAITDWSG  401
tr|D1LM97|D1LM97_9INFA        N8  GYGVKGFGFRQGNDVWMGRTISRTSRSGFEILRVRDGWIQ-NSKEQIKRQVVVDNLNWSG  401
sp|P18881|NRAM_I000F          N7  APGVKGFGFLNGDNTWLGRTISPRSRSGFEMLKIPNAETD-PNSRIIERQEIVDNSNWSG  404
tr|A0A0C4K198|A0A0C4K198_9INFA N9  NNGVKGFSYLDGANTWLGRTISTASRSGYEMLKVPNALTD-DRSKPIQGQTIVLNADWSG  400
tr|A0A0K0YAR4|A0A0K0YAR4_9INFA N6  DPGVKGFAFLDGENSWLGRTISKDSRSGYEMLKVPNAETD-TQSGPTSYQLIVNNQNWSG  394
tr|A6M7W4|A6M7W4_9INFA        N3  GPGVKGFGFKTGDDVWLGRTVSISGRSGFEIIRVAEGWINSPNHAKSVTQTLVSNNDWSG  404
sp|P09573|NRAM_I83A6          N2  NPGVKGWAFDIGDDVWMGRTISKDSRSGYETFRVIGGWATANSKSQTNRQVIVDNNNWSG  385
                                    *:**:.:  * . *:*** *  .***:* :    .           * :*   :***

sp|Q809V2|NRAM_I01A2          N1  YSGSFVQHPELTGVDCIRPCFWVELIRGRPKES--TIWTSGSSISFCGVNSDTVGWSWPD  459
tr|D1LM97|D1LM97_9INFA        N8  YSGSFTLPVELTRRNCLVPCFWVEMIRGKPEEK--TMWTSSSSIVMCGVDHEIADWSWHD  459
sp|P18881|NRAM_I000F          N7  YSGSFIDCWD-EANECYNPCFYVELIRGRPEEAKYVWWTSNSLIALCGSPVSVGSGSFPD  463
tr|A0A0C4K198|A0A0C4K198_9INFA N9  YSGSFMDYWA-E-GDCYRACFYVELIRGRPKEDK-VWWTSNSIVSMCSSTEFLGQWNWPD  457
tr|A0A0K0YAR4|A0A0K0YAR4_9INFA N6  YSGAFIDYWA-N-KECFNPCFYVELIRGRPKEID-VLWASNSMVALCGSRERLGSWSWHD  451
tr|A6M7W4|A6M7W4_9INFA        N3  YSGSFIV----ENNGCFQPCFYVELIRGRPNKNDDVSWTSNSIVTFCGLDNEPGSGNWPD  460
sp|P09573|NRAM_I83A6          N2  YSGIFSV----ESKSCINRCFYVELIRGRPQE-TRVWWTSNSIVVFCGTSGTYGTGSWPD  440
                                  *** *          *   **:**:***:*::   . *:*.* : :*.        .: *

sp|Q809V2|NRAM_I01A2          N1  GAELPFTIDK-     469
tr|D1LM97|D1LM97_9INFA        N8  GAILPFDIDKM     470
sp|P18881|NRAM_I000F          N7  GAQIQYFS---     471
tr|A0A0C4K198|A0A0C4K198_9INFA N9  GAKIEYFL---     465
tr|A0A0K0YAR4|A0A0K0YAR4_9INFA N6  GAEIIYFK---     459
tr|A6M7W4|A6M7W4_9INFA        N3  GSNIGFMPK--     469
sp|P09573|NRAM_I83A6          N2  GANINFMPL--     449
                                  *: : :
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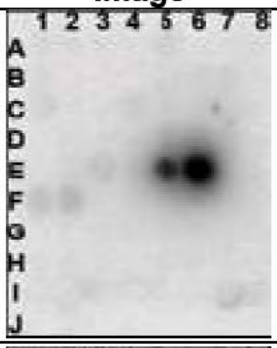
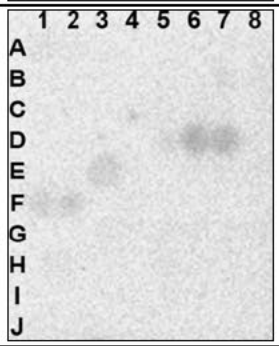
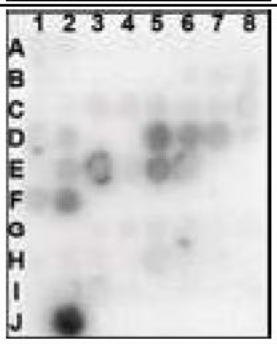
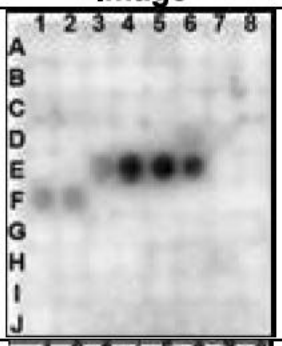
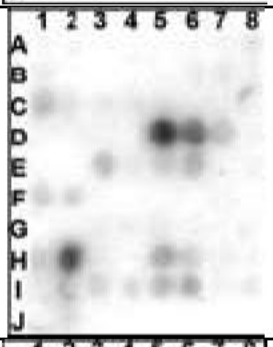
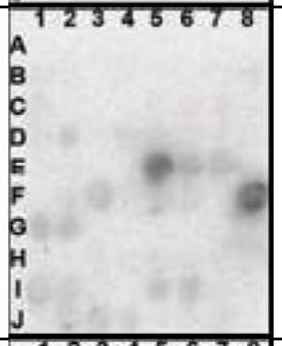
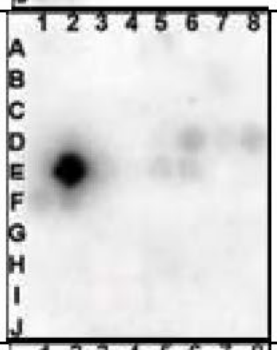
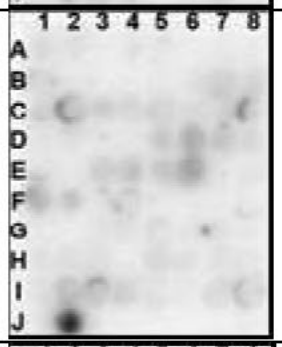
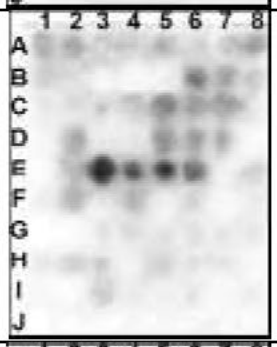
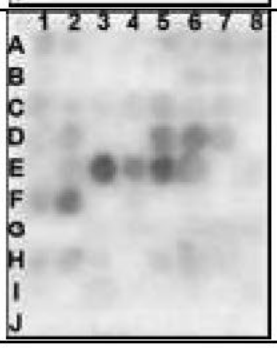
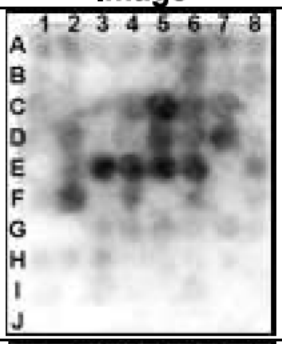
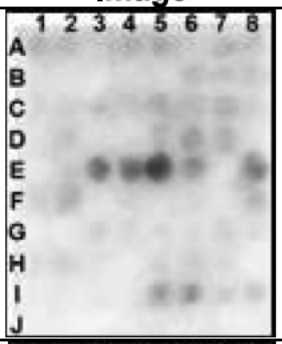
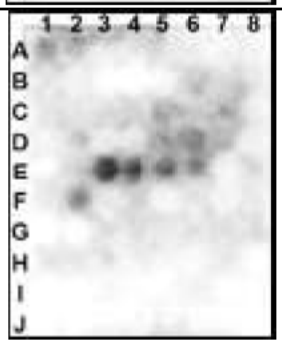
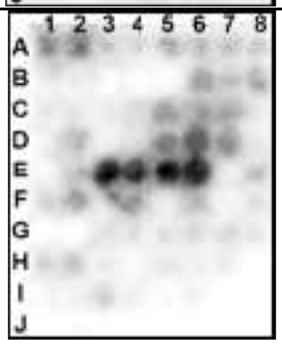
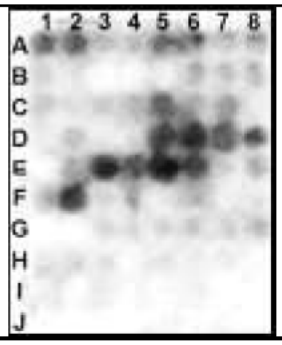
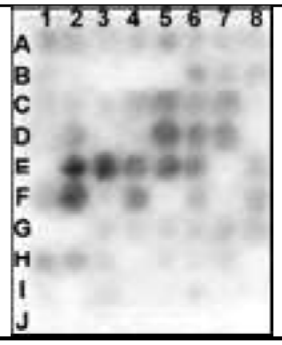
53. Figure 3 shows the raw testing results with the H5N1-specific SPOT array developed by Kinexus using egg yolk samples from 18 different UOF ostriches that were sampled in the summer of 2024. These findings conclusively show that these ostriches possessed antibodies that could bind to multiple parts of the H5 and N1 proteins of the H5N1 avian influenza virus, which is strongly

indicative of prior exposure to the virus, and can account why most of the birds did not succumb to the subsequent infection with H5N1 in December 2024 up to January 15, 2025, and none afterwards. As noted for the SARS-CoV-2 antibody response in humans, the patterns of antibody response to H5N1 appears to be unique in each ostrich.

Figure 3. H5N1 peptide SPOT arrays probed for IgY antibodies with ostrich egg yolk samples originally obtained in June, July and August of 2024. Testing was performed between January 31 and February 7, 2025. The appearance of a dark spot indicates positive immunoreactivity with a 14 amino acid long peptide derived from the H5 hemagglutinin protein (Spots A1 to E7) and N1 neuraminidase protein (Spots E8 to J2). In some cases, the peptides in adjacent spots shared 11 of the 14 amino acids. For example, Spot E3 (FEAVGREFNNLERR), Spot E4 (VGREFNNLERRIEN), Spot E5 (EFNNLERRIENLNK) and Spot (NLERRIENLNKKME), which were commonly immunogenic, encompassed a 23 amino acid stretch in the H5 hemaglutinin protein. The peptide in Spot I6 (TDSSFSVKQDIVAI) was the most commonly immunoreactive sequence from the N1 neuraminidase protein. The anti-chicken IgY secondary antibody weakly reacted directly with the peptides in Spots D6, D7, E3, F1 and F2, but none of the other peptides on the array.

Information	Image	Information	Image
KPL01CDY-01a T-TBS (neg. control) T-TBS (neg. control) – Background Control for anti-IgY antibody binding		KPL01CDY-05a H July 11, 2024 P5 H	
KPL01CDY-03b T-TBS (neg. control) Background Control for anti-IgY antibody binding		KPL01CDY-06a P5 T2 D1 July 21, 2024 P5 T2 D1	

Information	Image	Information	Image
KPL01CDY-02a A July 5, 2024 P5 A		KPL01CDY-07a T July 14, 2024 P5 T	
KPL01CDY-03a A1 July 19, 2024 A1		KPL01CDY-08a T2 Aug 19, 2024 P4 T2	
KPL01CDY-04a B July 6, 2024 P5 B		KPL01CDY-09a P5 PRE ANTIBODY June 20, 2024 P5	
KPL01CDY-11a W2 Aug 5, 2024 P5 W2		KPL01CDY-05b L July 15 2024 P4 L	
KPL01CDY-12a R July 14, 2024 P5 R		KPL01CDY-06b N July 14, 2024 P4 N	

Information	Image	Information	Image
KPL01CDY-01b D July 9, 2024 P4 D		KPL01CDY-07b O July 13, 2024 P5 O	
KPL01CDY-02b P4 E July 9, 2024 P4 E		KPL01CDY-11b X July 17, 2024 P4 X	
KPL01CDY-04b J July 11, 2024 P4 J		KPL01CDY-12b Y July 18. 2024 P4 Y	

54. It should be appreciated that the Kinexus H5N1 antibody test was specifically designed to distinguish previous H5N1 infections from other avian influenza virus infections. Many more peptide sequences of H5 and N1 could have been tested that would likely have cross-reacted with the hemagglutinin and neuraminidase proteins of these other influenza strains. This would have further demonstrated a high degree of immune protection against future influenza infections, which would include other mutated forms of H5N1.

55. Without further exposure to a pathogen, the levels of antibodies against the pathogen will decline over time, but the B-cell that produce these antibodies still remain as plasma or memory B-cells. These B-cells will rapidly multiple and generate more antibodies as needed. Re-exposure

to the H5N1 virus at the end of last year and the beginning of 2025 likely boosted the levels of the anti-H5 and anti-N1 IgY levels to much higher than what was evident in our tests of the egg yolks from last summer.

56. Any future encounters of the ostrich herd at the UOF site with wild birds infected with the H5N1 virus at this point is likely to permit natural boosting the anti-H5N1 antibody levels in the ostriches with causing them to be sick. Any requirements to prevent wild birds from frequenting the UOF site, for example by netting ponds, or restricting access to the bird feed, would appear to me to be unnecessary. Rather, it would be better for the wild birds to come to the UOF site than another site where the herd immunity against the H5N1 virus has not been established. Infected wild birds are highly unlikely to successfully transmit the H5N1 virus to the ostriches, and vice versa.

57. Since the remaining UOF ostriches have successfully developed resistance to the H5N1 virus, they could be valuable breeding stock for the propagation of future ostriches that genetically are less prone to become infected and sick. While previous history of survival from an infection and development adaptive immunity is a major factor in herd immunity, careful breeding can also contribute to improved immunity to a pathogen.

58. Another important consideration in preserving the UOF ostriches is that they are already an excellent source of industrial scale production of anti-H5 and anti-N1 antibodies in their eggs. One ostrich hen can produce up to 100 eggs in a season. Using the knowledge from the Kinexus H5N1 SPOT array study, these antibodies can be affinity-purified and used to develop rapid antigen tests for diagnosis of H5N1 outbreaks in chicken, cattle, pigs and minks. These ostrich antibodies can also be purified for therapeutic purposes to isolate neutralizing antibodies that specific block the binding of the virus to host cell receptors.

59. The CFIA should be seizing the opportunity to use the UOF site to obtain important information about the establishment, effectiveness and durability of protective herd immunity. The longevity and careful tracking of these valuable ostriches on an isolated farm as birds that have no flight risk make this an ideal location for further research.

60. It is evident from the continuous outbreaks of H5N1 on commercial chicken farms in Canada, and recently in cattle in the US, that the policy of testing, culling and compensation is not working with at least 523 outbreaks in Canada and no sign of this abating as long as migrating wild birds carry the H5N1 virus.[15] It may be many years before herd immunity against H5N1 is established in the wild bird populations. It is time to explore other strategies to mitigate the threat of the H5N1 virus. History with influenza vaccines has shown that at best 50% of influenza infections in people and animals can be achieved with vaccines. However, natural immunity is much more effective against future infection and transmission.

Respectfully submitted by,

Steven Pelech, Ph.D.

Professor,
Department of Medicine,
University of British Columbia

President and Chief Scientific Officer,
Kinexus Bioinformatics Corporation

Vice-President, and Co-Chair,
Scientific and Medical Advisory Committee,
Canadian Citizens Care Alliance

Exhibit A

CLEVELAND DOAN
Barristers & Solicitors

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Phone 604 536 5002
File No. 26408

February 8, 2025

VIA EMAIL

Dr. Steven Pelech
University of British Columbia
Department of Medicine, Division of Neurology
2775 Laurel Street
Vancouver, BC V5Z 1M9

Dear Dr. Pelech,

Re: Medical Opinion regarding Universal Ostrich Farms Ltd.

We write to request a supplement to your report dated January 29, 2025. In the questions set out below we wish to further clarify the opinion you provided to the following question: "*[w]hat is the likelihood that the [ostriches] presently [are] transmissible for H5N1 to each other and wild migratory birds such as ducks*" (ie, paragraphs 52 – 61 of your report).

In addition to the facts set out in our letter to you dated January 27, 2025, you may rely on the following facts:
1) There have been no additional ostrich deaths from H5N1 type symptoms; and

2) The ostriches are not showing any signs of illness and appear healthy.

In the questions below I have assumed that the terms "live virus" and "circulating virus" are synonymous, with the term "live virus" being the 'layperson's' term. If that assumption is not correct please clarify those terms in your report.

Requested Opinion
In your supplemental report please provide your opinion on the following questions:

1. What is the risk that the ostriches may be asymptomatic and still actively replicating, mutating and shedding the virus?

2. If there is no circulating virus in the ostriches, what is the risk of the following occurring:

a. the ostriches facilitating mutation of the virus;

b. the virus being transmitted to humans;

c. the virus serving as precursor to a human flu pandemic;

d. the ostriches becoming infected with more than one subtype of the virus, which may allow HPAI variants to mix with other circulating influenzas creating new combinations; and

e. the ostriches contributing genetic mutations to AI viruses that may increase viral adaptability to mammalian hosts.

Yours truly,
CLEVELAND DOAN LLP
Per:
MICHAEL D. CARTER