THE UNIVERSITY OF BRITISH COLUMBIA

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Date: 13 March 2025

Re: Expert 2nd Follow-up 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 SOURCES OF THE INFORMATION CONSIDERED

1. I have previously provided two expert reports dated January 29, 2025, and February 12, 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 highly pathogenic avian influenza (HPAI). I am responding in this second follow-up expert report in view of new information that has been disclosed on March 7th by the Canadian Food Inspection Agency (CFIA) in four respondent affidavits and their appendices, which are from:

    a. Dr. Abed Harcharoui, Affidavit 1 dated March 6, 2025;

    b. Dr. Sumnder Sawhney, Affidavit 1 dated March 6, 2025;

    c. Dr. Cathy Furness, Affidavit 2 dated March 7, 2025; and

    d. Dr. Shannon French, Expert report dated March 7, 2025.

2. I will respond in the above order to several of the statements that have been provided in these documents, and after reading some of the citations to which they have referred. However, before doing so, I will comment on what appears to be main impetus for the CFAI to order the culling of the ostrich herd at the UOF.

PART 2: SUMMARY OF KEY ISSUES

3. I note that the CFAI is not inclined to go against guidelines set by an international organization, namely the World Organization for Animal Health (WOAH), which is loosely associated with the World Health Organization (WHO). Dr. Harchaoui goes into detail about this in his affidavit. Basically, as long as a single bird in a commercial flock has tested positive for H5N1 influenza virus in a polymerase chain reaction- (PCR-) based test, all members of the flock that have been in contact with the infected birds must be culled, whether or not they show signs of infection and even complete recovery. This is known as a "stamping out policy." This policy is viewed by the WOAH and the CFAI as the most effective strategy to regain the status of "influenza free" for Canada or zones within the country that wish to trade products from farmed birds internationally. There are narrow exceptions to the "stamping out policy" primarily around whether the birds have unique genetic aspects, such as being part of an endangered or rare species. The CFAI determined that such an exception does

not apply to ostriches. It is evident to me from reading the original CFAI guidelines that they were not drafted with ostriches in mind.

4. The situation with the UOF ostriches is rather unique.

    a. Unlike typically farms in Canada that collectively raise tens of millions of chickens, turkeys, geese or ducks (see Exhibit "F" of Dr. Sawhney's affidavit for exported birds), the UOF has about 390 ostriches that represent a significant portion of all of the ostriches in Canada, which likely numbers in only a couple thousand.[1]

    b. The ostriches are expensive to procure, and it takes years for them to adjust comfortably to handling by an individual caretaker. Most of the birds at the UOF are over 20 years of age.

    c. While ostriches are birds, they have many unique features, including being the largest avian species and with the largest eggs. They can live up to 75 years of age and be reproductive for up to 55 years, laying on average 30 or more fertile eggs per season.

    d. The UOF is also remotely located in central British Columbia, and not in a zone associated with other commercial scale operations involving birds.

    e. The UOF ostriches and their eggs are not being used for human consumption, but for research purposes and to create products that may be useful in the detection of infectious pathogens and treatment of infectious diseases, such as SARS-CoV-2 and COVID-19.

    f. Serological testing of the UOF birds from eggs samples that were performed by Kinexus Bioinformatics (described in my February 12, 2025 expert report) revealed that these birds already had a high degree of specific antibodies for unique H5 and N1 protein-based peptides prior to the recent outbreak at the UOF site. Antibodies against H5 hemagglutinin and N1 neuraminidase influenza viral proteins were tested for in the yolks of 18 ostrich eggs produced at the UOF farm in the summer of 2024.

    g. This may account for why none of the birds that were originally in the UOF herd prior to 2021 succumbed to an infection with the H5N1 virus in December, 2024 and January, 2025

    h. All of the ostriches at the UOF site appear to be healthy since January 15, 2025. Within 2 weeks of H5N1 infection, birds are considered to no longer be infectious as their viral loads are rarely detectable after this time.[2] This supports the contention that the ostrich herd has robust natural immunity, which should protect them against future infections. It is also likely that these birds do not pose a significant risk for transmission of the H5N1 influenza virus to other wild or domesticated animals.

PART 3: COMMENTS RELATED TO THE AFFIDAVIT OF DR. ABED HARCHAROUI

5. My responses to Dr. Harcharoui's affidavit are brief, since he largely outlines Canada's commitment to following the guidelines of the WOAH for economic reasons. He also appears to have some role in the establishment of

[1] Collins, T. (2018) Eastern Ontario: Ostrich farming a niche market one farmer has managed to sustain. Farmers Forum. https://farmersforum.com/eastern-ontario-ostrich-farming-a-niche-market-one-farmer-has-managed-to-sustain/

[2] Olivier, A.J. (2006) Ecology and epidemiology of avian influenza in ostriches. Developments in biologicals. 1;124:51-7. https://www.researchgate.net/publication/7327440_Ecology_and_epidemiology_of_avian_influenza_in_ostriches

these guidelines. He notes in para. 8 of his affidavit that "*Canada has implemented a stamping-out policy in response to AI since the first outbreak of AI in Canada, which occurred in 2004 in British Columbia*." The most recent plan was adopted in 2022, and is entitled "HPAI Event Response Plan 2022" (provided as Tab 1 of his affidavit).

6. Dr. Harcharoui noted in para. 27 to 29 that the CFIA has considered alternatives to stamping out, including allowing a "*virus to run its course or 'burn out' on an infected premises, and a 'selective killing' approach.*" However, this has not been practiced, because the CFIA has had sufficient resources to implement stamping out and it was deemed preferable because of the risks it believed to be associated with allowing the virus to burn out.

7. In para. 43, Dr. Harcharoui identified that the 2013 version of the Notifiable Avian Influenza Hazard Specific Plan (NAI HSP) provided the following policy guidance regarding exemptions:

> *"Once NAI infection has been declared, the CFIA may form an exemption team, comprising local and national experts who evaluate exemption requests to preserve rare genetics as long as they are negative on the NAI surveillance testing. Various parameters, such as the* ***species****, breed, genetic value, and other criteria, will be assessed to determine whether or not there should be exclusion from an order of destruction, if the bird or group of birds is deemed of sufficient value."* [bolding is mine]

8. In para. 44, he further states that to his "*knowledge, an exemption has never been granted in a flock that previously tested positive for HPAI*."

9. One wonders that if ostriches are indeed a rare species in Canada, why this would also not be sufficient to be classified a distinct unit alone? This is in the interests of food security and the potentially important role that the UOF ostrich herd could play in the development of innovated new products, including diagnostics and therapeutics for infectious diseases.

10. I noted in Exhibit "C" of Dr. Harcharoui's affidavit, which related to a 2022 update of HPAI distinct unit recognition process and criteria, that it does not seem to have been developed with ostriches in mind. On page 35 on the pdf copy of Dr. Harcharoui's affidavit, it mentions that "*The distinct unit galliformes such as turkeys or chickens (i.e. does not contain anseriformes such as ducks and geese*)." Ostriches are not galliformes, but of the Struthionidae family.

PART 4: COMMENTS RELATED TO THE AFFIDAVIT OF DR. SUMINDER SAWHNEY

11. As for Dr. Harcharoui, affidavit my comments related to Dr. Sawhney affidavit are limited.

12. Dr. Sawhney noted in para. 14 that:

> *The Scientific Commission plays a role in the development of WOAH standards for responding to outbreaks of pathogenic diseases in animals. Ultimately, any new standards or changes to existing standards are set by the World Assembly of Delegates at its annual meeting. Canada participates in this standard setting process."*

13. In this regard, I am surprised that he does not seem to be aware that the WOAH has recently changed its policy with respect to influenza disease "free status" following culling of influenza infected commercial flocks in countries and zones. In para. 30 and 31, Dr. Sawhney stated:

*"Criteria for regaining free status following an HPAI outbreak in poultry in a previously free country or zone are provided in Article 10.4.6 of the Terrestrial Code. Free status may be restored **28 days** (two flock-level incubation periods) after the disinfection of the last infected premises, provided that:*

a. a stamping-out policy has been implemented; and

b. surveillance of poultry as described in the Terrestrial Code has been carried out with negative results (post outbreak surveillance, as per Articles 10.4.26 to 10.4.30, and especially Article 10.4.28).

If a stamping-out policy is not implemented, Article 10.4.3. applies and absence of HPAI infected over the past 12 months must be demonstrated to regain free status."

14. The WOAH has just updated its overall strategy to combat H5N1 influenza.[3] The stamping out policy, while presented as a possible strategy, is not a requirement. The new guidelines for disease free status have been available sometime prior to February 23, 2025.[4] It stated in Article 10.4.4 that:

"Country, zone or compartment free from infection with high pathogenicity avian influenza viruses in poultry

A country, zone or compartment may be considered free from infection with high pathogenicity avian influenza viruses in poultry when:

1. *it has been shown that infection with high pathogenicity avian influenza viruses in poultry has not been present in the country, zone or compartment for the past 12 months, although its status with respect to low pathogenicity avian influenza viruses may be unknown; or*
2. *when, based on surveillance in accordance with Articles 10.4.27. to 10.4.33., it does not meet the criteria for freedom from avian influenza but any virus detected has not been identified as high pathogenicity avian influenza virus.*

The surveillance may need to be adapted to parts of the country or existing zones or compartments depending on historical or geographical factors, industry structure, population data, or proximity to recent outbreaks.

*If infection has occurred in poultry in a previously free country, zone or compartment, the free status can be regained **three months** after a stamping-out policy (including disinfection of all affected establishments) is applied, providing that surveillance in accordance with Articles 10.4.27. to 10.4.33. has been carried out during **that three-month period**."*

15. What appears to be new here is that the country has to be disease-free for **3 months**, rather than the previous **28 days**, if stamping out is done, but **one year** if it is not, which is the same as before. Consequently, it is going to be even harder for the Canada and the CFIA's stamping out policy to go for three months without another HPAI infections at the currently rate of new outbreaks and achieve "free status."
16. As of March 5, 2025, the CFIA lists on their website that 30 premises in Canada have active HPAI virus infections and another 497 HPAI infections were previously documented, which has resulting in the culling of 14,498,000

[3] https://www.woah.org/app/uploads/2025/02/web-gf-tads-hpai-strategy-woah.pdf

[4] https://www.woah.org/fileadmin/Home/eng/Health_standards/tahc/2018/en_chapitre_avian_influenza_viruses.htm

birds in Canada.[5] With at least 527 separate outbreaks of HPAI in domestic bird flocks, and a policy of originally 28 days without avian influenza infections for "free status", it would seems that Canada has not held such status for at least 3 years, although a stamping out policy has been in effect since 2004 starting in British Columbia. Moreover, since the HPAI virus remains rampant and unchecked in the wild fowl population, it is hard to envision how Canada would achieve such status, which is now even harder with the 3-month requirement for no cases of HPAI outbreaks. In any event, without the HPAI "free status" it is evident that Canada has still been able to continue its trade in products from commercial poultry operations (see Exhibit "F" of Dr. Sawhney's affidavit for exported bird products).

17. Dr. Sawhney discussed in para. 50 about how Canada would not accept poultry-related products from France, because this country adopted a preventative program that involved vaccination of poultry against HPAI. South Africa ultimately also permitted the vaccination of poultry in their commercial industry, but adopted strict guidelines for those farms that adopted this practice. One issue is that it is difficult to distinguish the production of antibodies in vaccinated birds from the vaccines and from natural infection. Influenza vaccines also have a significant breakthrough infections, by moderating the signs of infections, infected birds may be more able to transmit the disease.

PART 5: COMMENTS RELATED TO THE AFFIDAVIT 2 OF DR. CATHY FURNESS

18. In para. 6 of Dr. Furness's affidavit, she noted that she is involved in Canada's planning and preparedness for HPAI in dairy cattle. Although there are no reported cases of HPAI in cattle in Canada, there have been outbreaks in 17 states in the US that has resulted in around 966 infected herds of dairy cows since March 25, 2024.[6] In the US, the policy of stamping out is not implemented for HPAI-infected dairy cattle, but rather they are isolated, treated for their illness and allowed to recover. It begs the question of whether the CFIA will follow the US lead with respect to not stamping out HPAI-infected dairy cattle in Canada. However, like cattle, ostriches are large and expensive animals, and one should expect consistency. HPAI infected cattle pose a greater risk to human health than ostriches, since they are mammals like humans.

19. In para. 31 of Dr. Furness's affidavit, she mentioned that "*approximately half of over 900 human cases of H5N1 reported globally since 1997 have been fatal*" and provides Exhibit "E" as a reference. Likewise, in para. 32, she stated that "*WHO similarly reports that 466 of 964 cases reported globally since 2003 have been fatal*" and cites Exhibit "F" as a reference. Evidently, there have been more reports of human deaths associated with H5N1 influenza with the WHO since 2003 than from the National Collaborating Centre for Infectious Disease (NCCID) since 1997. However, these fatality rates are over-estimates, because mild infections are usually undetected or under-reported. The NCCID document notes that "*as of January 20, 2025, a total of 964 confirmed human H5N1 Al A infections and 466 deaths were reported to the WHO between 2003-2025, including 103 cases and 11 deaths since 2020*" (Exhibit "E", page 67/498). While this information is published on the WHO website,[7] it is not referenced.

20. Exhibit "F" of Dr. Furness's affidavit provides a breakdown of the human cases and deaths associated with H5N1 influenza in countries around the world. Of the 964 human cases of H5N1 influenza since 2003, six countries (Cambodia, China, Egypt, Indonesia, Thailand, and Vietnam) account for 842 reports (87%) and

[5] https://inspection.canada.ca/en/animal-health/terrestrial-animals/diseases/reportable/avian-influenza/latest-bird-flu-situation/status-ongoing-response

[6] https://www.avma.org/resources-tools/animal-health-and-welfare/animal-health/avian-influenza/avian-influenza-virus-type-h5n1-us-dairy-cattle

[7] https://www.who.int/news-room/questions-and-answers/item/influenza-h5n1

446/466 deaths (92%). Of the 11 deaths since 2020, 6 of these were in Cambodia. Since 2003, there have been only 5 reported human H5N1 cases and 1 death (prior to 2015) in Canada. The low incidence of H5N1 influences cases in human from any animal source for more than 20 years indicates how rare serious human illness arises from this virus, especially in North America and Europe. In the US, there has recently been 70 human cases of HPAI H5N1, with one fatality of a 65 year-old male with comorbidities, so it does not have the ~50% human fatality rate that has been inferred by Dr. Furness.

21. In para. 45, Dr. Furness re-iterated that "*the* [WOAH] *Terrestrial Code states that a country or zone can regain "free status" from HPAI a minimum period of 28 days after a stamping-out policy has been completed.*" Like Dr. Sawhney, she does not appear to be aware of the change in the requirement to 3 months without any evidence for further outbreaks before a country or zone can regain "free status" from HPAI after completion of a stamping out action. This was previous detailed above in paragraphs 13 to 15 of this report.

22. In para. 51, Dr. Furness argues that due to the stamping out policy, that "*a relatively small number of poultry premises in Canada have become infected with HPAI since the start of the 2021 outbreak, despite the fact that habitats and migratory pathways for wild birds span across the country*." With some 527 flocks comprised of 14.5 million birds that have been culled in Canada as a result of the stamping out policy, this is hard to qualify as a success. For one thing, we have no comparables in domestic poultry flocks as to the lethality if the infected birds were allowed to recover and achieve herd immunity. In the wild bird population in Canada, it is difficult to assess the overall lethality from the H5N1 clade 2.3.4.4b virus. It has been estimated that at least 40,000 wild birds across 45 species died within a few months of the first detection of this virus in Eastern Canada.[8] However, it has been estimated that there are 2 to 4.6 billion birds in Canada depending on the season.[9] Consequently, unchecked in the wild bird population, the mortality rate from the HPAI still seems relatively low. In addition, the rate of outbreaks of HPAI in Canada in commercial poultry would appears to somewhat stable on a seasonal basis over the last two years as compared to the first year of the H5N1 clade 2.3.4.4b outbreak as shown in Figure 1.

23. In para. 62, Dr. Furness stated that she is not aware of any currently available treatment that would effectively eliminate or prevent the spread of the disease within or from" an infected flock of birds with HPAI. However, there are several neuraminidase inhibitors that have been commonly used to treat influenza infections, including oseltamivir (also known as Tamiflu), peramivir and zanamivir.[10] There are some mutations of H5N1 influenza neuraminidase (*e.g.,* H275Y, which is rare in the H5N1 clade 2.3.4.4b) that have been shown to decrease the sensitivity to oseltamivir and peramivir, but not to zanamivir.[10] These are just a few of the drugs that used in combination with other treatments (*e.g.,* amantadine and baloxavir marboxil, which is an endonuclease inhibitor) may significantly reduce the incidence of death from HPAI.

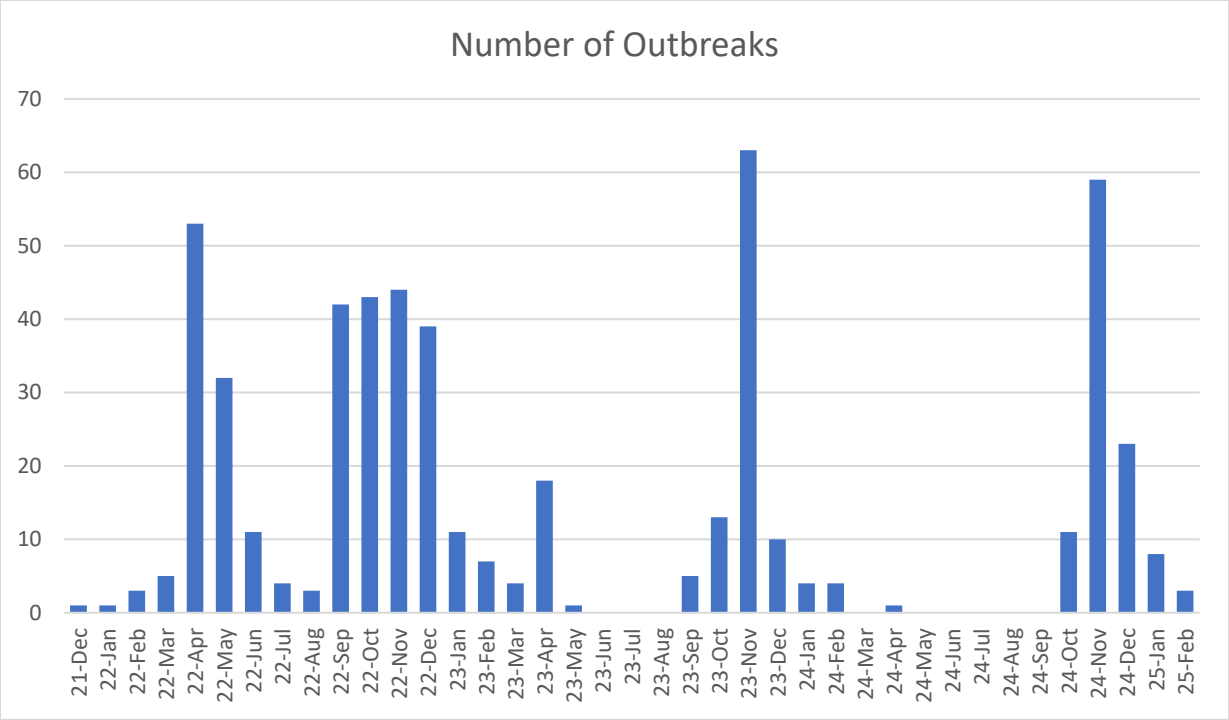
Figure 1. Monthly frequency of HPIA outbreaks in Canada (includes non-commercial, and no-poultry as well) from December 2021 to February 2025. Data was retrieved from the CFAI website.[11]

[8] Avery-Gomm, S.A., Barychka, T., English, R., Roconi, R.A., Wilhelm, S.I., *et al*. (2024) Wild bird mass mortalities in eastern Canada associated with the Highly Pathogenic Avian Influenza A(H5N1) virus, 2022. Echosphere. 15(9):e4980. https://doi.org/10.1002/ecs2.4980

[9] https://www.birdful.org/how-many-birds-live-in-canada/

[10] Signore, A.V., Joseph, T., Ranadheera, C., Erdelyan, C.N.G., Alkie, T.N., et al. (2025) Neuraminidase reassortment and oseltamivir resistance in clade 2.3.4.4b A(H5N1) viruses circulating among Canadian poultry, Emerg Microbes Infect. 18:2469643. doi: 10.1080/22221751.2025.2469643. [Note that this is a publication from CFAI scientists] https://www.tandfonline.com/doi/epdf/10.1080/22221751.2025.2469643?needAccess=true

[11] https://inspection.canada.ca/en/animal-health/terrestrial-animals/diseases/reportable/avian-influenza/latest-bird-flu-situation/investigations-and-orders#dataset-filter



24. Dr. Furness noted in para. 63 that "*Under CFIA policy, stamping¬out would still be applied to all birds infected with HPAI, vaccinated or not*." She suggested that vaccination against HPAI in advance may help vaccinated birds from becoming infected by reducing their susceptibility to infection and also transmission by reducing the shedding of active virus if infected. While an infected human or animal that does not have clinical signs of infection is less likely to transmit the virus, recent experience with COVID-19 has demonstrated that vaccinated humans with COVID-19 illness produce SARS-CoV-2 viral loads that are no different than from unvaccinated people.[12,13] It remains to be established whether this is also true for vaccine breakthrough HPAI infections. The best protection against future infections appears to be from recovery from a natural infection, which is not at all considered with the current CFAI policy or the WOAH guidelines in Chapter 10.4 of the Terrestrial Code. Presumably, if a bird can be demonstrated to already possess antibodies that immunoreact with H5N1 viral proteins, and they are not sick or PCR- or rapid antigen test-positive for the virus, even if others in the flock have evidence of a HPAI infection, it is illogical to cull them too.

25. In para. 67, Dr. Furness reiterates that "*quarantine and the use of additional testing are not "treatment" within the meaning of subsection 48(2) of the HAA, and are also not considered by CFIA to be an appropriate mitigative measure for HPAI."* Again, this disregards acquired natural immunity, which should be potential higher in long-lived birds such as ostriches. I suspect that this is not such a hard and fast policy when it comes to larger animals such as dairy cows should they become infected with HPAI virus.

26. In para. 78, Dr. Furness finally disclosed that the specimens collected from the deceased ostriches from the UOF site were subjected to full genome sequencing of the 8 chromosomes of the HPAI virus. This is why the

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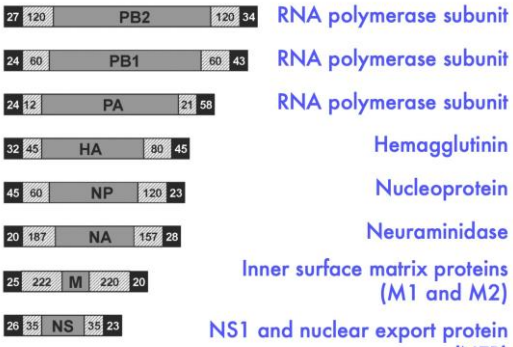
[12] Riemersma, K. K., Haddock, L. A 3rd, Wilson, N. A., Minor, N., Eickhoff, J., *et al.* (2022) Shedding of infectious SARS-CoV-2 despite vaccination. PLOS Pathog. 18(9):e1010876. doi:10.1371/journal.ppat.1010876.

[13] Franco-Paredes, C. (2022) Transmissibility of SARS-CoV-2 among fully vaccinated individuals. Lancet Infect Dis. 22(1):16. doi:10.1016/S1473–3099(21)00768–4.

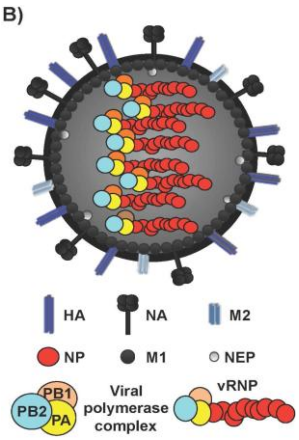
CFAI had suggested without any clarification of the methodology used that "*the HPAI strain detected in the ostriches at UOF represents a unique reassortment (genotype) of H5N1 not previously identified in Canada.*" This analysis was apparently undertaken in early January, 2025, and the data was available to the CFAI on January 8, 2025, based on the time stamp of the report (Exhibit "T"). It is unclear whether this viral genome sequencing was performed on the samples from both ostriches separately or mixed together, although it seems that the samples were pooled. If both birds had the same reassortment, this increases the prospects that the wild duck that originally infected the flock may have already had this reassortment. According to this report, from the result of the 2 vials/submitter samples, it is indicated that "*genes segments PB2, PA and NP belonging to North American lineage and gene segments PB1, HA, NA, M and NS belonging to Eurasian lineage*" were identified. This means that five of the viral chromosomes arose from a high pathogenicity avian influenza virus (*i.e.,* H5N1 2.3.4.4b clade) and three of the viral chromosomes arose from a low pathogenicity avian influenza virus (not specified in the report). Figure 2 provides a diagram of the genetic organization of the influenza virus genome. More information about the reassortment of influenza A virus chromosomes is available in Dadonaite *et al.* (2019).[14]

Figure 2. **Influenza A virus (IAV) genome organization and virus particle (virion) structure.** (**A**) Genome organization: The eight single-stranded, negative-sense, viral (v)RNA segments PB2, PB1, PA, HA, NP, NA, M and NS of IAV are shown. M encodes the inner surface envelop matrix 1 (M1) protein and the ion channel matrix 2 (M2) protein. NS encodes the NS1 protein and nuclear export protein (NEP). The Black boxes at the end of each of the vRNAs indicate the 3′ and 5′ non-coding regions (NCR). Hatched boxes indicate the packaging signals present at the 3′ and 5′ ends of each of the vRNAs that are responsible for efficient encapsidation into nascent virions. Numbers represent nucleotide lengths for each of the NCR and packaging signals; (**B**) Virion structure: IAV is surrounded by a lipid membrane bilayer containing the two viral glycoproteins hemagglutinin (HA), responsible for binding to sialic acid-containing receptors; and neuraminidase (NA), responsible for viral release from infected cells. Also in the virion membrane is M2 protein. Under the viral lipid bilayer is a protein layer composed of M1 protein, which plays a role in virion assembly and budding; and NEP involved in the nuclear export of the viral ribonucleoprotein (vRNP) complexes. Underneath is the core of the virus made of the eight vRNA segments that are encapsidated by the viral nucleoprotein (NP). Associated with each vRNP a complex is the viral RNA-dependent RNA polymerase (RdRp) complex made of the three polymerase subunits PB2, PB1 and PA that, together with the viral NP are the minimal components required for viral replication and transcription. This figure and information are reproduced from Breen *et al.* (2016).[15]

A)



B)



[14] Dadonaite, B., Gilbertson, B., Knight, M.L., Trifkovic, S., Rockman, S., *et al.* (2019) The structure of the influenza A virus genome. Nat Microbiol. 4(11):1781-9. doi: 10.1038/s41564-019-0513-7. https://pmc.ncbi.nlm.nih.gov/articles/PMC7191640/

[15] Breen, M., Nogales, A., Baker, S.F., Martinez-Sobrido, L. (2016) Replication-competent influenza A viruses expressing reporter genes. Viruses. *8*(7):179. https://doi.org/10.3390/v8070179

27. More insight into the results of the genome sequencing of the H5N1 influenza virus recovered from the two UOF ostriches that died and were tested by the CFAI is revealed in an internal January 4, 2025 e-mail memo between CFIA AI Lab Liaison to CFIA AI Commander (see Appendix A for full memo). In particular, it is stated:

> *"The entire genome of the virus was amplified from two original swab samples and submitted to our Genomics group for NANOPORE sequencing. Eight gene segments of the virus were sequenced. The HA of the virus from the samples belong to Eurasian Gs/GD lineage HPAI H5N1 (2.3.4.4B) with cleavage site motif of "PLREKRRKR/GLF" compatible with HPAI viruses that came to Canada via the Pacific flyway. The H5N1 virus is a reassortant virus with PB2, PA, and NP originated from North American lineage low pathogenic avian influenza virus, and PB1, HA, NA, M and NS gene segments from Eurasian viruses. The virus is similar to the D1.1 viruses circulating in North America, but has the neuraminidase segment identical to WIN-AH-2022-OTH-0033 virus. PB2 627E (avian)."*

28. This correspondence confirms that three of the chromosomes (specifying the PB2, PA and NP genes) found in the retrieved samples of the influenza strain were derived from a low pathogenicity strain of avian influenza virus. It also indicates that the PB2 gene feature the E627 allele of this RNA polymerase subunit. This is important, because it is the E627K variant that has been linked with more pathogenic strains of H5N1 that can infect mammals.

29. The PB2 protein is a subunit of the viral RNA polymerase, and it has also been implicated in inhibition of interferon expression by associating with the mitochondrial antiviral signaling (MAVS) protein.[16] The avian version of PB2 does not normally support mitochondrial import of this protein, and its ability to suppress immune responses via inhibition of PB2. The normal PB2-E627 form of the avian H5N1 is highly impaired in its ability to infect mammals.[17] However, this is compromised with PB2 E627K or D701N mutations.[18] It appears that the PB2 gene in the virus isolate from the UOF ostriches does not contain the E627K mutant, which the CFAI has been concerned increases infectivity of humans.

30. The bottom line is that the variant of H5N1 detected in the two ostriches was likely evolved from a mutated form of the H5N1 influenza virus that contains the surface proteins of the high pathogenicity, 2.3.4.4b clade of the avian influenza virus, which was already present in the infected ducks that transmitted the virus to the ostriches. However, due to the substitution of the three of the viral genes for internal proteins in the influenza particles that are critical for its replication of the virus with variants from a less pathogenic strain, it has lower virulence than found with the 2.3.4.4b clade. It is also less likely to cause illness in humans and other mammals that are infected, because it does not feature the PB2 – E627K mutation.

31. The emergence of a less virulent but more infectious variants of a virus, as may be the case with the medium pathogenicity strain of H5N1 influenza virus in the UOF ostriches, is typical. With reduced pathogenicity, the infected host is less sick and more likely to successfully transmit the virus to other hosts. However, because it

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[16] Long, J.C., Fodor, E. (2016) The PB2 subunit of the Influenza A virus RNA polymerase is imported into the mitochondrial matrix. J Virol. 90(19):8729-38. doi: 10.1128/JVI.01384-16. https://pmc.ncbi.nlm.nih.gov/articles/PMC5021425/

[17] Bogs, J., Kalthoff, D., Veits, J., Pavlova, S., Schwemmle, M., *et al.* (2011) Reversion of PB2-627E to -627K during replication of an H5N1 Clade 2.2 virus in mammalian hosts depends on the origin of the nucleoprotein. J Virol. 85(20):10691-8. doi: 10.1128/JVI.00786-11. https://pmc.ncbi.nlm.nih.gov/articles/PMC3187502/

[18] Steel, J., Lowen, A.C., Mubareka, S., Palese, P. (2009) Transmission of influenza virus in a mammalian host is increased by PB2 amino acids 627K or 627E/701N. PLoS Pathog. 5(1):e1000252. doi: 10.1371/journal.ppat.1000252. https://pmc.ncbi.nlm.nih.gov/articles/PMC2603332/

features nearly identical viral surface proteins, the immunity that it induces in the host protects it well from future infections. This phenomenon is exemplified by how the more infectious, but more benign Omicron variants were able to rapidly out compete the earlier SARS-CoV-2 variants during the SARS-CoV-2 pandemic. Thus, the H5N1 variant discovered in the UOF ostriches may actually perform as a natural attenuated vaccine against the more pathogenic versions of the H5N1 influenza virus.

32. In para. 96, Dr. Furness argued that "*testing cannot wholly evaluate the current and future risk of disease spread posed by an entire flock of confirmed infected birds continuing to exist on a known contaminated premises*." Basically, the point of the culling is to prevent the spread of the H5N1 virus. Yet, the CFAI chooses to ignore any evidence that the infected flock no longer is shedding the virus and that natural immunity has been established by way of production of neutralizing antibodies, all of which can be easily tested for. Moreover, it has forbidden any treatment or testing of the ostriches following the culling order that it issued to the UOF owners. It should also be appreciated that it is not necessary to individually test birds for shed virus with PCR- or rapid antigen tests, and pooled samples are sufficient for detection.

PART 6: COMMENTS RELATED TO THE AFFIDAVIT 2 OF DR. SHANNON FRENCH

33. In para. 19, Dr. French made the point that all species of birds are naturally prey animals, even though raptors are predators as well. She cautioned "*that instinctively, they will hide signs of illness or pain to the best of their ability*" and therefore clinical signs of infection can be subtle or absent. However, I do not think that ostriches are prey animals per se. While for example, lions on rare occasions can kill ostriches, with their speed, vigilance, and powerful kicks, present a formidable challenge to such an apex predator, as a lion.[19] They are clearly not defenseless creatures. They are the largest living bird on Earth, capable of reaching impressive speeds over **43 mph** in sprints. This speed, coupled with their exceptional vigilance and powerful legs with strong, sharp claws on their two-toed feet, makes them challenging prey. A well-placed kick from an ostrich can easily injure or even kill a predator. Most predators, including lions, would rather avoid a potentially dangerous and difficult hunt with an ostrich.

34. In para. 21, Dr. French noted that "unlike chickens and turkeys, many ducks (for example a flock only showing decreased egg laying) will recover from the infection." This may also reflect the establishment of a level of natural herd immunity possibly from recovery prior infection with HPIV or a highly related avian influenza strain. With the culling that occurs with domestic chickens and turkeys, such herd immunity is unable to develop.

35. In para. 22, Dr. French cited studies by Abolnik *et al.* (2007)[20] and Van Helden *et al.* (2016)[21] to support the contention that adult ostriches can shed virus without any clinical signs. However, these studies were not based on the HPAI H5N1 strains that are more virulent and likely to cause illness. The Abolnik *et al.* (2007) publication is based on the H6N8 and H9N2 avian influenza viruses and the Van Helden *et al.* (2016) publication is likely based on H5N2. This latter article stated that "*the presence of clinical signs on the farms was included in the case definition but did not play a large role in identifying infected properties, as the majority of positive farms did not subjectively show an increase in morbidity or mortalities of ostriches*." In view of the high rate of false-

[19] https://enviroliteracy.org/are-ostriches-eaten-by-lions/#google_vignette

[20] Abolnik, C., Bisschop, S., Gerdes, T., Olivier, A., Horner, R. (2007) Outbreaks of avian influenza H6N2 viruses in chickens arose by a reassortment of H6N8 and H9N2 ostrich viruses. Virus Genes. 34:37-45. doi: 10.1007/s11262-006-0007-6. https://pubmed.ncbi.nlm.nih.gov/16927114/

[21] Van Helden LS, Sinclair M, Koen P, Grewar JD. Description of an outbreak of highly pathogenic avian influenza in domestic ostriches (Struthio camelus) in South Africa in 2011. Preventive Veterinary Medicine. 2016 Jun 1;128:6-11. https://doi.org/10.1016/j.prevetmed.2016.03.019

positives with the PCR test at the thermal cycle thresholds that were likely used, it is not clear that all of the birds were even necessarily infected.

36. In para. 24, Dr. French cites the publication by Abolnik *et al.* (2021)[22] to support the contention that ostriches without clinical signs of illness can shed influenza virus into the environment. This is based on H7N1 low pathogenic and H5N8 highly pathogenic influenza viruses, and **not** H5N1 high pathogenic influenza, and the infection of 7-week-old ostriches, which are likely to have little immunity initially. Antibodies against H5N8 HPAIV and H7N1 LPAIV only appeared after day 7 post exposure, with higher antibody titres induced by the HPAIV compared to the LPAIV. By 14 days post-infection, there was little if any detectable shedding by PCR testing with 40 thermal cycles. Live virus cultivatable in eggs was not detected from the H7N1 LPAIV if more than 30 thermal cycles was required for amplification of the viral RNA by PCR. The authors concluded that their findings "*show that LPAI and HPAI viruses are unlikely to circulate at low levels or for short periods within ostriches*."

37. In para. 24, Dr. French claimed that "*AIV have been demonstrated to survive for months or **even years** in fresh water at low temperatures, so allowing the virus to spread through a herd has the potential to create a large source of infective virus that will remain in the area even after the individual ostriches have recovered and are no longer shedding the virus themselves*." This based on a single study with a duration of up to a year by Ramey *et al.* (2022)[23] using PCR detection with up to 44 thermal cycles as corresponding to a positive result. While "positive samples" were subsequently tested via inoculation of embryonating chicken eggs, it seems highly improbable that the virus would be stable in a non-sterile environment, and it would likely be highly diluted in a pond or stream so the viral load would be very low. Moreover, this study was performed with the H5N2 influenza virus and not an H5N1 strain.

38. In para. 25, Dr. French argued that mutations can occur in avian influenza viruses that might foster cross-species transmission, especially since ostriches are allowed run more freely and potentially interact with wild animals. Firstly, the rate of mutation of the influenza viruses is dependent on the genome and its encoded proteins, such as the error rate in the viral RNA polymerase complex that facilitates its replication and not so much mutations of host proteins. The rate of mutation in a host species is orders of magnitude much slower than a virus or bacteria. Efficient replication of a virus requires an optimization of the virus in its evolution for the successfully entering and hijack the host's cells. The PB-E276K mutation referred to by Dr. French, which increases infectivity in mammals, was shown by the CFAI **not** to be present in the H5N1 virus RNA isolated from the UOF ostriches as discussed in para. 27 to 29 above.

39. Secondly, historically farms were more open environments where livestock tended to be more mobile and grazed in wide spaces on diverse vegetation. In a natural environment, ecosystems are extremely complex with a high degree of inter-species interactions. In the large factory farms that now dominate in the livestock industry, the animals are often confined and kept in high density, often under stressful conditions. This increases the prospects for infection by a pathogenic virus or bacteria. If we accept Dr. French's argument, then open farms should really be banned outright in general, not just ostrich farms. Moreover, we should also be discouraging the establishment of zoos, game farms, animal shelters and even wild bird refuges, since these

[22] Abolnik, C., Ostmann, E., Woods, M., Wandrag, D.B., Grewar, J., *et al.* (2021) Experimental infection of ostriches with H7N1 low pathogenic and H5N8 clade 2.3. 4.4 B highly pathogenic influenza A viruses. Veterinary Microbiology. 263:109251. https://pubmed.ncbi.nlm.nih.gov/34656859/

[23] Ramey, A.M., Reeves, A.B., Lagassé, B.J., Patil, V., Hubbard, L.E., *et al.* (2022) Evidence for interannual persistence of infectious influenza A viruses in Alaska wetlands. Science of the Total Environment. 210;803:150078. https://royalsocietypublishing.org/doi/full/10.1098/rspb.2020.1680

allow the mixing of diverse species in crowded surroundings. People should also not have pets such as dogs, cats or rodents, since this also increases the chances of the spread of infectious diseases by zoonosis.

40. In para. 28, Dr. French argued that enforcement of a stamping out policy is the best way for "minimizing viral transmission and contamination of the environment, with the final intention of viral eradication." However, with such as wide range of wild animals that can be infected and transmit the H5N1 influenza virus, there is too large a reservoir of the virus to hope to eradicate it from the domestic animal and human populations. The biosecurity measures would have to be so draconian that the domestic animals on the farms would be even more inhumanely treated. Curtailment of the spread of a pathogenic virus or bacteria is more likely achieved by acquisition of natural immunity, breeding between survivors with genetics that confer more resistance to the pathogen, and the natural evolution of the pathogen to more infectious and benign forms.

41. In para. 30, Dr. French suggested that the stamping out policy has successfully stop previous HPAI outbreaks since 1957. However, as illustrated in Figure 1, influenza outbreaks tend to be seasonal, and largely wane over a few months on their own accord even without a stamping out strategy. This is clearly evidence, for example, in the rise of the flu in human populations during the winter months in the Northern hemisphere, and during the summer months in the Southern hemisphere. It is hard to rule out that the displacement of more virulent strains by more benign and infectious strains, and acquisition of herd immunity are not also contributing to a decline in an epidemic or pandemic of a pathogen. Ultimately, it is a matter of debate whether the mass culling of all of the birds in a flock or herd results in better overall welfare, as compared to having a few die and most recovering from an infection with resulting herd immunity and protection from future infections.

42. In para. 39, Dr. French noted that while HPAI illness may start in the respiratory tract in birds, it can progress to the brain and other organs resulting in multiple organ failure. She distinguished this with the actions of HPAI in humans and other mammals, which is primarily respiratory. However, influenza viruses can exert effects in humans and mammals that also results in neurological damage.[24] For example, recently a house cat was infected with HPAI H5N1 virus and it developed neurological damage before it was euthanized.[25]

43. In para. 40, Dr. French largely dismissed the relevance of data transmission, treatment and prevent with vaccines in human influenza pandemics with respect to avian influenza outbreaks. However, a major concern of the CFIA was the possibility with HPAI H5N1 virus for infection, mutation to a more pathogenic form, and the generation of a pandemic in the human population.

44. In para. 42, Dr. French suggested that difficulties associated with the PCR testing protocols identified for the SARS-CoV-2 may not be applicable to a different virus, such as the influenza virus, even though both are respiratory RNA viruses with relatively few genes and similar sized genomes. The fundamental problems associated with PCR tests at high thermal cycle (CT) cut-offs is not virus-dependent. Abolnik *et al.* (2021) showed that with CT requiring 30 or more cycles, there are no replication competent avian influenza virus particles in a sample.[22]

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[24] Jang, H., Boltz, D., Sturm-Ramirez, K., Shepherd, K.R., Jiao, Y., *et al.* (2009) Highly pathogenic H5N1 influenza virus can enter the central nervous system and induce neuroinflammation and neurodegeneration. Proc Natl Acad Sci U S A. 106(33):14063-8. doi:10.1073/pnas.0900096106. https://pmc.ncbi.nlm.nih.gov/articles/PMC2729020/

[25] Naraharisetti, R., Weinberg, M., Stoddard, B., Stobierski, M.G., Dodd, K.A., *et al.* (2025) Highly pathogenic avian influenza A (H5N1) virus infection of indoor domestic cats within dairy industry worker households — Michigan, May 2024. MMWR Morb Mortal Wkly Rep 74:61–5. DOI: http://dx.doi.org/10.15585/mmwr.mm7405a2

45. To my knowledge, the PCR testing for H5N1 performed by the CFIA is not certified. At the time that I prepared my earlier expert reports, the CFIA had not disclosed that it had performed full sequence analysis of the viral genome samples retrieved from the two dead UOF ostriches, and the PCR tests that were described were limited to the H5 gene and did not appear to test for the N1 gene. I agree now that it is highly likely that these ostriches were exposed to HPIV, although the sequencing information revealed that this virus was a likely hybrid from reassortment mixing with low pathogenic avian virus.

46. In para. 43, Dr. French dismissed the utility of rapid antigen tests for the presence of active shed virus from birds of an infected flock, because the CFAI does not use such tests, even though they are more rapid, cheaper and convenient to use than PCR tests. Such H5N1 influenza rapid antigen tests are commercially available, and they should not be ignored as a complementary strategy to monitor viral shedding on-site.

47. In para. 44, Dr. French questioned whether it was possible to develop antigens for antibodies that could distinguish different H5N1 avian virus variants. However, the epitopes for antibody recognition can be as little as a single amino acid in a peptide sequence from an antigen protein. In my own lab, we have routinely developed hundreds of polyclonal antibody preparations from the serum of immunized rabbits that can distinguish a difference of the phosphorylation state for a single amino acid in an otherwise identical peptide sequence.

48. It is completely feasible to develop assays that can distinguish between different mutant forms of the various influenza proteins and even neutralizing antibodies against the hemagglutinin protein. For example, neutralizing monoclonal antibodies were developed against the Wuhan SARS-CoV-2 spike protein that did not work on the Omicron variants. A single amino acid change in an epitope recognized by an antibody in an antigen can result in a profound loss of immunoreactivity.

49. In para. 45, without offering any evidence to the contrary, Dr. French disagreed with my suggestion that the study of the antibody reactivity of the naturally infected UOF ostrich herd would be a good starting point to develop a diagnostic test. This is certainly an approach that we took at my company Kinexus Bioinformatics in development of a 40-marker antibody test (against 8 of the SARS-CoV-2 proteins) that we used in a 4500-person clinical study. This blood test first involved the careful screening over 6000 possible peptide fragments predicted from the amino acids sequences of the 28 proteins encoded by the SARS-CoV-2 genome and testing with the serum from hundreds of patients that were naturally infected with SARS-CoV-2 (usually confirmed by PCR) and had symptoms of COVID-19. As shown in my February 12, 2025 expert report, we were able to identify several portions of the hemagglutinin and neuraminidase proteins of H5N1 that were highly immunoreactive in antibodies tested in the egg yolks from UOF ostriches collected in the summer of 2024.

50. In para. 46, Dr. French correctly pointed out that the sensitivity and specificity of a serological test is dependent on having samples from infected and non-infected birds or their eggs, and performing proper controls. It would be ideal to have samples from animals that were confirmed to be PCR or rapid antigen positive to follow their antibody levels, especially for specific epitopes. However, the policy of the CFIA to not permit the testing of the ostriches during and after the infection has made this difficult. Nonetheless, the recognized basis of immunity is that the human or animal possess antibodies that can clearly recognize a peptide sequence from an infectious pathogen. The more different epitopes unique to the pathogen that are detected in a specimen from the person or animal, the greater can be the confidence that they have recovered from an infection and have future protection from that or highly related pathogens.

51. In para. 49, Dr. French concurred that in her opinion, "*it is reasonably possible that the birds may have no longer been shedding virus by January 29, but it is impossible to say with any degree of certainty. The reality is that we simply do not know what the disease status of the birds was at that time*." Since the birds have continued to show no signs of an ongoing influenza infection at the UOF up to March 7th, when Dr. French

signed her affidavit, and now up to March 13th, the likelihood that the ostrich present a threat for spread of the HPIV only continues to decline.

52. In para. 50, Dr. French noted that in a study of 929 ostriches on South African farms,[26] that "roughly 90% of the flock had antibodies to influenza (had seroconverted)." While 90% of the birds in the herd had evidence of antibodies against the HPAI virus, PCR testing with a positive result was done on only one combined sample. It is likely that most of the birds were infected with the H5N2 or a related virus, and had only low levels of infection that did not cause serious signs in the birds. In this study, the detection of 17% of sera from a farm (identified as Farm AI18) that contained H5-specific antibodies, had no NP-specific antibodies. It also appears that the presence of anti-N2 antibodies was not assessed in this study. Consequently, it is feasible that many of the ostriches tracked and culled may have been infected previously with low pathogenicity H5 influenza strains. It was probably unnecessary to cull this herd.

53. In para. 53, Dr. French acknowledged that "*given all the variables, we simply cannot say with any certainty whether any individual ostriches were transmitting virus on January 29" at the UOF site.*" In view of this lack of knowledge, it would be prudent to perform PCR tests and serological tests on the ostriches before any of these ostriches are culled. This information would be valuable in understanding the effectiveness of natural immunity in this species against the H5N1 influenza virus.

54. In para. 59, Dr. French disagreed that the lack of deaths from the December 2024 – January 2025 infection of the ostriches at the UOF site that were established before 2021 could be due to natural immunity against a previous avian influenza infection. She noted that younger birds are more susceptible to death from infection. However, in my previous expert reports, I did not refer specifically to younger birds, and many of the birds that were added to the herd after 2020 were older birds. Within this group, there appears to be a 16% mortality rate. Since none of the birds on the UOF site prior to 2021 died, this is a 0% mortality rate, and strongly supports natural immunity. However, immune status can only be achieved by actually testing for antibodies, and under control conditions, looking at morbidity and mortality rates when birds are actually infected in a pre-clinical study.

55. In para. 60, Dr. French disagreed that the UOF ostrich herd could actually provide protection to wild birds, at least in a significant way. Surely, if the ostriches have herd immunity, they will not be passing on the virus to virus-naive wild ducks and geese that may land on the farm. I do agree that the ostriches will have little impact on the overall transmission of H5N1 to other commercial livestock whether they were infected or not, or be transmissible or not for the virus in view of the relatively small size of the ostrich herd and the hundreds of thousands of ducks and geese that migrate through BC, and the actual portion of these wild birds that specifically frequent the UOF site. The main value of preserving the UOF ostrich herd is for the valuable information that they provide about the effectiveness and duration of natural immunity, and the important products that can be developed from the production of antibodies in their eggs for a wide range of applications, including the detection and treatment of avian bird flu in animals and humans.

56. In para. 61, Dr. French suggested that even if the UOF ostriches have natural immunity against the particular avian influenza strain that infected them in the December 2024-January 2025 period, they could still become infected with new variants of the influenza virus the following spring. She further suggests that the birds may still harbor the original influenza strain that infected them, and exposure to new variants could trigger further reassortment of their chromosomes to produce additional variants. If the ostrich herd was culled and replaced with new and likely younger ostriches that are naïve to the H5N1 virus, the chances of infection leading to

[26] Abolnik, C., Fehrsen, J., Olivier, A., van Wyngaardt, W., Fosgate, G., Ellis, C. (2013) Serological investigation of highly pathogenic avian influenza H5N2 in ostriches (Struthio camelus). Avian Pathology. 42(3):206-14.

sickness and death is actually much higher than with the existing herd that has already survived the infection outbreak. The sequences of the various influenza virus proteins actually have a high degree of amino acid identity and similarity amongst the various influenza strains. Since infection of the ostriches induces a polyclonal antibody response, it would seem most likely that a high degree of immune protection would be afforded to the birds. Repeated exposures of these long-lived birds would likely naturally booster and further expand their immunity to future influenza variants. It seems highly improbable that the influenza virus would be maintained at even low levels for 6 months to a year in the ostriches to allow for potential recombination with other variants of avian influenza virus the following migratory seasons.

57. In para. 62, Dr. French expressed the concern that AIV-naïve birds could come to the UOF site and become infected by shed virus from the ostriches. However, any avian influenza virus that infected the ostriches would likely have already been placed earlier into the environment by infected migratory wild fowl. It seems much more likely that the AIV-naïve birds will get infected from other wild birds than from the UOF ostriches and certainly at the vast number of other ponds in BC than the two at the UOF site.

58. In para. 63, Dr. French again expressed the concern that the immune protection afforded to the UOF herd following exposure to the H5N1 strain they encountered during the recent outbreak on the farm may be insufficient against a future variant of the influenza virus. The amino acid sequences of the 10 influenza virus proteins actually have a high degree of amino acid identity and similarity amongst the various influenza strains. The mutations that might be generated in the H5N1 strains is usually restriction to less than 1% of the overall primary amino acid structures of the virus's proteins. Consequently, the vast majority of the polyclonal antibodies that recognize avian influenza virus in these birds, along with their T-cell immunity, should still provide strong protection against newer variants.

59. In para. 69, Dr. French questioned whether UOF ostrich herd has acquired “resistance” to the H5N1 virus, despite the evidence of no deaths in the ostriches on the farm in the group on site prior to 2021 and the presence of detectable anti-H5- and anti-N1-specific antibodies in the yolks of the eggs from these ostriches collected in the summer of 2024. No arguments are offered by her to counter the claim that having ostriches that are more resistant to future influenza virus infections would not be useful for preventing further infection and spread. While the repertoire of antibody producing B-cells that an animal is born with is partly generated de novo by mutation *in utero* in the B-cells, it is also partly hereditary.

60. In para. 70, Dr. French pointed out the antibody reactivity in the egg yolks from the ostriches that were tested by Kinexus may not have arisen from a HPAI H5N1 variant. This is correct. However, the fact remains that all of the birds that were tested for the presence of the H5-specific and N1-specific peptide sequences demonstrated immune reactivity against multiple epitopes, and they did not succumb to the influenza strain that infected the flock in December 2024. If these ostriches were now tested, I fully expect that their antibody levels that can target the HPAI H5N1 would be boosted and much stronger signals would be evident on the immunoblot tests from Kinexus.

61. In para. 71, Dr. French noted that animals are commonly used for production of antibodies for research purposes, but there could be batch to batch issues related to reproducibility of results. Polyclonal antibodies for commercial purposes are routinely produced in larger animal species such as horses, goats, and donkeys. The yield from an ostrich egg is sufficiently high and following affinity purification, would have the desired purity and specificity for industrial applications such as diagnostic kits and therapeutic antibodies. The UOF has already been involved in partnerships to develop antibody-based products for a wide range of applications such as treatment of acne, digestive enzyme neutralization in the gut for weight loss and reduction of blood triglycerides and sugar in diabetics, reduction of bacterial contamination of food, cosmetics and tooth paste, and improvement of masks and filters for protection against SARS-CoV-2 and other viruses and allergens.

62. In para. 72, Dr. French was unsure if selective breeding of ostriches that fully recover from infection with HPAI can permit development of more AIV-resistant birds. The fact that these birds can bred within a couple of years and produce so many eggs in a season for over 55 years makes them highly suitable for genetic improvement in infectious disease resistance. The survival from infection would be an important criterion in a careful breeding program. Another criterion would be how marked is the antibody response in the birds when immunized with portions of the HPAI proteins such as hemagglutinin and neuramidase.

63. In para. 75, Dr. French concurred that it is not unreasonable that the UOF ostriches are no longer shedding active avian influenza virus, but to be certain, each ostrich should be individually tested. Rather than the costly testing of individual birds by PCR, combined specimens from multiple birds can be tested on an ongoing basis. Rapid antigen tests could also be performed. Such antigen tests could use antibodies that are generated and available in the yolks of recently produced ostrich eggs following affinity purification with peptides with amino acid sequences from H5N1 virus proteins that were found to be high immunogenic (i.*e*., antibody reactive) in the Kinexus-based tests.

64. In para. 76, Dr. French referred to PCR-based studies that indicate that viral shedding of H5N8, and other low and high pathogenic H5 influenza strains may be beyond 14 days even though the birds may present no clinical signs. The problem with all of these studies is that the PCR tests used do not necessarily detect replication competent virus particles, but rather degraded fragment of the virus that may arise from their destruction by immune cells. At 40 thermal cycles of amplification of the viral RNA in the test samples, the false-positive rate likely exceeds 95%. Note that with each thermal cycle the amount of genetic material is doubled with the polymerase chain reaction. Thus, with 40 cycles, the original starting amount of RNA is increased by 2^{40} (or 1.1×10^{12}). With each thermal cycle, the chances of false amplifications and mutations in the generated nucleotides induced by the polymerase increase.

65. In any event, by 14 days after infection, the titers of the detected viral RNA fragments were extremely low and mostly undetectable within a week following initial infection. Since the UOF ostriches have continued to show no signs of sickness or infection since January 15th, over 7 weeks ago, the chances that any ostriches in the herd are shedding active virus in levels that support transmission is exceedingly remote.

66. In para. 77, Dr. French questioned the relevance of studies of human transmission of respiratory viruses like influenza to how the viruses are transmitted in birds, which are deemed to be less hygienic. Nevertheless, the basic principles of the chain of infection apply to animals as well as humans.

67. In para. 78, Dr. French pointed out that it is the drinking water that is the main source of transmission of influenza for ostriches. While the study by Abolnik *et al.* (2021)[22] that she cited indicates that the H7N1 virus could be detected in drinking water from infected ostriches by PCR tests, it was not tested whether this was replication competent virus. Seven-week-old chicks were used in this study, with initial infection by direct inoculation. It was not evaluated whether the birds that acquired subsequent infection was via the shared water.

68. It remains controversial whether influenza virus can really be spread through drinking water.[27] Influenza virus is classified as a respiratory virus because that is the main means of its transmission.

69. In para. 79, Dr. French asserted that "*the traditional presentation of avian influenza in ostriches is usually mild with more severe clinical illness and mortalities generally limited to younger birds unless the birds are under*

[27] WHO (2022) Guidelines for drinking-water quality: fourth edition incorporating the first and second addenda. Geneva, Switzerland: WHO Press.
https://www.who.int/publications/i/item/9789240045064
https://iris.who.int/bitstream/handle/10665/352532/9789240045064-eng.pdf

significant external stress. For this reason, the fact that on UOF the mortalities were observed in younger birds is what would be expected as a result of infection and should not implicitly be interpreted as a sign of preexisting exposure to this viral subtype." As pointed out earlier, the UOF ostriches that were the most susceptible to getting sick and dying were not necessarily younger birds, but those that were not in the herd prior to 2021. The fact that many of the ostriches tested positive for anti-H5 and ant-N1 antibodies in the yolk of their eggs collected in the summer of 2024 further supports the contention that there was prior exposure to an H5N1-like influenza virus before this time.

70. In para. 81, Dr. French strongly expressed her disagreement that "*the possibility of mutations occurring in ostriches that would increase influenza infectivity in mammals is "extremely remote*" and stated that this was "*well documented in the literature*" without providing any references. I am unaware of any publications that show that mutations in avian influenza viruses that resulted in increased ability to infect and cause disease in mammals, which were generated and identified first identified in ostriches. Dr. French does cite the study by Shinya *et al.* (2009),[28] which documented that ostriches with H5N1 influenza variants PB2-E627K and D701N are more able to propagate the virus. There is certainly no compelling no reason why the risk of generation of these mutants would be higher in ostriches than any other bird. The influenza virus identified in the UOF ostriches did not have these mutations. The risk of successful transmission to a human would be much higher from an H5N1 species that has already successfully infected a mammalian host such as a dairy cow. For example, 41 of the 70 human cases of HPAI H5N1 virus identified in the US were in those working on dairy farms and 24 were in those that worked with poultry.[6]

71. In para. 83, Dr. French described how the PB2 E627K mutant of HPAI H5N1 increases its ability to propagate in mammals, and she mentioned that the RNA polymerase is more active at lower temperatures based on citation of a review by Olivier (2006).[2] While wetter and colder months are associated with increased influenza virus survival detection (hence the seasonal incidence of influenza in early winter), this is more likely due to decreased host resistance than a more active viral RNA polymerase at lower temperatures. Generally, enzymes are less active at low temperatures, but identification of temperature-sensitive mutants is a common strategy to explore the role of a protein. However, usually higher temperatures than physiological lead to a loss of function. The immune system of the host is commonly more active at higher body temperatures to fight viral and bacterial infections.

72. The body temperature of a bird is typically 40 to 42 °C, whereas in humans it is around 37°C. If an ostrich has a normal body temperature between 34 to 36 *C, it is not ideal for a virus that has been propagated primarily in ducks and geese. A virus that normally infects a host will have its enzyme activities optimized for the environment in the host. Infection with a virus or bacteria typically induced increased temperatures in humans and animals.

73. In para. 85, Dr. French noted that mutations in HPAI viruses that increase infectivity do not necessarily increase virulence and the presentation of more severe clinical disease. The basis for use of attenuated viruses for vaccines is that they are able to infect a host, but they do not replicate as fast and provide time for the immune systems to respond before it is necessary to turn on further host responses that produce sickness. Thus, if a mutation increases infectivity without causing illness, this is actually desirable provided that it does not increase virulence. This exposure may induce immunity against more pathogenic variants of the virus in the future.

[28] Shinya, K., Makino, A., Ozawa, M., Kim, J.H., Sakai-Tagawa, Y., *et al.* (2009) Ostrich involvement in the selection of H5N1 influenza virus possessing mammalian-type amino acids in the PB2 protein. Journal of Virology. 83(24):13015-8. https://europepmc.org/article/PMC/2786862

74. In para. 87, and based on a study by Abolink *et al.* (2021),[20] Dr. French suggested that "*there is documented molecular evidence that an endemic strain of avian influenza that circulates in chickens in South Africa most likely originated from the recombination of two strains (H9N2 and H6N8) that occurred in ostriches."* Since South Africa has the largest commercial population of ostriches, with numbers around 350,000 birds with 350 registered ostrich farms, this speculation is not unreasonable,[29] but the number of ostriches on the UOF site is a thousand-time lower, so the chances of such a recombination with H5N1 strains is also lower by this magnitude.

75. In para. 89, Dr. French described some of the events that would be necessary for an HPAI virus to cause a serious pandemic in humans. The transmission of H5N1 influenza from one human to another has yet to be documented. In the one instance of a recent human infection with H5N1 HPAI in Canada, this was a 13-year-old girl that was obese and had asthma.[30] She also had the PB2-E627K mutation. One of the other necessary changes would be mutations that would permit the hemagglutinin protein to better binds to the alpha-2,6 sialic acids found in mammals rather than the terminal alpha-2,3 sialic acid residues found in receptors in birds, reptiles and amphibians. She also mentioned that the virus has to be more transmissible as an airborne pathogen. There are many other factors that would also be necessary too. It would have to optimize for humans, better evade the immune system of humans, and also be less virulent to more widely increase transmission.

76. In para. 93, Dr. French suggested that co-infection of the same host and the same cells with two different influenza strains could transpire such that "*a virus that may cause relatively mild disease in one host could in theory cause significant disease in an abnormal host*." This seems highly unlikely as successful viruses tend to optimize to bind to a host cell, enter and successfully replicate, and exit the host cell. Cross-species transmission of viruses is generally a rare event, considering that hundreds of thousands of different strains of viruses are believed to infect mammals, and only around a hundred are truly pathogenic in humans. If the infections from both virus strains are mild, then it is most likely that the immune system should be able to easily handle both viruses before coinfection can occur in the same host cells. The theoretical concerns raised by Dr. French that have yet to be fully manifested with the H5N1 influenza virus in mammals, and so far this virus strain generally leads to mild disease in mammals with very rare instances of severe illness.

77. In para. 98, Dr. French repeated her earlier concerns that having commercial birds in an open environment with access to wild birds and other animals increases the prospects of viruses crossing over to other species. By this logic, the CFAI would prefer the permanent closure of livestock operations that permit their animals to roam more freely outdoors in the interests of maintaining biosecurity. This all seems fruitless if these viruses are already commonly found within the wild animal communities.

78. In para. 101, Dr. French argued that selective breeding for resistance to influenza infections is not an immediate practical response to an outbreak. While this is true, it should be part of a longer-term strategy to protect commercial livestock. Farmers have had thousands of years of selective breeding practices to create the diversity of characteristics found in domesticated animals today.

79. In para. 105, Dr. French suggested that allowing the development of natural immunity in a herd of animals by not intervening is not an acceptable strategy on its own to deal with an outbreak of a potentially highly pathogenic virus or bacteria. I agree. It should be combined with preventative measures such a vaccination and other means of boosting the immune systems of animals, testing, isolation and treatment of infected animals,

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[29] https://stickymangorice.com/2021/04/09/ostrich-farming/

[30] Jassem, A.N., Roberts, A., Tyson, J., Zlosnik, J.E.A., Russell, S.L., *et al.* (2024) Critical illness in an adolescent with influenza A (H5N1) virus infection. N Engl J Med. 392(9):927-929. doi: 10.1056/NEJMc2415890. https://pubmed.ncbi.nlm.nih.gov/39740022/

and should the animals fully recover, reintegration into the herd or flock. Culling of whole herds of animals on the basis of a few infected animals in the unit is not a viable, long term strategy as is now clearly evident. The concept of recognizing natural immunity, which was largely ignored during the COVID-19 pandemic, is in fact endorsed by a wide range of scientists and medical practitioners. In the case of the COVID-19, which is another respiratory disease with similar morbidity and mortality to influenza, it is the opinion of tens of thousands of doctors and scientists world-wide as documented in the Great Barrington Declaration,[31] the Public Health Agency of Sweden,[32] the conclusions of the US Congressional Select Subcommittee on the Coronavirus Pandemic,[33] the National Citizens Inquiry into Canada's response to COVID-19,[34] and the recent Alberta's COVID-19 Pandemic Response[35] report that natural immunity should be adopted as a viable approach to curtail pandemics with special attention to isolated and protect those that are of higher risk than the general population.

Respectfully submitted by,

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[31] https://gbdeclaration.org/

[32] https://en.wikipedia.org/wiki/Swedish_government_response_to_the_COVID-19_pandemic

[33] https://oversight.house.gov/release/final-report-covid-select-concludes-2-year-investigation-issues-500-page-final-report-on-lessons-learned-and-the-path-forward/

[34] https://nationalcitizensinquiry.ca/national-citizens-inquiry-issues-commissioners-final-report/

[35] https://open.alberta.ca/publications/albertas-covid-19-pandemic-response

Appendix A

Page 213 of the filed affidavit for Court File No. T-432-25 in Federal Court between Universal Ostrich Farms Inc. (Applicant) and Canadian Food Inspection Agency (Respondent) entitled:

"Rule 318 Certified Tribunal Record of the Canadian Food Inspection Agency" filed by Cortnie Fotheringham, Incident Commander, Western HPAI Response. Dated February 22, 2025. This corresponds to internal communications listed under Tab 6D as a January 4, 2024, e-mail from CFIA AI Lab Liaison to CFIA AI Commander re Ostrich sample results.

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From:	West AI Lab Liaison / Ouest IA Liaison de laboratoire (CFIA/ACIA)
To:	West AI Commander / Ouest IA Commandant (CFIA/ACIA)
Subject:	Ostrich sample results
Sent:	1/4/2025 11:16:06 AM

Ostrich sample results from NCFAD, just in case you need this info over the weekend
Thanks
Nicki
WIN-AH-2025-FAV-0003
System ID#: 2025DCS-0000000033-4
Reason for submission: lab referral for confirmatory testing for HPAI, ref# 24-10316 (but being sent directly from the premise)
Owner: Universal Ostrich
Location: Edgewood
Submitter: Nicole Conner
Dist office: (2840) Vernon
Species: ostrich
Samples: 2 swabs
Test: AIV-PCRMX
History: Ostrich farm. Tested positive at BCMAL, duplicate samples being sent from the Vernon District office Michelle Li will be sending out the other set of samples from AHL on Monday with other positives as she is worried about the issues we have been having with shipping.
NCFAD Results 2025-01-03:
AVIN-PCRMX: Nucleic acid was extracted from the 2 swab samples and tested for the presence of influenza A genomic material using the matrix, H5 and H7 gene-based real-time RT-PCR assays. 2/2 swab samples tested **positive** on the matrix and on the **H5 gene**-based real-time RT-PCR assays, 2/2 samples were **negative on the H7** real-time RT-PCR assay. 2/2 swab samples tested **positive on the clade-specific** H5 2.3.4.4 real-time RT-PCR assay.
NCFAD Results 2025-01-04
Molecular pathotyping: The entire genome of the virus was amplified from two original swab samples and submitted to our Genomics group for NANOPORE sequencing. Eight gene segments of the virus were sequenced. The HA of the virus from the samples belong to **Eurasian Gs/GD lineage HPAI H5N1 (2.3.4.4B)** with cleavage site motif of "**PLREKRRKR/GLF**" compatible with HPAI viruses that came to Canada via the **Pacific flyway**. The H5N1 virus is a reassortant virus with **PB2, PA, and NP** originated from North American lineage low pathogenic avian influenza virus, and **PB1, HA, NA, M and NS gene segments from Eurasian viruses.** The virus is similar to the D1.1 viruses circulating in North America, but has the neuraminidase segment identical to WIN-AH-2022-OTH-0033 virus. PB2 – 627E (avian).