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Climate change impacts on bird migration and highly pathogenic avian influenza

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The unprecedented extent of highly pathogenic avian influenza coincides with intensifying global climate changes that alter host ecology and physiology, and could impact virus evolution and dynamics.

Climate change patterns appear to parallel an unprecedented global spread of highly pathogenic avian influenza (HPAI). Recent outbreaks have caused mass mortalities in traditionally unaffected bird species, including colonially nesting waterbirds and seabirds, many of which are species of conservation concern[1]. The ongoing HPAI outbreaks pose increased threat to agriculture and human health, given evidence of mutation towards mammalian adaptation[2] and sustained presence across seasons in poultry systems, neither of which were commonly documented in previous outbreaks. Introduction of HPAI H5 clade 2.3.4.4b viruses into the Americas in 2021–2022 marked this unprecedented expansion in the geography and impact of HPAI. This pattern is associated with strong adaptation to wild ducks, including increased replication with lower-dose exposures, shorter time to infection, greater shedding from respiratory and intestinal tracts, and potential increased persistence (ability to remain viable while outside the host) of virus particles in water[3].

Anthropogenic climate change could amplify current and future HPAI dynamics in wild birds at multiple spatial and temporal scales (for example, geographic distribution and seasonality). Climate change is characterized by a gradual increase in annual mean temperature and change in precipitation, accompanied by sea-level rise and water chemistry alterations. These changes can affect wild bird distributions and HPAI evolution over multi-year timescales. Meanwhile, climate variability introduces extreme weather events (EWEs) such as heatwaves, droughts, floods, storms and wildfires[4] with increasing frequency, severity and duration, which may induce immediate behavioural and physiological responses of individual birds leading to shifts in host and pathogen distribution, survival and evolution, thereby escalating disease risks[5].

Climate events, and bird and virus ecology

Long-term climate change and short-term EWEs can affect host ecology and virus evolution in parallel and in possibly conflicting ways. The biology of wild bird hosts is important at multiple interconnected scales: large-scale processes that affect bird distributions can explain virus dispersal, transmission and reassortment, while within-host processes, such as body condition, immunity and mortality, can act as selective forces on viral replication and evolution, which can also feedback to affect both local and broad-scale processes such as population densities and changes in distributions (Fig. 1).

Host migration, species' ranges, and interactions. Waterfowl (order Anseriformes) and shorebirds/gulls (order Charadriiformes) are the most competent hosts for HPAI. These taxa are found globally and are predominantly migratory. Millions of potential hosts move across the landscape each year, connecting breeding and non-breeding sites through migrations that can drive virus dispersal. Such movements can increase local viral diversity as individuals from different origins interact in mixed flocks, increasing the probability of virus reassortment or antigenic shift (exchange of gene segments when two or more virus strains co-infect the same host) that can lead to the emergence of novel strains with higher pathogenicity or to the ability to jump species barriers.

Long-term climate change impacts migration phenology (timing of migration) and species' ranges[6], with multiple potential outcomes for virus transmission such as increased or decreased population density and species diversity within a stopover location, leading to altered risk of virus dispersal during the annual cycle (Fig. 1). For example, wintering ranges for some species are shifting towards the poles and breeding (spring) migrations are occurring earlier due to warming temperatures. This behaviour can promote virus reassortment if novel contacts occur between previously distinct populations[7]. Similarly, following EWEs, birds may abandon territories, form newly mixed flocks or move into human-altered habitats, causing changes in contact rates within and between species[8]. Reductions in snow cover on agricultural fields and hence greater food availability can lead populations to forgo migration and increase the probability of transmission across the wildlife–agriculture interface. HPAI viruses evolve and transmit differently in agricultural populations compared with wild populations (for example, the high density of immunologically naive poultry allows for more rapid viral genetic mutation compared with less concentrated wild hosts with varying levels of immunity against low pathogenic avian influenza, LPAI), so this contact has implications for reassortment between LPAI and HPAI viruses. Finally, some studies suggest that influenza infection itself can affect host migration propensity and phenology in some taxa, in which case these climate-related effects on virus dynamics could further feedback to affect host migration (Fig. 1, middle panel).

Three HPAI expansion events demarcating spread from a source region to a new, previously uninfected continent have coincided with EWEs and an increased amount of virus in the source region. In 2005, the first spread of H5N1 out of Asia to new regions (for example, Europe) followed an increase in outbreaks in Asia (source region) and earlier frosts and extreme low temperatures in Europe (virus introduction region) that presumably altered wild bird host movements[9]. In December 2014, incursion of H5N8 into North America from Asia followed an explosion of outbreaks preceding Super Typhoon Nuri, the world's most powerful storm in 2014 that moved up the coast of Asia and across the Bering Strait to the North American Pacific Flyway. The current, unprecedented global expansion of H5N1 into the Americas following the 2021–2022 winter transatlantic introduction of H5N1 from Europe to North America[10] followed an increase in cross-seasonal

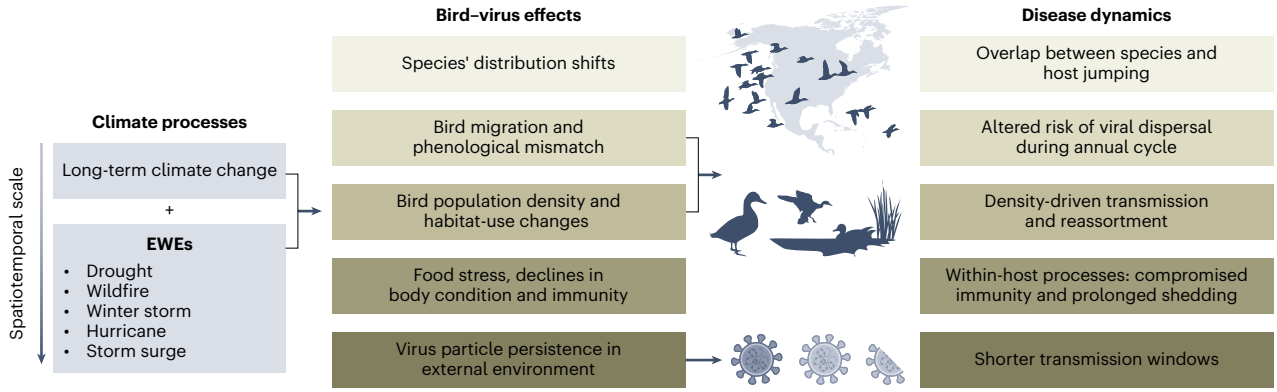

Fig. 1 | Climate processes affect HPAI disease dynamics via changes in host and virus ecology at multiple spatial and temporal scales. Climate processes occur at multiple scales, including broad extent long-term climate change (temperature and precipitation) that can alter vegetation phenology and communities, and short-term EWEs, such as wildfire or storm surge, that occur at local scales. Climate processes affect bird and virus ecology at multiple spatial and temporal scales, from generational changes in species' distributions, migration phenologies and population densities to day-by-day changes in virus persistence in the environment. These effects can have cross-scale feedbacks (for example, food availability and quality that trigger declines in body condition and immunity at the individual bird level and affect processes such as population and species' distributions). Disease dynamics depend on these ecological processes in wild birds, domestic birds and the environment, again with cross-scale interactions via virus dispersal, transmission and reassortment. Goose icon reproduced from ref. 16.

outbreaks in poultry and wild birds in Europe and in North Atlantic cyclone storms in this period. Phylogenetic analyses have shown high virus sequence identity between source and introduction regions, supporting the hypothesis of wild bird dispersal of HPAI, whether from natural migration patterns where species from cross-continent wintering grounds share breeding locations (for example, Iceland[10] or Bering Strait region), or from rapid EWE-induced vagrant bird movements. Leveraging new studies and datasets to understand how infection with HPAI affects bird movements[11] and how they will respond to EWEs is a first step for further work.

Host population dynamics. Host population dynamics determine infection prevalence, which in turn influences transmission dynamics and associated virus evolution as increased transmission rates provide more opportunities for mutations in a population (Fig. 1). For waterfowl, avian influenza infection prevalence peaks in autumn when immunologically naive juveniles enter the population, followed by declines as birds leave their breeding sites and recently infected juveniles develop immunity. Warmer temperatures that favour earlier migrations to the breeding grounds[6] can extend breeding duration and increase reproductive success of the avian hosts. More immunologically naive hosts at cooler latitudes that favour virus persistence in the environment could increase opportunities for transmission, dispersal and reassortment. Alternatively, if birds are unable to adjust their timing to match a shift in food resources, population decline can be expected, thus reducing virus potential due to low host density[7]. EWEs can also cause stressors, such as increased predation and competition, mortalities by direct impact (for example, death within a fire or storm), starvation or mass reproductive failures, that would similarly reduce host availability for the virus[12]. Species and populations inevitably vary in their population dynamic responses to the same climate and weather events, so more research into individual and population-level responses to climate change and EWEs will help to forecast the impacts of these changes on virus dynamics.

Host physiology and immune function. Seasonal variations in food availability, weather conditions, predation, competition and behaviour (for example, migration, molt and reproduction) not only drive migration, but also affect an individual's energetic state[13]. This impacts their ability to control infection and their suitability for viral replication and shedding (Fig. 1, right panel). If birds experience phenological mismatch during migration or increased stress following exposure to EWEs, the host's condition is likely to decline. Poor body condition can prolong viral replication (that is, decreased immune function), increase viral loads, and possibly increase disease severity and host mortality. Conversely, shorter and less energetically demanding migrations associated with poleward winter range shifts could increase the host's capacity to fight infection. The effects of protracted climate change and immediate EWEs on avian hosts at the individual level are complex and require combined study at the microbiology scale to understand physiological, immunity and transmission effects.

Virus evolution and environmental persistence. Virus transmission depends on circulation inside and outside the host, both of which are likely to be affected by climate events (Fig. 1). Avian influenza spreads when hosts are exposed to virions in water, land or air – a process that is sensitive to temperature and the physicochemical properties of the external environment[14]. Influenza particles degrade more quickly in warm, salinized or dry conditions, so climate change may reduce viral persistence outside the host as drought frequency and duration increases. Similarly, aberrations in temperature linked to winter storms or summer heatwaves may induce more frequent freeze–thaw cycles or extreme temperatures that limit virus stability. Alternatively, increased rainfall and flooding could temporarily increase the surface area of standing water that could increase the area for environmental transmission to occur and/or dilute virus concentrations in water. As viral strains continue to evolve, paired field and lab microbiology studies will help to understand and forecast potential for further virus evolution and persistence in the environment.

Future directions and need for integrated study

Advancing research on the current and future global state of HPAI in an era of rapid climate change requires an interdisciplinary and cross-national approach. Host ecology, virus dynamics, and anthropogenic and climate factors all require detailed research across multiple scales, which presents challenges for integrated study where they intersect. Here, we highlight three important scientific questions and approaches to help tackle this challenge.

First, how will altered contacts between influenza host species and populations influence viral reassortment, genetic shift and evolution? Answering this question will require the combination of data on virus diversity (by sampling wild birds) with ecological models of wild birds' movements and connectivity based on individuals' movement data, environmental data and climate projections. State-of-the-art animal tracking devices that pair smaller GPS units with auxiliary sensors can simultaneously detect bird mortality and behaviour, as well as local environmental conditions. The increasing availability of this valuable high-resolution data will allow us to understand how individuals respond to environmental stimuli. When combined with remotely sensed climate and weather data that are gathered on a regular basis, valuable insights can be attained. For example, high-resolution GPS telemetry of geese has been coupled with three-dimensional models of smoke plumes to determine how individuals reacted to megafires encountered during spring migration[15]. This type of information providing basic ecologic response is a first step to understanding how virus dynamics might be affected and could be further combined with information on host infection prevalence and virus diversity to predict transmission and dispersal.

Second, what are wild birds' physiological responses to climate change and EWEs, and how do they affect within-host disease dynamics? Using auxiliary sensors on marked birds in combination with field observations could help explain how hosts respond to environmental conditions. In addition, laboratory studies could characterize host responses to infection using transcriptomics (gene expression) or proteomics (protein expression). These methods can help reveal the immune and physiological stress responses of novel and reservoir host species. The associated outcomes for viral replication and within-host viral diversity could be measured using metagenomic tools that are increasingly available. These within-host microbiology studies and field ecology observations could be combined in models to explore potential changes in disease dynamics.

Third, how will selective forces of climate change affect influenza dynamics in the environment? Field studies that collectively span a gradient of environmental characteristics (for example, temperature, pH and salinity) will be important to sample virus circulation and persistence in nature, coupled with experimental studies to simulate viral kinetics under climate change scenarios. Ecological modelling informed by field surveillance and experimental data can be applied to forecast virus transmission within wild birds, as well as at wild bird–poultry and poultry–human interfaces that experience different environmental conditions. Understanding the thermal tolerance of the virus relative to key amplifying hosts and their overlap (or mismatch) will be useful for forecasting future distributions of HPAI.

To advance multiscale, interdisciplinary questions such as those outlined above, collaborations across disciplines are needed, such as among virologists, microbiologists, ecologists, ornithologists, climate specialists, remote sensing experts, social scientists, spatial modellers and computer scientists. In parallel, as technology advances and more datasets become available, application of targeted data streams, such as remotely sensed Earth systems, movement data from animals, genetic sequencing and -omics tools, requires understanding across the fields of expertise, as well as standardizing data collection, deposition and integrated analysis. This science should be applied across national borders, in conjunction with decision-makers and from a One Health perspective, as human, animal and environmental health are interconnected.

Responding to the urgent challenges of global climate change and avian influenza pandemic potential requires swift action at the intersection of science and policy, as it is crucial for wildlife conservation, economics and public health.

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Competing interests

The authors declare no competing interests.