

Changing climate and the Covid-19 pandemics: more than just heads or tails

Climate change can both facilitate zoonotic spillovers and have an effect on transmission chains. These effects alongside human behavior and awareness should be integrated in pandemic forecasting models.

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After emerging in China and rapidly ravaging Europe and the USA in a catastrophic first wave, it seemed a matter of time COVID-19 would harmfully strike low-and-middle income countries with limited resources. Despite the deadly toll left in places like India or Brazil, where nonetheless the disease did not spark as rapidly as in Europe – i.e. the effective reproduction number (R_t) remained low, tropical countries kept relatively untouched compared to those in temperate regions¹.

This latitudinal heterogeneous spread was initially attributed to the fact that tropical countries have in general younger populations, lower densities, acquired immunity and prior preparedness to former pandemics². At this stage of the pandemic, additionally, climate has not yet stood up as a recognized driver of SARS-CoV-2 and the mechanistic understanding of the virus' climatic sensitivities remains limited³. But what are the stages in the zoonotic spillover process where climate forcing is to be expected? And more generally: which is the effect of climate in disease emergence and spread?

Climate Change and the emergence of new pathogens

Zoonotic spillover is the multilevel process by which pathogens manage to overcome a series of natural barriers⁴ and infect other animal species (e.g. SARS-CoV-2, Ebola virus, VIH or Avian Influenza⁵). Zoonotic spillover theory, bearing into account the complexities of eco-epidemiological processes, specifies the types of barriers that have to be overcome in each situation⁴. Understanding which of these barriers can be affected by climate - either suppressed or enhanced, therefore facilitating or buffering spillover-, will be crucial to anticipate how climate change will affect these cross-species pathogen transmissions.

On the direct side, climate can facilitate pathogen's survival, development and dissemination, thus easing spillover. However, it is unlikely that in this regard climate will play a critical role in massively promoting the appearance of new spillover events. On the indirect side, instead, climate effects are much wider and far more complex. Climate change superimposed to a dramatic anthropogenic alteration of ecosystems is leading to a gradual substitution of species, shrinking of ecosystems and

decreases in species diversity⁶. These trend-like changes can clearly lead to spillovers in different ways and to closer and more general encounters between wildlife and humans (Fig. 1, A, B). But also climate extremes, acting on a much shorter timescale, can directly or indirectly affect the frequency and intensity of forest fires, droughts, floods, famines and migrations, equally acting as point stressors and exerting intense zoonotic pressure⁷. An example might be yellow fever virus (YFV) epidemics in non-human primates in the Brazilian Atlantic Forest initiating in 2016. There, the howler monkeys (*spp Alouatta*) are highly susceptible to YFV infection and epizootics driven by climatic anomalies often precede human cases, in epidemics that mirror zoonotic waves (Fig. 1C^{8,9}). Despite this facilitating role, climate should primarily be seen as a necessary but not self-sufficient factor contributing to disease emergence.

Impact of climate in transmission chains

The mechanistic pathway by which climate can directly affect spillover can dramatically contribute to inter-human transmission. For instance, it seems well-established that in the emergence of the 6th cholera pandemics at the turn of the 20th century, the takeover by the *Classical* strain was facilitated by a concurrent large climate anomaly in 1905 (e.g. extreme flooding followed by very cold months¹⁰). Subsequent strain replacements during the 20th century happened simultaneously with other large climate anomalies. However, while the former is true, the converse did not always apply. In fact, in many years Bangladesh experienced more intense climatic anomalies not accompanied by cholera strain replacements. Therefore, while climate stability seems to be appropriate for pathogen evolutionary differentiation¹¹ enhancing continuous transmission, climate extremes may also promote strain selection by exposing new niches for colonization, thus favoring takeover or spillover if outperforming strains are around in their environmental (or reservoir) hosts¹².

However, when inspecting climate change globally, an interesting and heterogeneous picture emerges, in which the tropics showcase the intensification of the hydrological cycle associated with the global temperature rise (see trends in absolute humidity, AH, Fig. 1A). As the atmosphere absorbs more heat due to the increase of greenhouse gases concentrations, its precipitable water content similarly increases (by approx. 7% per additional degree of warming). Interestingly, Central Africa and Southeast Asia concentrate the largest positive trends in AH, of 3g/m³ and over, while no major change has instead occurred as a whole in the Amazon basin, central Asia nor China. Thirty-year climatological differences in both rainfall (R, Fig. 1B left) and mean temperatures (T_{mean} , Fig. 1B right) between 1959-1989 and 1989-2019 are displayed for Wuhan (Fig. 1B top), the Congo (Fig. 1B middle) and the Amazon basins (Fig. 1B bottom), respectively. Clear rises in T_{mean} dominate in both the Amazon and the Congo areas with, also –albeit smaller- significant changes in precipitation occurring in these same regions. Those larger differences inform of the varying impacts of global warming worldwide, when assessing the relationship between climate change and disease emergence.

The tropical areas contain the largest diversity in mammal species (Fig. 1D color scale¹³), also considering the main taxonomic groups bearing zoonosis (e.g. a maximum in chiropteran species diversity in S. America¹⁴). A linear relation between the number of zoonotic spillovers and the diversity and abundance of reservoirs has been described (e.g. in tropical regions, Johnson et al., 2020). An enhanced number of spillover events should therefore be expected randomly distributed all year-round also in the Tropics, as food markets similarly exist in all these regions. Latitudinal distribution of spillovers, though, shows a clear maximum, instead, between 30°N-50°N, with both direct and airborne transmission dominating in those same latitudes in terms of disease burden, but not in the Tropics (Fig. 2A). Despite the large uncertainties and limitations existing in zoonosis databases, these results seem stable, also when weighted against population densities.

As seen, the distribution of zoonotic outbreaks occurring since 1940 indicates that larger outbreaks in terms of disease incidence (for major vector borne diseases), took place in both tropical Africa and SE Asia (Fig. 1D, crosses). Instead differences are evident *wrt* S. America, which appears largely under-

represented. Regarding disease emergence, data on zoonotic spillover events spanning the period 1940-2013 shows again a maximum in the 1980s and 1990s, centered in the temperate latitudes of the NH, followed by a slow but persistent decrease afterwards (Fig. 2A) not mimicked by global warming¹⁵.

Respiratory zoonoses

Special attention should be paid to the region around South China, where seasonal flu epidemics have every year a surge in respiratory infections (e.g. 87 percent of the most successful, globally-spreading strains of H3N2 from 2000 to 2010 originated there¹⁶), and where also SARS and COVID-19 major outbreaks initially emerged. It might therefore seem counterintuitive that successful zoonotic spillovers characterized by respiratory routes of transmission are mostly concentrated in the northern hemisphere (NH) winter, and in particular in and around China and nearby countries. However, it is also interesting to highlight that of all the regional biodiversity hotspots where trade of wildlife species is most established, South China is the only region located in the NH and therefore (Fig. 1D), subject to the strong seesaw effects of climate seasonality. Noticeably, SARS and also several outbreaks of imported MERS, Asian flu 1957-H2N2, Hong Kong flu 1968-H3N2 and the A flu 2009-H1N1 were all linked to cold seasons and all also seeded in the NH, not in tropical countries¹⁷. The relevance of cold-dry weather for the progression of respiratory infections therefore seems crucial and independent of the geographical source of the spillover, an aspect much overlooked. For instance, flu epidemics tend to start in “cold-dry” and “humid-rainy” conditions in temperate and tropical regions, respectively¹⁸. But still, scientific literature on the subject has not fully established which are the relevant predictors of influenza seasonality, nor why flu epidemics peak differently in different regions (e.g. with unimodal, bimodal and all-year round transmission¹⁹).

Mechanisms by which climate affects respiratory infections. Lessons from COVID-19 and Influenza

AH and/or T have been related to both outbreaks and the facilitated epidemic progression of influenza¹⁹. For temperature variations at high ends (>30°C), a blockage of aerosol transmission in the case of influenza virus has equally been described but controversy remains, because of the strong collinearity with humidity and unclear mechanistic associations^{20,21}. Indeed, the drying time of a droplet and the growth rate of the spread of COVID-19 have also been found to be at most, weakly related. Apparently, SARS-CoV-2 presents an enhanced airborne survival and transmission than influenza virus in temperate regions. Evidence also suggests that SARS-CoV-2, as well as other coronaviruses, can be dispersed and potentially transmitted by aerosols²⁶.

However, the limited COVID-19 spread in tropical countries might alternately indicate that the tropical high AH values render the aerosolized route poorly effective. Fomite transmission can, instead, in these settings be a crucial pathway to consider, as enhanced AH and T render this pathway more effective. Therefore, in the tropics, viral viability and persistence in fomites in surfaces may be favored, enhancing direct-contact and oral transmission if there is limited access to water and poor sanitation systems. Additionally, the large increases in AH (>3g/m³) seen in Fig. 1 for India and Africa may have an additional effect by decreasing the temporal stability of aerosols, therefore restricting also for virus-laden particles the window of time they can persist suspended in air.

A fundamental role of climate in modulating the different COVID-19 pandemic waves occurring so far has been largely disputed²² and remains as a challenging open question at this moment. But interestingly, a substantial difference in terms of epidemic progression surprisingly shows up if climate is added mechanistically in a process-based mathematical model for COVID-19. In the case of Japan, for instance, explicit inclusion of temperature through the model’s transmission (β) term yields, as a fundamental difference, the current peak in winter 2020/21 (see changes in A, R and D compartments

in Fig. 2). This model proved in the past successful in simulating and forecasting COVID-19 evolution in different countries, while accounting for a variety of public health interventions²³.

Fig. 2 displays results of the model fitted, for comparison, with and without climate. In one simulation, this β parameter is a constant with no influence of external factors, while in the other, a seasonal temperature-dependent component $\beta(t)$ is added (Temp, Fig. 2B,C). This term is inversely related to temperature and is normalized with the historical mean daily temperature from 1970. In the sensitivity analysis of Fig 2B, most of the parameters in the model with seasonality, are below a critical value except for the temperature-dependent infection rate β . This rate achieves a much higher value (e.g. compare β and Temp in Fig. 2B), thus denoting the high relevance of the temperature forcing.

To sum up, without the effect of colder temperatures, but keeping with the ongoing intervention measures and degree of lockdown, Japan would not likely have experienced a third wave (Fig. 2C). Of course, uncertainties are large and mostly derived from the limited data quality but trends in the patterns are undeniably clear. Such a fundamental qualitative difference underscores the role of climate, more so given its large public health implications.

If as for other beta coronaviruses (SARS, MERS) and wintertime respiratory viruses (e.g. rhinoviruses), a facilitating role of climate for SARS-CoV-2 transmission is demonstrated, tailored environmental surveillance and a large-scale transdisciplinary modelling initiative would be justified.

Contrasting the role of human behavior in response to epidemics also adds an important and complex domain to integrate within the explicit modelling of climate. Namely, during the 2003 severe acute respiratory syndrome (SARS) outbreak²⁴ and the 2009 A/H1N1 influenza pandemic²⁵, people's awareness in Hong Kong and Beijing appeared to have effectively contributed to limit the outbreak (e.g. by complying with precautionary actions such as use of face masks, hand-washing, and social distancing in public transportation as well as avoiding mass gatherings). At a time when the world is at stake with this global challenge, there is a critical need and, perhaps, a unique opportunity to assess our understanding of the environmental drivers of SARS-CoV-2 (and similar diseases) and their potential value in prediction. The traditional discipline divide among epidemiologists and climatologists needs to be effectively bridged at these hectic times.

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Figure captions

Figure 1. A) Average Absolute Humidity (AH) change 1989-2019 [g/m^3], retrieved from the Copernicus Data Store (CDS). B) Mean Monthly Temperature and Precipitation changes between the period 1959-1989 and 1989-2019 in a $3^\circ \times 3^\circ$ grid centered, respectively, around Wuhan (Lat 30° , Long 113°), Amazonia (Lat -4° , Long -67°) and Sub-Saharan Africa (Lat 1° , Long 19°). Data retrieved from ERA5 through the CDS. C) Zoonotic emergencies from the period 1940-2013, provided by the Emerging Infectious diseases Repository (<https://eidr.ecohealthalliance.org>), size-adjusted by the total number of human infections that resulted from them. In the background, a map of mammal species richness obtained from the SEDAC (<https://sedac.ciesin.columbia.edu/data/set/species-global-mammal-richness-2015>) D) Histogram of zoonotic emergencies by latitude and by their transmission pathway.

Figure 2. A) Histogram of zoonotic emergencies by latitude and by their transmission pathway. B-C) Model variability when the temperature is considered as a modulator of the infection rate. B) The Kolmogorov–Smirnov statistic (KS) measures the goodness of the model in terms of how close to a normal distribution the variability indices are. The dashed horizontal line is the critical value of the KS

statistic at the confidence level of 0.05. Infection rate is seasonal and parameters above the confidence level are considered as sensitive to the model's output. For the model with temperature, this measure shows how determinant the temperature component is in the model's output. C) Model dynamics for constant and seasonal infection rate. The shaded area between the curves shows the difference in the number of active cases (A, upper panel), recovered (R, middle panel), and deaths (D, lower panel) between the model with constant against the temperature-dependent infection rate. The epidemiological data used for the model fitting was obtained from the John Hopkins University and temperature from the European Climate Assessment & Dataset. To assess the influence of each parameter in the model performance, a sensitivity analysis was performed by using a moment-independent GSA method (PAWN method).

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