

Supplementary Materials for
The costs and benefits of primary prevention of zoonotic pandemics

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INTRODUCTION

The extensive discussions between the twenty authors of this paper led to materials too voluminous to be included in the main text. This supplement contains several sections that emerge from these discussions.

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CRITICAL THRESHOLDS FOR PANDEMICS

Figure S1 expands on the information presented in Figure 1. The vertical lines correspond to years when epidemics caused by emerging viral pathogens first appeared. The red dots quantify cumulative births since the last epidemic. The blue dots quantify cumulative human years of life since the previous pandemic, for example, the number of people alive in each successive year, summed over all the years since the previous epidemic).

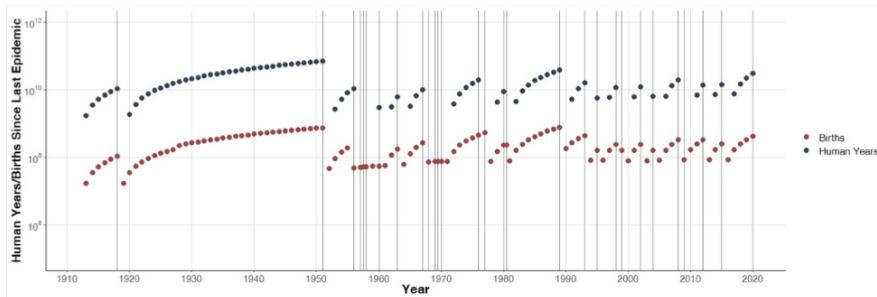


Fig. S1. Cumulative births and person-years since previous epidemics.

These data suggest the underlying presence of some form of criticality in the size of the human population required to trigger a new epidemic. Similar patterns have a long history in

epidemiology. Black and Barlett first posited them for measles (99, 100). They noticed host populations in cities or on oceanic islands had to exceed a critical community size of around half a million people to sustain measles continuously. More recently, similar patterns have been observed by Rhodes and Anderson for measles (101) and by Roy et al. for cholera and for forest fires (102). We explicitly acknowledge that we would not expect identical thresholds to determine the critical conditions for epidemic outbreaks in viruses with very different etiology. We also acknowledge that the pathogen outbreaks have started on different continents, so it may be more appropriate to use the number of births and cumulative human years for the continent where each outbreak initiated. That said, the increasing connectedness of the global human population through airline travel might justify the use of data for the whole human population.

ASSESSMENT OF THE VALUE OF LIVES LOST DUE TO EMERGING VIRAL ZOOSES

Baseline calculations

To estimate a probability distribution for viral zoonoses, we identified all novel viral zoonoses that have emerged since 1950 that resulted in >10 deaths. We include all outbreaks known to be severe (i.e., killed at least one million people) since 1900 to improve the tail estimation.

Only one disease, HIV, would meet this criterion otherwise. (This adds the 1918 Spanish influenza to our sample.) Table S1 in the paper lists the events used in our analysis.

Most of these outbreaks produced a spate of deaths in just a year or so. HIV is an exception, killing over 32.5 million people over the last 40 years. Spreading deaths over time is probably preferable to enduring all of them in a single pulse. Thus, we use the annual death count from HIV from UNAIDS (103, 104) and a discount rate of 5% to find the present discounted value (at the time of HIV's emergence) of the future stream of total deaths from HIV, resulting in about 10.7 million deaths.

We quantified the severity of an outbreak in standardized mortality units, or SMUs, where one SMU is the percent of the population who die multiplied by 10^4 . For example, if 0.05 percent (0.0005) of the population dies, then the SMU equals 5. With today's world population of 7,874,965,825, one SMU corresponds to about 779,480 deaths in 2021.

	P_a	μ_a	Expected annual SMUs	m	P_x	μ_x	Expected annual SMUs (extreme events)	f
Baseline from actual data	0.4	10.69	4.28	0.23	0.02	148.61	2.97	-6.68
Extreme outbreaks 10% more severe	0.4	11.72	4.68	0.21	0.02	163.47	3.27	-6.5
Extreme outbreaks 10% less severe	0.4	9.67	3.87	0.26	0.02	133.75	2.67	-7.65
Prevention cuts outbreak frequency by 1/3	0.27	10.69	2.89	0.35	0.013	148.61	1.93	-7.83
Prevention cuts outbreak frequency by 1/2	0.2	10.69	2.14	0.48	0.01	148.61	1.49	-11.2

Table S1: Parameters for baseline distribution of outbreak severity s

Notes: P_a is the annual probability of any outbreak; μ_a is the average severity of all outbreaks; P_x is the annual probability of an extreme outbreak, and μ_x is the average severity of extreme outbreaks. The parameters m and f for the distribution are calculated from those four as described in the text, with $m = 1/(P_a \mu_a)$. Results are shown for actual data and four hypothetical scenarios.

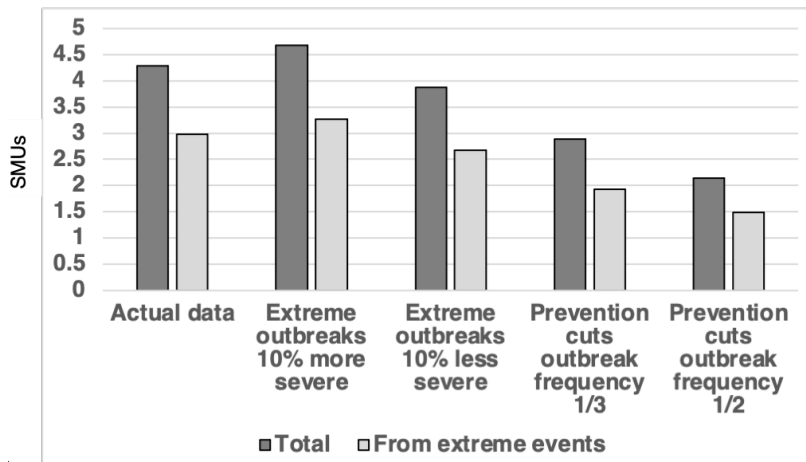


Fig. S2 graphs the various standard mortality units for the different scenarios presented in Table S1.

We follow Fan et al. (13) and use the frequency and severity of disease outbreaks observed in our sample to calibrate a hyperbolic distribution of outbreaks. The hyperbolic complementary cumulative distribution is given by:

$$r(s=Pr(S > s)) = [1 + m(1 - f)s]^{-[1+1/(1-f)]} \quad (1)$$

where s is the severity in SMUs of the outbreak; $1/m$ equals the mean of the distribution, and f indicates the fatness of the tail. (A smaller value of f implies that the tail of the distribution is fatter.) We quantify $r(s)$ for serious viral zoonotic diseases by deriving m and f based on the diseases in Table 2.

This process uses four parameters: the probability (frequency) that any outbreak happens in a given year, P_a ; the average value of severity s for all outbreaks, μ_a ; the probability that an extreme outbreak occurs in a given year, P_x ; and the average severity of extreme outbreaks, μ_x . Like Fan et al., we define an extreme pandemic to be one with an SMU greater than 10.

Our analysis includes 29 zoonoses (including the Spanish flu). For our baseline parameterization, we assume values for the parameters based on the frequencies and severities of outbreaks realized in Table S1. We set μ_a equal to 10.69 SMU, the average value of all SMU in the table. To set the annual probability of any outbreak, we use the frequency of outbreaks since 1950 (the first year at which reasonable mortality data are available for most

outbreaks). We observe 28 episodes in 70 years or about 40%; therefore, $P_a = 0.4$, which implies an average outbreak return time of about every 2.5 years (Table S1).

This calculation implies an expected annual outbreak severity of 4.28 SMU. At the 2021 world population, this is 3.3 million expected annual lives lost from outbreaks. We will set this equal to the mean of the hyperbolic distribution, $1/m$, in our calibration, implying that $m = 0.23$. To calibrate the tail, we observe that there have been two extreme pandemics ($s > 10$) this century, so we assume $P_x = 0.02$. We assume that the average SMU severity of such extreme events is the average of Spanish flu and HIV observed in the 20th century, or: 148.61 SMU. (Fan et al cite a modelling exercise for the insurance industry that concluded that the annual risk of an influenza

average SMU severity of such extreme events is the average of Spanish flu and HIV, with severity increased by 10% to 163.47 SMU. For comparison, we also consider the mirror-image case, in which the deaths from extreme outbreaks are reduced by 10%.

A 10% severity increase of extreme events results in an expected overall annual outbreak severity of $11.72 \times 0.4 = 4.68$. Thus, the mean SMU increases from 4.28 ($m = 0.23$) to 4.68 ($m = 0.21$). The expected annual damages from extreme pandemics are set to the new extreme pandemic average: $163.47 \times 0.02 = 3.27$ SMU. Using $m = 0.21$, we can similarly solve for $f = -6.5$. Similarly, an expected 10% decrease results in an expected overall annual outbreak severity of $9.67 \times 0.4 = 3.87$. Thus, the mean SMU decreases from 4.28 ($m = 0.23$) to

	P_a	μ_a	Expected annual SMUs	m	P_x	μ_x	Expected annual SMUs (extreme events)	f
Baseline from actual data	0.23	19.38	4.46	0.22	0.02	148.61	2.97	-5.36
Extreme outbreaks 10% more severe	0.23	21.24	4.89	0.20	0.02	163.47	3.27	-5.06
Extreme outbreaks 10% less severe	0.23	17.53	4.03	0.25	0.02	133.75	2.67	-6.32
Prevention cuts outbreak frequency by 1/2	0.11	19.38	2.13	0.47	0.01	148.61	1.49	-10.17
Prevention cuts outbreak frequency by 1/3	0.15	19.38	2.91	0.33	0.013	148.61	1.93	-6.08

Table S2: Parameters for distribution of outbreak severity s , small outbreaks dropped. Note: Calculations in this table are similar to those in Table S1 except the data include only zoonoses with greater than 1,000 deaths. Results are shown for actual data and four hypothetical scenarios.

outbreak on the scale of the 1918 pandemic lies between 0.5% and 1.0%. Our study considers potentially catastrophic outbreaks of a broader set of diseases.) The expected annual damages from extreme pandemics alone ($s > 10$) are then $148.61 \times 0.02 = 2.97$ SMU; more than half the annual expected deaths from pandemics comes from the risk of extreme events. Using this in combination with $m = 0.23$, we then solve for $f = -6.68$

We wished to consider the possibility that, as a result of globalization and increased population densities, extreme pandemics might become more severe. To do so, we consider a scenario in which the expected severity from extreme outbreaks increases by 10%. Thus, for this scenario we set the

3.87 ($m = 0.26$). The expected annual damages from extreme pandemics are then $133.75 \times 0.02 = 2.67$ SMU. Using $m = 0.26$, we can then solve for $f = -7.67$.

We also wished to model the effects from policies described in the main paper's sections on preventing deforestation and addressing wildlife trade on the frequency of outbreaks of all types. We considered the following hypothetical scenario. Suppose that prevention cuts the frequency of all outbreaks by 1/2 relative to the baseline. In other words, we have P_a falling from 0.4 to 0.2, and P_x falling from 0.02 to 0.01.

We calibrate the distribution implied by this prevention scenario. The table gives a mean SMU of 10.69. The prevention

	P_a	μ_a	Expected annual SMUs	m	P_x	μ_x	Expected annual SMUs (extreme events)	f
Baseline	0.4	1.32	0.53	1.89	0.01	24	0.24	-5

Table S3: Parameters for distribution of outbreak severity s , Spanish influenza dropped

This implies an expected annual outbreak severity of 0.53 , thus, $m = 1.89$. To calibrate the tail, we assume $P_x = 0.01$. We assume that the average SMU severity of such extreme events is that from HIV or: 24 SMU. The expected annual damages from extreme pandemics alone ($s > 10$) are then $s^*(10) = 24 \times 0.01 = 0.24$ SMU. Using this in combination with $m = 1.89$, we then solve for $f = -5$.

scenario leads to an expected yearly severity of $10.69 \times 0.2 = 2.14$. Prevention cuts expected annual outbreak severity by a half. This implies $m = 0.48$. The expected annual severity from severe pandemics ($s > 10$) is now $148.61 \times 0.01 = 1.49$ SMU. This value gives $f = -11.2$, reflecting a fatter-tailed distribution for total expected annual damages than under the baseline scenario. Table S2 also provides an equivalent estimate for prevention reducing the frequency of all outbreaks by $1/3^{\text{rd}}$.

To translate our findings in the paper's Table S2 into terms familiar to policy analysts, we use estimates of the value of a statistical life (VSL) to monetize mortality for benefit-cost analyses. VSL is an estimate of people's willingness to pay to avoid death and varies with income. Viscusi and Masterman (10) estimate that the average VSL for countries with different ranges of wealth varies from \$107,000 to \$6.4 million. We do not know the incidence of pandemic deaths among different countries of the world, so we calculate total willingness to pay to avoid lives lost with both of those VSL numbers to provide a range. Note that these VSL estimates are conservative; other analyses of the mortality costs of pandemics use a VSL equal to \$10 million per life lost. This is the value the U.S. EPA uses to analyze environmental regulation benefits.

Sensitivity to dropping small outbreaks

We redid the above analysis including only those zoonoses in the sample that resulted in at least 1,000 deaths (instead of the lower bound of 10 deaths in the main exercise). We retain the definition of an extreme event as one involving > 10 SMU. (As before, this set constitutes of the Spanish flu and HIV/AIDS). This leaves us with a smaller sample of 16 zoonoses (including the Spanish flu). This is a rather small sample and reported mainly for robustness. The results are below.

For our baseline parameterization, we set μ_a equal to 19.38 SMU, the average value of all SMUs in the restricted sample table. We observe 16 episodes involving over 1,000 deaths in 70 years or about 23%; therefore, $P_a = 0.23$, which implies an average return time of an outbreak about every 4.35 years.

Table S2: Parameters for distribution of outbreak severity s , small outbreaks dropped Note: Calculations in this table are similar to those in Table S1 except the data include only zoonoses with greater than 1,000 deaths. Results are shown for actual data and four hypothetical scenarios.

Sensitivity to excluding Spanish influenza

To demonstrate how results change if we ignore the serious pandemic associated with Spanish influenza, we replicate the calculations in Table S1 excluding that one extreme event. Our analysis now includes 28 zoonoses. For our baseline parameterization, we assume values for the parameters based on the frequencies and severities of outbreaks realized in Table S3. We set μ_a equal to 1.32 SMU, the average value of all SMUs in the table. We observe 28 episodes in the last 70 years, or about 40%; therefore, $P_a = 0.4$, which implies an average return time of an outbreak about every 2.5 years (Table S3).

Alternative distributions to model disease mortality

Here we briefly discuss two alternative distributions. If $r(s)$ is the exponential survival function, its CDF is given by:

$$1 - r(s) = Pr(S > s) = 1 - e^{-ks}, \text{ if } x \geq 0, \text{ and } 1 - r(s) = 0, \text{ if } x < 0.$$

Parameterizing k results in $k = 1/15.7 = .064$. The estimated distribution implies that an event of the order of the Spanish flu (273 SMU) has an annual probability of 0.00000003, resulting in an expected return time of 39 million years, which is unreasonable (Fan et al. cite a modelling exercise for the insurance industry that concluded that the annual risk of an influenza outbreak on the scale of the 1918 pandemic lies between 0.5% and 1.0%).

A Generalized Pareto distribution survival function is given by:

$$r(s) = Pr(S > s) = \left(\frac{x_m}{s}\right)^k, \text{ when } x \geq x_m, \text{ and } r(s) = 1, \text{ if } x \leq x_m.$$

Here, x_m is the scale parameter and $k > 0$ is the tail index. For our sample, using MATLAB, we can use maximum likelihood to estimate the following parametrization and corresponding 95% confidence intervals: $k = 4.0259$ (2.2320, 5.8199); $x_m = 0.0012$ (0.0004, 0.0037). The estimated distribution results in similarly unreasonable expected return times.

SPILLOVER IN SAVANNAS

Before the emergence of HIV, most human pathogens had their origins in domestic livestock. Savannas and grasslands were the habitats from which the earliest human pathogens arrived. The domestication of grass-eating ungulates, combined with

the dogs used to herd them, provided steppingstones for many past human pandemics. Examples include measles, mumps, and smallpox (136). Without vaccination, these pathogens would have as large an impact on human health as Covid-19 does today (137).

Research in and around savannas continues to provide insights into the emergence of zoonotic pathogens, the best practices for monitoring and managing disease reservoir species and working with local people to mitigate the risks they may face from zoonotic diseases. As in forests, veterinarians have had a leading role in obtaining these insights in savannas.

Sarah Cleaveland and colleagues have shown that savannas continue to be a source of pathogens for humans (138, 139).

Their projects in and around Serengeti National Park provide a template for collaboration between veterinarians and local people to discover and control already known as well as novel emerging pathogens: brucellosis (*Brucella* species), Q fever (*Coxiella burnetii*), leptospirosis (*Leptospira* species), rickettsioses (*Rickettsia* species), bartonellosis (*Bartonella* species), plague (*Yersinia pestis*), as well as vector-borne diseases such as Rift Valley fever and Chikungunya (140).

Virus	Start	Deaths	World population	Percent deaths per population	SMU	Mortality source
Spanish influenza	1918	50,000,000	1,830,000,000	2.732240437	273.2240437	(105)
<u>Hantaan virus</u>	1951	46,430	2,584,034,261	0.001796803	0.17968028	(106)
South American hantaviruses	1956	1990	2,822,443,282	7.05063E-05	0.007050629	(107, 108)
<u>Kyasanur forest disease</u>	1957	1000	2,873,306,090	3.48031E-05	0.003480311	(109)
H2N2 influenza	1957	1,100,000	2,873,306,090	0.038283426	3.828342563	(110)
<u>Junin virus</u>	1958	5900	2,925,686,705	0.000201662	0.020166206	(111)
Lacrosse virus	1960	300	3,034,949,748	9.88484E-06	0.000988484	(112)
<u>Machupo virus</u>	1963	290	3,211,001,009	9.03145E-06	0.000903145	(113)
Marburg virus	1967	370	3,478,769,962	1.06359E-05	0.001063594	(114)
H3N2 influenza	1968	1,000,000	3,551,599,127	0.028156331	2.815633083	(115)
Lassa fever	969	250,000	3,625,680,627	0.006895257	0.689525708	(116)(117)
Venezuelan equine encephalitis	1969	300	3,625,680,627	8.27431E-06	0.000827431	(118)
Monkeypox	1970	5000	3,700,437,046	0.000135119	0.013511917	(119)
Ebola	1976	12930	4,154,666,864	0.000311216	0.031121629	(120)
Rift valley fever	1977	3000	4,229,506,060	7.09303E-05	0.007093027	(121)
HIV	1980	10,700,000	4,458,003,514	0.240017756	24.00177561	(122)
<u>Puumala virus</u>	1980	10	4,458,003,514	2.24316E-07	2.24316E-05	(123)
<u>Guanrito virus</u>	1989	140	5,237,441,558	2.67306E-06	0.000267306	(124)
<u>Sin nombre</u>	1993	260	5,581,597,546	4.65816E-06	0.000465816	(125)
Andes	1995	130	5,744,212,979	2.26315E-06	0.000226315	(126)
<u>Nipah</u>	1998	200	5,984,793,942	3.3418E-06	0.00033418	(127)
West Nile	1999	2330	6,064,239,055	3.8422E-05	0.003842197	(128)
SARS	2002	770	6,301,773,188	1.22188E-05	0.001221878	(129)
Chikungunya	2004	35,000	6,461,159,389	0.000541698	0.054169845	(130)
H1N1 influenza	2008	284,000	6,789,088,686	0.004183183	0.418318294	(131)
Severe fever thrombocytopenia syndrome	2009	370	6,872,767,093	5.38357E-06	0.000538357	(132)
MERS	2012	860	7,125,828,059	1.20688E-05	0.001206877	(133)
Zika	2015	50	7,379,797,139	6.77525E-07	6.77525E-05	(134)
COVID-19	2020	479,310	7,794,798,739	0.0061491	0.614910039	(135)

Table S4. Sources and SMU calculations for mortality estimates used in Table S2

Their work began with rabies, a highly lethal virus that is a risk to anyone working with dogs who live in rural areas in close association with wildlife (141). They prioritized the health of domestic animals, which frequently guard houses and livestock. Similar dynamics between veterinarians and the communities they work with occur in the Arctic, where local communities view veterinarians as some of the few trustworthy people from outside the close-knit Arctic communities (142).

INTERNATIONAL TRADE

Fig. S3 illustrates trade data from the Convention on International Trade in Endangered Species (CITES). It provides a snapshot of wildlife trade through Singapore over the past 40 years. Singapore is a compelling choice to understand variations in the international animal trade. It is an economic hub of Southeast Asia, and none of the species traded has its origins in the country. Data from the early years reflect an increase in compliance with CITES, with imports to the United States rising quickly and then remaining stable for decades. In contrast, imports to China have steadily increased, suggesting that trade may follow a country's economic fortunes or global demand for wildlife. The CITES data from Singapore reveal that more than 10,000 transactions (some of which can include parts from thousands of animals) brought wildlife to the United States in recent years.

National importation databases provide another source for wildlife trade flows. The US Fish and Wildlife Service inspects all wildlife shipments. The data show that most animals in trade have lower zoonotic infectious risk — examples include corals, fish, reptiles, amphibians (143). Moreover, the trade volume is high but stable, with tens of millions of individual animals imported into the US each year. Neither CITES nor USFWS data provide much information on zoonotic surveillance and animal origin. Furthermore, there is a lack of clarity on, and verification of, whether animals are wild-caught, captive-bred, or 'ranched'.

Compounding data shortfalls related to the scope of trade is inadequate surveillance for zoonoses in traded animals. One might infer a species' potential as a pathogen reservoir from knowledge about its taxon's contribution to past emergence events (21, 48). However, current databases of pathogen diversity are inadequate to make predictions of viral host preferences with confidence. This failing makes such an approach prone to allowing novel pathogens to slip through surveillance (21, 48). Furthermore, data in wildlife trade databases are otherwise mostly silent on zoonotic disease risk. The US Fish and Wildlife Service officers inspect all legal shipments of wildlife imported to the US on arrival at designated ports to ensure compliance with CITES. The Service only tests for a few infectious diseases routinely. Examples are psittacosis in parrots, foot and mouth disease in ungulates, and highly pathogenic avian influenza (HPAI) in some poultry (144). Many countries have limited or no disease surveillance for imported wildlife, with surveillance often proportional to

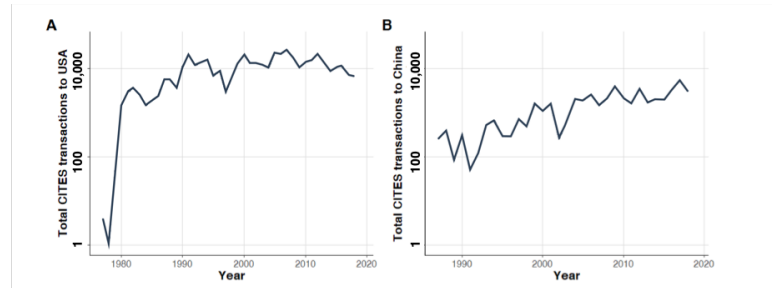


Fig. S3 Annual animal imports from Singapore to the United States (A) and China (B) as recorded by CITES (Convention on International Trade in Endangered Species). The data start with CITES' establishment in 1975. Data are the cumulative number of transactions (within each year) for species listed under Appendix I, II and III. The records are predominantly for mammals, birds, fish, and reptiles. They do not include fish harvested for food.

the country's affluence (145). We could find no reference to surveillance for unknown or novel pathogens in the wildlife trade for any country.

The legal framework that the World Organisation for Animal Health (OIE) uses in this regard could be effective if applied to the wildlife trade. Wildlife trade has generally not been part of its bailiwick, but if enacted, it could provide incentives for countries to test and report diseases so that they can trade freely. It also can enable an expansion of within-country monitoring of animals in trade via the creation of disease-free zones. In larger countries, this could fill a major surveillance gap, which may have contributed to the emergence of Covid-19 (146). Shipments could be certified as 'tested', with the onus on CITES to verify testing status. The groundwork for greater collaboration between CITES and OIE was laid down in a 2015 memorandum of understanding in place since 2015 (<https://cites.org/eng/node/18857>). It aspires to deepen their communication and cooperation "to protect CITES-listed species and conserve biodiversity by ensuring the efficient implementation of surveillance and disease control measures needed to protect animal and human health worldwide."

Another means to control zoonotic virus emergence risk from the wildlife trade could come from strengthening wildlife enforcement networks (WENs). Regional WENs developed 15 years ago to create cross-border linkages between national task forces made up of CITES, customs and police authorities (85). WENs consist of people involved in wildlife trade monitoring and wildlife law enforcement and are organized according to regional trade blocs (e.g., EU, CARICOM, SADC). Inadequate financial backing, anemic political support, lack of local leadership, and interference from foreign countries in trade, and other factors, have stymied WEN's mission. While imperfect, WENs offer an existing mechanism to coordinate enforcement around wildlife trade. At present, none monitors animal or human health.

Policies that restrict wildlife capture and trade in countries with high emerging disease risk may gain additional value when they mirror policies that reduce wildlife consumption in wealthier countries with lower emerging disease risk. For example, fur production destined for the international fashion trade drives the farming of raccoon dogs (*Nyctereutes procyonoides*) and other species in China. Raccoon dogs were among the mammal species infected by SARS-CoV in the wet markets of Guangdong before the human outbreak (147). They

are also susceptible to SARS-CoV-2 infection (148). The ability of people to infect mink (*Mustela lutreola*) with SARS-CoV-2 that can then transmit it back to people underscores the need to monitor captive-bred species for pathogens (149). Legislation requiring all fur used in garments to identify their species content and country of origin could reduce demand. So would social pressure to reduce the wearing of factory-farm-sourced fur for fashion, whether it be for the fur on a mass-produced ski jacket hood or a supermodel's shawl.

Corporate social responsibility campaigns can drive down demand for animal skins and fur, and along with it, the risk of disease emergence from wild-caught or captive-bred suppliers. Such an effort requires robust tracing of supply chains that could be enabled by a platform similar to TRASE (<https://trase.earth/>).

All such measures must be assessed for their efficacy. For example, restrictions on wildlife capture or other barriers to entry in the legal trade of wildlife can divert animals into illegal trade. More than a decade ago, this happened after a ban on hunting and consumption of primates in Equatorial Guinea (150). More animals moving to illicit trade will compromise the ability to conduct surveillance, rapidly identify outbreaks, and trace infection sources.

Restrictions on wildlife for food in China

China's ban applied to capture for food but not for research, medicines, pets, and fur production. In other nations — Peru, for example — there have been calls to improve sanitary conditions in markets, segregate species (especially domestic species), and improve policing of illegal wildlife trade (151).

Primary prevention of zoonotic viral disease entails more vigorous enforcement of national and international laws that determine the wildlife species that can be traded ethically, legally, and sustainably. In the months following the emergence of Covid-19, the Chinese government banned wildlife food consumption and prohibited hunting and breeding wild species explicitly to reduce spillover risk.

The list of wildlife under special state protection was officially revised for the first time on February 9, 2021 — some 30 years after its release. Wildlife-sourced medicine was also removed from the national basic medical insurance coverage in 2019. This change increases the out-of-pocket cost for medicines sourced from wildlife and disincentivizes the consumption of wildlife for medicinal uses.

Despite these prevention measures within China, international efforts are critical to reducing wildlife trade and disease emergence risk. In particular, they are needed to curb the trans-border supply and improve regional diseases surveillance in the countries neighboring China.

Pathogen surveillance in China

Since HPAI and SARS, China has invested in zoonotic disease surveillance. The Chinese National Influenza Center (CNIC) has developed a surveillance network covering 554 hospitals and 408 diagnostic laboratories in 31 provinces and autonomous regions. These facilities collaborate with the Animal Disease Control Center in China on surveillance and response to disease outbreaks in humans and livestock. In addition, the National Forestry and Grassland Administration (NFSA) established the Central Monitoring Station for Terrestrial Wildlife Epidemics and Epidemic Sources in 2005. More than 350 monitoring stations across the country collaborate with the conservation community for terrestrial wildlife disease surveillance in China. That includes avian influenza in wild

birds. In October 2020, following Covid 19, the NFSA has promoted a key science and technology program for national wildlife-borne pathogens surveillance and transmission risk assessment. Another viral surveillance program discovered more than 350 novel coronaviruses in Chinese bat populations and detected viral spillover into communities of southern China (146).

DEFORESTATION

The Brazilian Amazon

The diseases most likely to appear after deforestation in the Amazon are vector-borne diseases such as yellow fever, Mayaro, Oropouche, and malaria (72, 152). At least 187 different arboviruses and other viruses in vertebrates have been isolated in the Amazon; two-thirds of these are pathogenic to humans (152). Fortunately, they may be less likely to result in pandemics than viruses transmitted in aerosols. Temperatures constrain their range, and they must pass through two hosts in their lifecycle (153). Some, like Zika virus, induce strong immunity in humans, which can rapidly curtail their spread (154).

Not all viruses discovered in the Amazon are vector-borne. Neotropical Brazilian bats carry coronaviruses from the same genera (beta) as SARS-CoV-2 (155). There has not been extensive sampling of bats for coronaviruses in the Amazon, and so the extent of the viral pool is largely unknown. Similarly, there has been limited viral discovery in South American rodents, although they are reservoirs for hantaviruses (156) — as they are throughout the world.

Many reasons should compel preservation of the Amazonian forest: conserving biodiversity, protecting Indigenous Peoples and their lands, and preventing carbon emissions, among others. The constellation of high-risk reservoir species and the potentially large number of presently undiscovered zoonotic viruses they carry provide another motivation to curtail the destruction of the Amazon. Fortunately, recent history shows the Amazon can be protected when political and financial stars align.

Rates of deforestation in the Brazilian Amazon fell approximately 70% between 2005-2012 due to public policies combined with public and private actions (91). Conceivably, reduced deforestation might have reduced crop production or curtailed economic opportunity. In the event, the reverse was true: during the same interval that deforestation rates fell, soy yields and overall soy production met or exceeded prior years with higher deforestation rates (91). GDP in the Amazon increased by 141% ([Instituto Brasileiro de Geografia e Estatística](#)).

Several policies enabled better protection of the Amazon. These policies expanded protected areas, recognized Indigenous territories, put market restrictions on illegal landholdings, placed credit restrictions on municipalities with high deforestation rates, and created payment for ecosystem service programs benefiting small farmers (91, 92). State-of-the-science satellite monitoring and improved enforcement of existing laws buttressed these policies (91).

These actions to curtail deforestation cost the Brazilian government US\$1 billion per year (~0.1% of Brazil's total federal budget), primarily from federal funds, but also with contributions from state and cities (92). An Amazon Fund, including a US\$1 billion commitment from Norway between 2009-2019, supported actions to reduce deforestation (91).

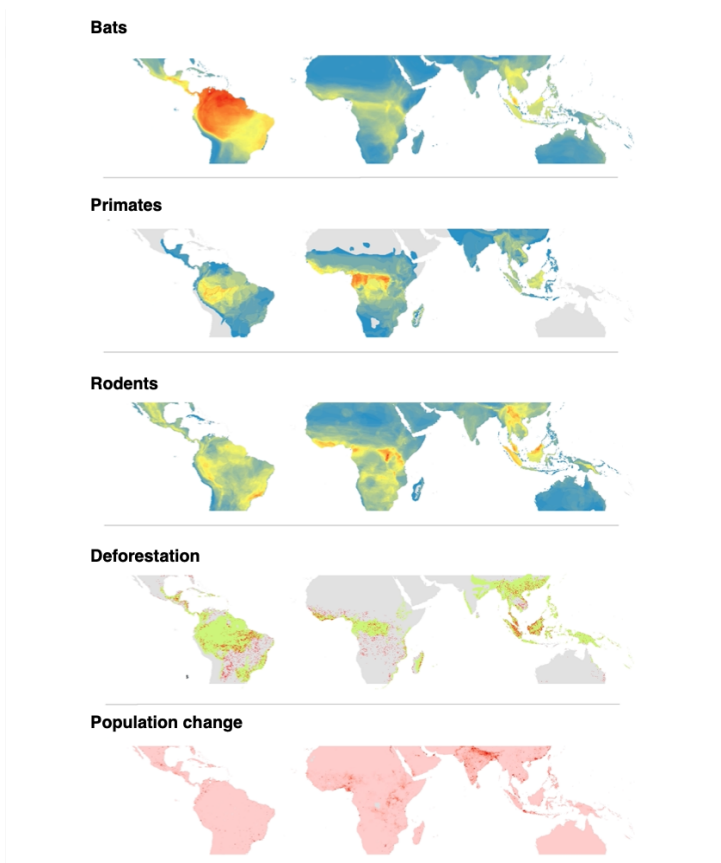


Fig. S4 Mammal species richness, deforestation (2000-2019), and human population growth per km² from (2000-2020), in tropical and subtropical regions. The latitudinal bands between 30°N and 30°S contain more than two-thirds of all known species. Spatial resolution — the pixel size, for species, forest, and population data are 10km x 10km, 30m x 30m and 1km x 1km, respectively. Species richness scales from blue (1) to the maximum numbers that are 120 species (bats), 21 species (primates), 62 species (rodents). Deforestation is in red, remaining forest in green. Population changes range from zero or less to an increase in 627 people per pixel.

As impressive as the success in protecting the Amazon achieved with resources and people living outside the forest may be, it does not match those who live within it. For millennia, Indigenous peoples have lived in the Amazon and used their resources sustainably. In the past century, Indigenous territories have been vital for forest protection in the Amazon. They have proven resilient to the vagaries of government policy and funding streams that can undermine other attempts to protect forests. Further designation of tropical forest areas as Indigenous lands may be among the most, if not the most, cost-effective means to ensure forest conservation (157).

Kibale National Park, Uganda

Kibale's small size may limit the risks of viral emergence. However, Ebola and Marburg viruses may be present in Kibale's bats threatening its primates and people who may contact them (158). Models of land conversion effects on disease transmission suggest that the risk of spillover may be greatest at intermediate levels of habitat loss in places like Kibale (58).

In the Kibale mosaic, people, livestock, and wild animals live in close proximity, and pathogens move readily among them

(159). Spillover surveillance is essential. Outbreaks fuel a vicious cycle. They impoverish people, and that impoverishment promotes greater wild meat consumption. That consumption, in turn, promotes pathogen emergence. As a result of Covid-19, the World Food Program estimates that an additional 130 million more people may face acute hunger owing to loss of livelihoods – a ~20% increase over baseline (160). Many of them live in emerging infection hotspots.

The challenge to reduce deforestation in places like Kibale is the continuity of effort and inclusion of local communities as rightful stakeholders and beneficiaries of both the financial profits and ecosystem services provided by protected areas. Kibale hosts a profitable ecotourism project based on chimpanzee trekking. Kibale raises funds from fees charged to tourists, scientific researchers, and film crews. In total, these fees, and contributions from conservation groups, amount to approximately \$2.6 million per year. Twenty percent of this goes to the local community governments (161). The Park had an annual budget of just under US\$2 million in the fiscal year 2019-2020. (Financial data from the authors are available upon request). The financial and overall success of Kibale is exceptional among the East African forest remnants. Many of the other remnant forests in the region, including the Mabira forest in Uganda and Kakamega forest in Kenya, face many threats but have far fewer

resources to protect them.

For example, the Kibale Health and Conservation Clinic and Kibale Mobile Health Clinic provide medical care to 16,000 people a year and, through additional outreach, they engage with an estimated 200,000 people(162). The Mobile Health Clinic provides isolated villages with medical care and guidance on prevention and focuses on sanitation, nutrition, intestinal parasites, family planning, and risks associated with bushmeat consumption, and provides an early warning system if a spillover event should occur. It also provides a forum for community members to air grievances about the park and develops mechanisms towards their resolution.

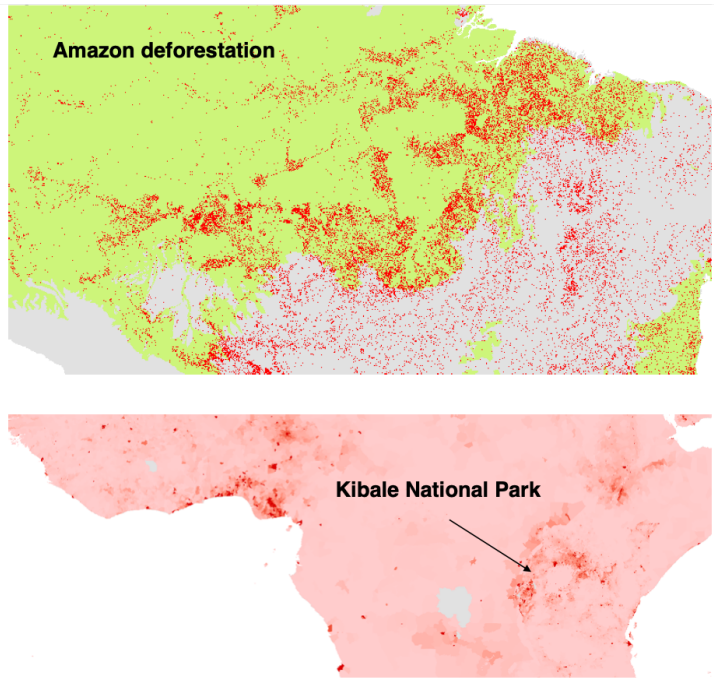


Fig. S5. Details of Amazon deforestation (top) and population growth in tropical Africa (bottom). Roads deep into the Amazon created extensive edge areas bringing people into contact with exceptionally diverse vertebrate communities. In West and Central Africa, rapid human population growth into previously forested areas spurred wild animal meat consumption and the starts of the various HIV spillovers.

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