

Mechanics of pandemics

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Summary

COVID-19 and previous pandemics have shown how diseases can disrupt, threaten, and transform daily life. Since pathogens and societies are continuously evolving, every pandemic is different. However, certain fundamental principles of disease transmission appear to hold true across different outbreaks. These "mechanisms" are grounded in natural laws or the very structure of our biology and societies. This paper compiles ten fundamental mechanisms, curated by a multidisciplinary team with backgrounds spanning public health, medicine, epidemiology, political science, mathematics, physics, and psychology. These mechanisms, although perhaps underappreciated, substantially shape how pandemics unfold and are controlled. The better we succeed in understanding these mechanisms and establishing this knowledge in our societies, the better we will be able to prepare for future pandemics and respond appropriately when they occur.

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Introduction

In our ever-changing world, pandemics will continue to pose a substantial risk to the well-being of our societies. While we cannot predict when or how the next crisis will emerge, its arrival appears inevitable because pathogens and societies coevolve: emerging infectious diseases drive societies to adapt through new technology, medicine, and preventive measures, thereby applying developmental pressure and prompting

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pathogen selection and ongoing evolution.^{1–5} As a result, no two pandemics are identical. Each pandemic possesses unique characteristics defined by, e.g., its transmission routes, clinical manifestations, and societal impacts.^{2,3,6} Yet, whether hidden deep down or visible in plain sight, fundamental similarities exist in how outbreaks unfold and can be controlled. These similarities arise from recurring epidemiological, behavioral, and societal dynamics that shape the spread of infectious diseases across contexts. Because these “mechanisms” are grounded in natural laws and the structure of our biology and society, they are likely to remain relevant in future pandemics even when the pathogen itself is novel. Importantly, these mechanisms reflect fundamental principles that can inform public health decision-making even before detailed quantitative evidence becomes available. Because they capture fundamental properties of disease spread and societal response, they remain valuable under substantial uncertainty, including when the characteristics of a novel pathogen are still only partially understood. In this paper, we therefore compile and synthesize core mechanisms of pandemics across disciplines, many of which are familiar to specialists but less accessible to decision-makers outside those fields, and present them in a form accessible to readers involved in pandemic preparedness and decision-making, including policy-makers, public health practitioners, and interdisciplinary researchers. We refer to them as the mechanics of pandemics.

Whether an outbreak escalates into a pandemic depends on the characteristics of the pathogen and the society in which it spreads, encompassing behavior, governance, and technological capabilities.^{7–9} Although definitions vary across the literature, here we define a pandemic as an epidemic that has spread across multiple countries or continents, typically affecting a large proportion of the population.^{2,10} Based on lessons from recent pandemics, some fundamental epidemiological and transmission principles have become widely recognized, such as the initial exponential growth of cases. At the same time, outbreaks are shaped by dynamic social and behavioral responses, such as changing risk perception and solidarity circles. These mechanisms and others will be discussed in this paper. While they can be considered individually, pandemics emerge from their interaction, and we therefore also discuss how these mechanisms reinforce or counteract one another in practice.

To obtain a broader perspective on the mechanisms that will shape our understanding of the next pandemic, we invited experts who work at the interface of epidemiology, sociology, physics, and public health to each distill a specific mechanism underlying disease transmission, control, or prevention. The contributions were iteratively reviewed, edited, and discussed. Rather than advancing a single disciplinary perspective, this

paper aims to integrate complementary insights from multiple fields into a coherent conceptual synthesis. Importantly, the work is not intended as a unified predictive model; instead, the mechanisms are presented as conceptual principles rather than empirically calibrated predictions. Our examples are not intended to be exhaustive; rather, they synthesize expert perspectives and lessons learned from recent pandemics, focusing on the fundamental aspects that can guide our understanding and mitigation of future pandemics.

Effective prevention requires monitoring the system rather than the outbreak

Emerging and re-emerging infectious diseases are not isolated events but natural outcomes of evolutionary and ecological dynamics.^{11,12} Pathogens continually evolve in animal reservoirs, environmental niches, and human populations, shaped by selective pressures including immunity, interventions, and habitat change. The critical transition occurs at spillover: when a pathogen crosses from its reservoir, animal, plant, or abiotic, into a susceptible human host.^{9,13} When introduced into a largely immunologically naive population, these pathogens may trigger extensive outbreaks either due to their natural transmissibility or stochastic amplification, yet most spillover events fail to sustain onward transmission.¹⁴

It is essential to distinguish the initial spark of spillover from the subsequent establishment of sustained human-to-human transmission. Each spillover represents an ecological accident—for example, the alignment of a shedding animal and a susceptible human—that has the potential to ignite epidemic spread.¹⁵ However, the magnitude and public-health impact of an outbreak are not determined by transmission potential alone, but also by population vulnerability, heterogeneity in disease severity, and condition-specific mortality risk.¹⁶ Near the epidemic threshold, random fluctuations in contact patterns, importation timing, vector abundance, or environmental conditions can generate large but transient outbreaks even when transmission is subcritical or only marginally supercritical, or extinguish sparks with pandemic potential by chance.^{5,17} When a pathogen with epidemic potential ($R_0 > 1$) continues to spread in a population, new cases are expected to grow exponentially, and stochastic extinction soon becomes unlikely. This mechanism therefore interacts directly with early exponential growth: once sustained transmission is established, even short delays in detection or response can translate into large increases in cumulative incidence. It also interacts with superspreading, since rare spillover events or small transmission clusters may either disappear unnoticed or expand rapidly before routine case-based surveillance detects a clear signal. Prevention based only on observed outbreaks therefore

responds to a late manifestation of a deeper system-level transition.

Therefore, when an outbreak does occur, reactive interventions—introduced only after detection—are, by design, deployed too late and are consequently costly.^{4,18–20} Effective prevention should also act upstream, by detecting instability within the broader One Health system before it becomes visible as a human outbreak. Preparedness, then, rests on continuous, multi-scale surveillance of a shared biological system, defined by interdependent species and environments (see Fig. 1).^{12,21} The applicability of this mechanism depends on surveillance capacity, data integration, and institutional trust. In high-resource settings, system monitoring may include wastewater sampling, genomic surveillance, sentinel animal monitoring, and environmental data streams; in lower-resource settings, it may rely more heavily on community-based surveillance, syndromic reporting, animal-health networks, and targeted ecological monitoring. Because upstream warning signals are often indirect and probabilistic, effective use also requires careful risk communication, so that authorities can justify early action before visible human outbreaks occur without undermining trust through poorly explained false alarms. Recognizing this interdependence is a scientific and societal imperative: preventing the next pandemic begins with acknowledging that our vulnerabilities are, and will remain, collective.^{4,9,22}

Further implications of this system-level view are discussed in Appendix A.

The effect of combined NPIs can be redundant

In the early phase of a pandemic, when effective vaccines and medications are unavailable, nonpharmaceutical interventions (NPIs), such as mobility restrictions and mask mandates, constitute the primary means of mitigating pathogen transmission. Their effectiveness is measured by their reduction of the effective reproduction number, and several studies have quantified their impact in different settings.^{23,24} Although effective, NPIs entail substantial societal costs; therefore, identifying the most cost-effective and least invasive combinations is crucial for both compliance and effectiveness.

However, because NPIs may share overlapping mechanisms of action, the effect of combined NPIs is often redundant, which means that the combined effect of two measures is smaller than the sum of their individual effects. For example, school closures would incentivize parents to work remotely, thereby reducing their mobility so that additional “work from home” mandates would not affect them (see Fig. 2). However, measures can also be synergistic and thus more effective when combined, e.g., testing and targeted isolation,^{25,26} or paid sick leave and quarantine adherence.^{27,28} This systemic perspective can be formalized as an

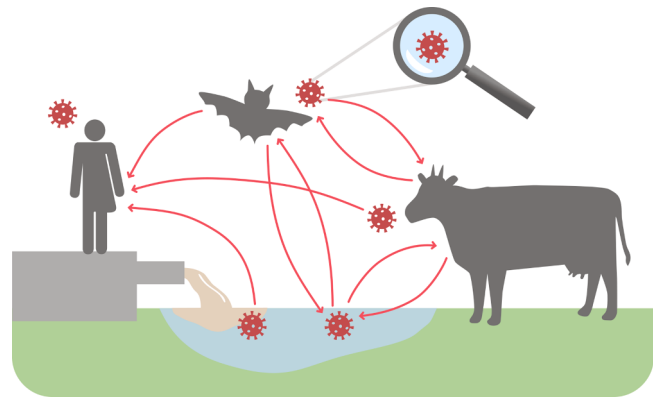


Fig. 1: Pathogens evolving in environmental reservoirs can spill over to the human population, each spillover being a “spark” with the potential to ignite epidemic spread within the immunologically naive population. These sparks unfold against slower-moving structural variables, including climate, land use, and mobility, which determine the ultimate outcome, ranging from transmission dies out to escalation into a global outbreak.

“algebra of NPIs”, where interventions interact through context-dependent rules that capture redundancy, synergy, and diminishing returns (see Appendix B).

All of this has direct consequences for policy design. First, it highlights that policymakers should not rely solely on static rankings of interventions, but instead assess how measures interact within the existing portfolio of controls,²⁴ though estimating these interactions in practice can present substantial challenges (see Appendix C). Second, if interventions have redundant effects, then stronger measures are over-proportionally difficult to achieve. The need for such strong measures can often be avoided through early, decisive action. Third, entirely different combinations of NPIs can achieve the same epidemiological effect.^{23,29} In

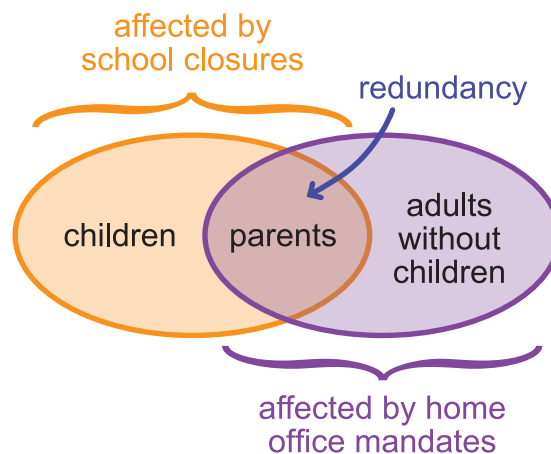


Fig. 2: Non-pharmaceutical interventions often have redundant or synergistic effects when combined. One plausible explanation for this is the overlapping effect they may have on contact patterns and transmission probabilities.

summary, NPIs do not operate as independent levers but as interacting elements of a complex system. Policies should therefore be designed and evaluated not only in terms of their direct epidemiological outcomes but also in terms of their broader societal impacts and feasibility in real-world governance.^{20,24,30,31}

Reducing event size can reduce new infections quadratically

Reducing the attendance at social gatherings (i.e., reducing the density of people at a given event) reduces disease transmission. However, the mathematical relationship is, in many cases, stronger than commonly assumed: the expected number of new infections decreases quadratically with the reduction in attendance.³² For example, reducing attendance to half reduces infections to one-quarter of the original level. The mechanism is as follows: Restricting the maximum attendance at events decreases both the number of infections that can be brought in and the number of susceptible individuals who can become infected. This results in a multiplicative (i.e., quadratic) reduction in the expected number of new infections: If only a fraction $\alpha < 1$ of a base turnout attends, then only α can cause infection, and only α can become infected. As a consequence, expected new infections scale as α^2 .^{32–34}

When wanting to extend this quadratic effect from a single gathering to all social gatherings, it implies that evenly thinning out attendance across activities is more effective than fully shutting down some while leaving others untouched. For example, assume there are only two independent activities, A and B, with the same transmission-relevant characteristics: contact intensity, room size, indoor/outdoor activity, and duration. Reducing attendance at both activities to half yields an overall activity reduction of half, but a reduction in expected infections to one-quarter (see Fig. 3). On the other

hand, completely shutting down activity B, but allowing individuals to pursue activity A as before, reduces the overall activity level again to half, but only reduces the expected number of infections to half (see Appendix D).

The effect has been shown for COVID-19 and also applies to other infectious diseases where the force of infection depends on the density of infectious individuals, i.e., when the well-mixed assumption holds. In such cases, preparedness should emphasize moderate restrictions across many sectors rather than complete closure of a few.

Limiting sporadic contacts prevents infections between clusters

Disease transmission dynamics depend not only on the number of contacts but also on their intensity. In this respect, human contacts can be broadly classified as repeated or sporadic, recognizing that real-world interactions span a continuum of contact frequencies and durations between these two extremes. Repeated contacts, such as those among family or co-workers, define clusters that limit the local outbreak sizes of respiratory infections. On the other hand, people have sporadic, one-time interactions, which allow pathogens to be transported between clusters (see Fig. 4). Understanding the interplay between these types of interactions is crucial for designing effective interventions.

In the context of respiratory infections, the number of individuals an infectious person can infect through repeated contacts, such as the daily interactions with the same household members and co-workers, is limited. Typically, such repeated contacts occur with a higher contact intensity and longer duration, leading to a high transmission probability per contact. In contrast, a set of daily-varying contacts, such as interactions on public transport or with strangers at work, provides a much larger set of potential transmission pathways,

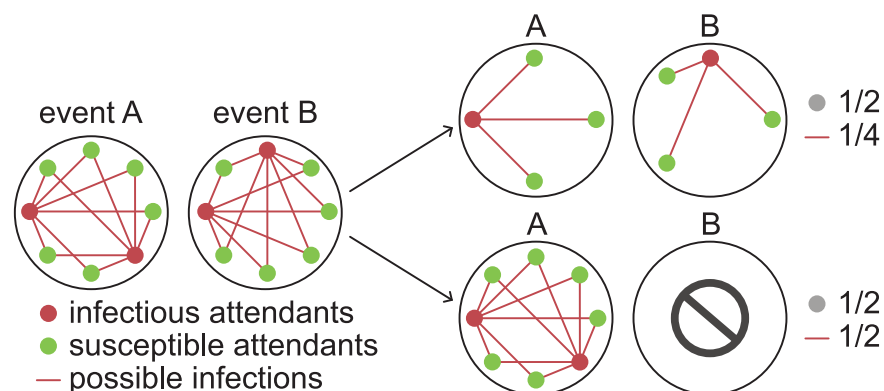


Fig. 3: Restricting the maximum attendance at events decreases both the number of infections that can be brought in and the number of susceptible individuals who can become infected. This results in a multiplicative (i.e., quadratic) reduction in the expected number of new infections. As a consequence, even though in both scenarios above total attendance is halved, in the upper scenario the possible infections are reduced to 6, whereas in the lower scenario they are only reduced to 12.

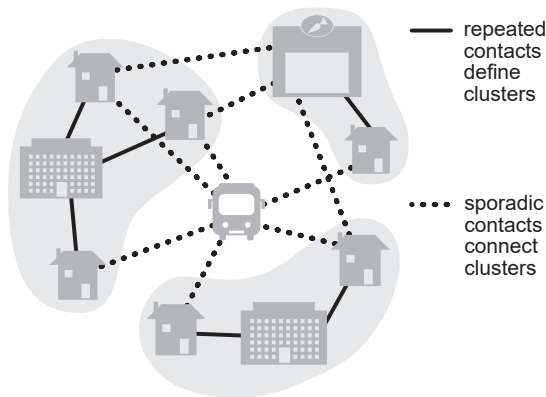


Fig. 4: Contact networks are a combination of repeated and sporadic contacts. Repeated contacts, typically organized in clusters (e.g., workplaces or households), naturally limit the number of individuals an infectious person can infect. Sporadic contacts provide paths for a pathogen to spread from one cluster to another. Thus, limiting sporadic instead of repeated contacts is often better for epidemic control.

even if daily contact numbers are the same. Here, the transmission probability per contact is often smaller than for repeated contacts. Still, as the infectious period typically lasts for multiple days, individuals with varying daily contacts may infect a much larger pool of people. The difference in number of infected individuals between repeated and daily varying contacts becomes especially pronounced for pathogens with high transmissibility. In this case the susceptibles in the repeated contact are rapidly depleted while daily varying contacts result in a new pool of potentially susceptible individuals each day.

Real-world contact networks are not tree-like but exhibit clustering: households, classrooms, and friend groups show high levels of triadic closures, the common a friend of a friend is my friend effect. This clustering further reduces epidemic spread, as many infection pathways overlap. For example, in a small household, the first infected individual can spread the pathogen to all other members. In that case, the second generation of infected cannot transmit the pathogen anymore as the pool of susceptibles has been depleted.³⁵ Limiting social contacts to highly clustered groups, i.e., allowing individuals to only (or mostly) interact with their “social bubbles” of repeated contacts in case of an epidemic, can thus substantially reduce the effective reproduction number and the social burden caused by interventions at the individual level.^{36,37}

Early and decisive action limits exponential pandemic growth

At the onset of a pandemic, disease spread is facilitated by the lack of widespread immunity. Under such conditions, where most of the population is susceptible,

classical mathematical models predict exponential dynamics (see Fig. 5). This leads to a simple exponential behavior, controlled by the average number of new cases generated by each infected individual. If the effective reproduction number R is larger than one, $R > 1$, the number of infections grows exponentially. If, on the other hand, $R < 1$, the number of infections decrease exponentially over time (see Appendix E). This has profound consequences for the early pace of an outbreak and for the design of intervention policies.

The first consequence concerns the timing of interventions: early action in the face of exponential growth means that delayed interventions must mitigate an exponentially larger incidence (see Fig. 5).^{20,38,39} An intervention deployed one generation later must control an outbreak that is about R times larger, and after k missed generations, the number of infections to be managed can grow by roughly R^k . This exponential growth means that a disease with $R > 1$ can escalate from a handful of cases to a major outbreak within a few weeks (depending on generation time). The second consequence concerns the intensity of interventions: mitigation efforts need to bring the reproduction number below the critical value of $R = 1$. Otherwise, case numbers will continue to grow exponentially, although at a slower growth rate, meaning that the infection wave is mainly shifted in time rather than avoided.⁴⁰

In summary, early, sufficient interventions can stop exponential growth before case numbers become large. The window of opportunity in which a targeted effort can avoid a larger outbreak narrows quickly as cases accumulate.

Superspreading diseases are highly susceptible to restrictions on non-repeated contacts

Disease spread in real-world settings is highly heterogeneous. The term “superspreading” describes diseases with exceptionally high heterogeneity, in which a small number of individuals account for a large proportion of secondary cases.^{5,41–43} Such variability is often rooted in behavioral differences, e.g., some people having very many contacts, and others have few,^{44–46} but can also arise from biological processes, e.g., marked differences in viral shedding.⁴⁷ We can categorize superspreaders based on whether superspreading is driven primarily by contact behavior or by infectiousness. The mechanism behind superspreading has direct consequences for disease spread and mitigation.

If superspreading is driven by individuals with many contacts, it is straightforward that interventions that limit the total number of unique contacts will prevent most of the cases they could generate. More interestingly, these superspreaders are also “super receivers”, i.e., more likely to encounter the pathogen and be infected. As they are infected and immunized earlier in an outbreak, this lowers the threshold for herd

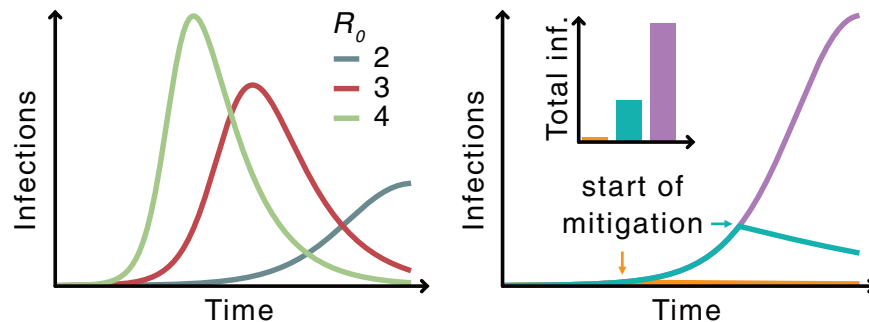


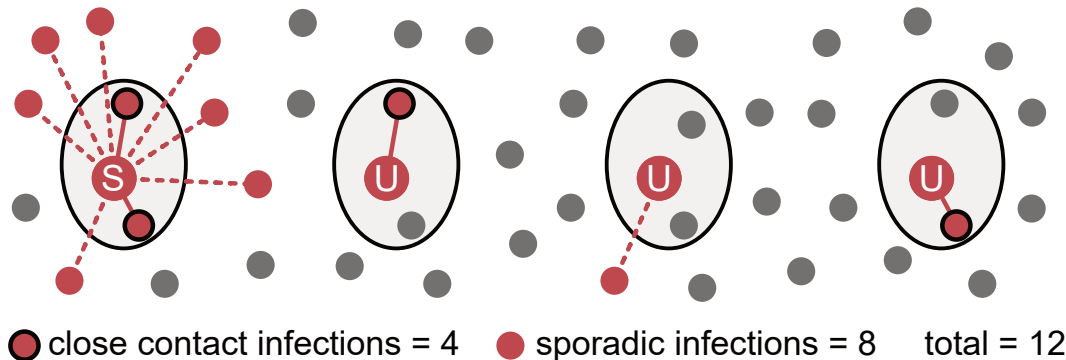
Fig. 5: The mostly susceptible population at the onset of a pandemic leads to exponential growth of infections. During this time, each infectious individual infects on average R_0 other individuals over the course of their infectious period. As a result, even small delays in implementing control measures can lead to substantially larger outbreak sizes.

immunity compared to homogeneous diseases.^{48,49} Moreover, potential superspreaders of this kind could be identified before infection, enabling highly effective targeted interventions like vaccination.^{46,50,51}

Conversely, superspreaders of the second kind (higher infectiousness, e.g., some people expressing

very high viral load⁴⁷) are not necessarily “super receivers” and are thus not immunized earlier in an outbreak. Furthermore, they will infect not only a large fraction of their close contacts, but also of their sporadic, non-repeated contacts (see Fig. 6). Therefore, interventions limiting the number of non-repeated

heterogeneous disease



homogeneous disease

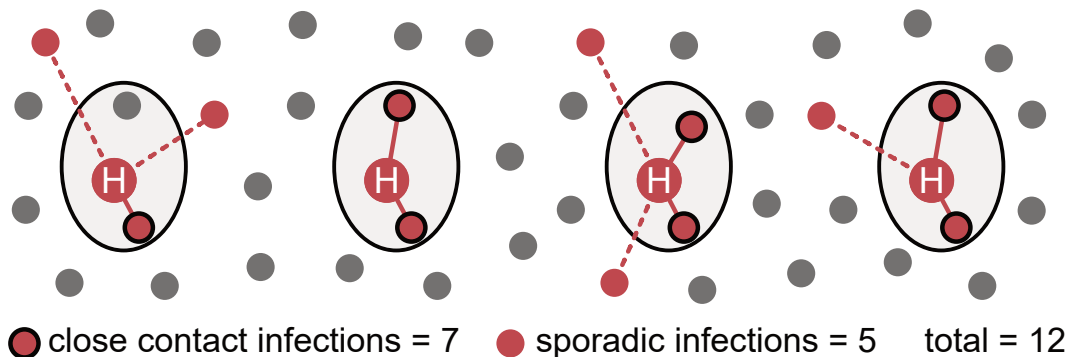


Fig. 6: In diseases characterized by superspreading, a small subset of infected individuals (S) accounts for a disproportionate number of transmissions, often through sporadic, non-repeated contacts outside their regular contact network, while most individuals infect very few (U). Consequently, restricting non-repeated contacts can substantially reduce transmission. In contrast, for homogeneous diseases, individuals infect approximately the same number of people on average (H), so limiting sporadic contacts has a much smaller effect on overall spread.

contacts will have a greater impact on transmission and containment than for a homogeneous disease with the same potential for spread.⁴²

Ultimately, while superspreading appears to be a major asset to the pathogen, it is actually an Achilles' heel. Identifying its nature early—via contact tracing and phylogenetics—enables interventions targeting specific transmission dynamics, effectively mitigating the disease's strongest pathways of spread.^{41,43,52}

Your friends are not ill if pandemic control is successful

In successful pandemic mitigation, we face a strong discrepancy between subjective experiences and objective threats to public health. This discrepancy arises in a pandemic situation with (i) a high risk of severe disease requiring intensive healthcare, (ii) limited treatment capacity of the healthcare system, and (iii) effective interventions that avoid its collapse.

In more detail, for a pathogen with high severity, we see a very low prevalence threshold at which the health care system would be overwhelmed. Consequently, interventions must suppress transmission very early and keep it below this threshold to successfully mitigate morbidity and mortality. At such low prevalence, the probability that any given individual knows about an active case within their immediate social circle is negligible. Thus, due to the effective pandemic control, most individuals are unlikely to personally know anyone who is sick (see Fig. 7).

This phenomenon can be illustrated by comparing COVID-19 to a common cold. Early COVID-19 variants were associated with high hospitalization rates per infection (requiring intensive care). In regions with successful mitigation strategies, infection rates

remained low, and few individuals knew someone who had contracted COVID-19; only a fraction of those cases were severe. In other regions with late or insufficient mitigation, many people knew of infections or even severe cases firsthand. In contrast, the common cold is considered a mild disease that does not require mitigation; consequently, most individuals know someone who has recently had a cold.

In summary, successful pandemic control poses a communication challenge due to the prevention paradox: the absence of severe illness in one's own social circle can be mistaken for overreaction rather than a sign of success.⁵³ Recognizing that not knowing anyone who is ill during a pandemic is a strong indicator of successful pandemic control is crucial for public understanding and support, and further implications of this prevention paradox are discussed in Appendix F.

Desynchronization of waves enables resource sharing

Epidemic growth from a networked perspective is a combination of local spread and transport of cases between cities or regions. Proactively limiting inter-regional mobility as a protective measure can desynchronize regional epidemic waves by delaying the introduction of cases and reducing the number of parallel infection chains.⁵⁴ This desynchronization enables the distribution of the public health burden between differently affected neighboring regions (see Fig. 8), and is thus desirable from a national perspective. Hospital beds can be shared across the country only when they are not needed everywhere at the same time. However, the mismatch between personal experiences and early-introduced interventions may be perceived as unfair and irrational by some individuals, making them particularly susceptible to generating and supporting narratives that reduce their likelihood of complying with restrictions.^{51,55,56}

Populations that perceive themselves as part of a collective effort are more likely to accept restrictions aimed at limiting disease spread.⁵⁶ In contrast, when individuals view interventions as disproportionately burdening their group, trust erodes and adherence wanes.^{57,58} In this sense, the effects of desynchronized waves are not only epidemiological but also social: regions with lower incidence may feel wrongly constrained, precisely when mobility restrictions are most critical for preventing the synchronization of outbreaks.

Preparedness, then, requires a dual strategy. Mechanistic models should be complemented by communication strategies that align narratives of fairness, solidarity, and shared identity with these underlying dynamics. While the mechanical properties of disease spread set the stage, it is narrative and identity that determine how societies navigate these trajectories. By

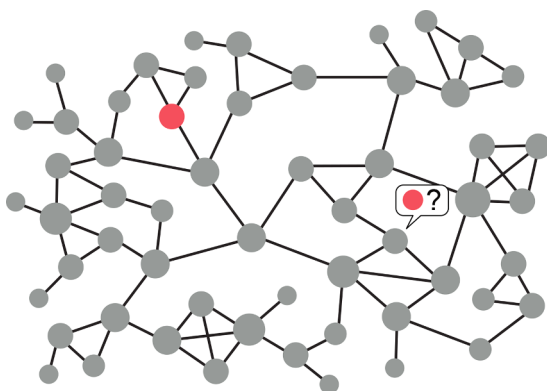


Fig. 7: In the absence of a cure or vaccine, mitigation measures are necessary to prevent hospitals from being overburdened during a pandemic. Protecting hospital capacity when hospitalization rates are high implies keeping prevalence very low, so most people are unlikely to experience firsthand the pandemic's severity or personally know someone who is ill.

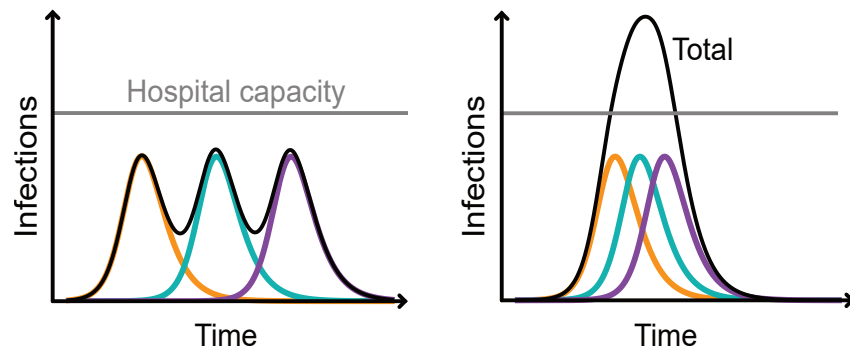


Fig. 8: By slowing the spatial spread of epidemics and desynchronizing regional waves (in color), the total number of infections (black) might stay below the hospital capacity limit. However, if these measures appear disconnected from the local reality, people may see them as overreactions, leading to frustration and reduced compliance.

integrating both dimensions, preparedness strategies can achieve both legitimacy and effectiveness.

Solidarity circles change over the course of a pandemic

The ties that unite societies matter before, during, and after crises. At the same time, crises can also intensify divisions, erode trust, and exacerbate inequalities, with consequences for compliance with interventions and undermining epidemic control.^{27,59} It remains debated whether crises foster solidarity or fragmentation.⁶⁰ While some studies emphasize the divisive effects of crises, others highlight how acute crises can activate solidaristic mechanisms.⁶¹ In this context, high institutional and interpersonal trust fosters compliance and solidarity, while low trust amplifies perceptions of unfairness, social cohesion, and receptiveness to misinformation. Likewise, perceived risk may initially encourage collective action, but prolonged or unevenly distributed risks can deepen social divisions.^{62,63} Solidarity shapes how pandemics unfold by influencing social cohesion, protective behaviors, and compliance with interventions. However, this effect is not necessarily positive, e.g., when populations are divided because some groups face disproportionate impacts, or when in-group solidarity increases hostility towards those outside one's own group. The boundaries and meanings of solidarity also depend on cultural and political contexts, shaping who is considered part of the moral community and how collective responsibilities are understood and expressed. Therefore, understanding what factors cause solidarity to materialize, grow, and shift is key to preparing for future crises.

Early in the COVID-19 pandemic, many societies experienced so-called inclusive solidarity, cutting across societal divides.^{64,65} This phase was characterized by openness to differences and willingness to support others regardless of their background, social status, or beliefs. Over time, inclusive solidarity often gave way to exclusive (in-group) solidarity—a narrowing of

solidaristic commitments to smaller, more homogeneous groups of like-minded individuals (see Fig. 9).⁶⁶ Mask-wearers expressed solidarity within their groups, while vaccine skeptics found support in theirs.^{27,67} Digital social environments and virtual communities could both sustain solidaristic ties across physical distance and reinforce segmentation into like-minded groups. For example, online studies reported increased network segmentation and hardened echo chambers,^{68–70} with severe consequences for real-world contact networks—but the evidence is still emerging. The shift from inclusive to exclusive, particularistic forms of solidarity, is not inevitable but likely when responses ignore the fact that pandemic measures may not impact all groups equally.²⁷ To prevent such unintended negative consequences, first, narratives that frame crises in terms of deserving and undeserving groups must be avoided. Second, it is essential to establish support systems—social, economic, and psychological—that are effective and inclusive. When people feel abandoned or disproportionately burdened, their capacity for broad-based solidarity declines.^{64,71}

Feedback between human behavior, spreading dynamics, and policy design

Epidemic and pandemic infectious disease outbreaks involve three major players with potentially conflicting aims: the pathogen, which tends to spread within a population; individuals, who aim to avoid becoming sick without restricting their daily activities; and policymakers, who aim to control the epidemic at minimal societal cost (see Fig. 10). These players interact to form a dynamical system with possibly complex dynamics, meaning that the actions of one player have non-trivial effects on the others. This has far-reaching consequences for how we build and interpret mathematical models for infectious diseases.

A typical example is the adaptation of human behavior in response to current incidence and interventions, which may cause interventions to

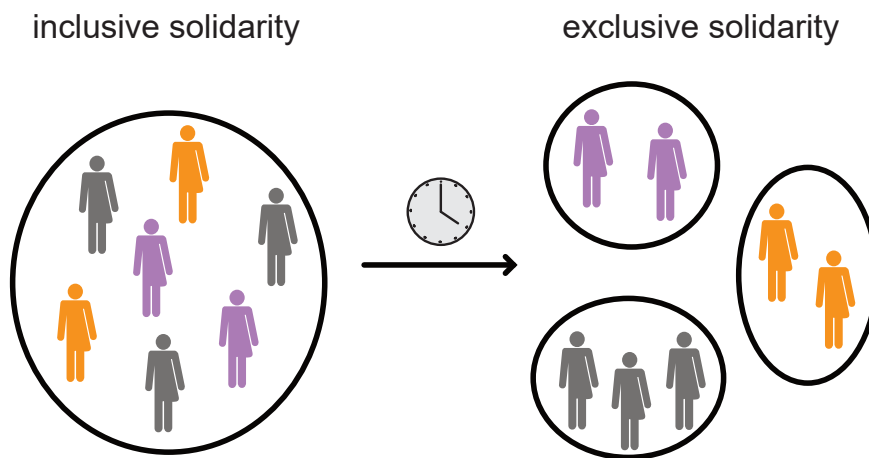


Fig. 9: Crises may ignite solidarity and prompt cohesion in societies in distress (left). Yet, prolonged restrictions often lead to fatigue, causing this effect to diminish (right). It is crucial to understand how the cohesive and repulsive forces balance each other and how this balance changes over time.

counteract or complement one another. For example, the rapid spread of risk awareness and subsequent voluntary adoption of protective behaviors, such as mask wearing and hygiene measures, complemented the mandatory NPIs during the first COVID-19 wave, helping to lower and postpone the epidemic peak.⁷² The same complementarity led to short but strict lockdowns to mitigate more effectively than prolonged moderate measures, as they avoid the effects of pandemic fatigue^{31,73} and declining adherence.⁷⁴ Conversely, the rollout of COVID-19 vaccinations in 2021 led some vaccinated individuals to increase their contact rates to pre-pandemic levels, partially counteracting the

mitigating effect of vaccination and potentially leading to higher short-term incidence.^{75–77} These effects incur complex feedback loops. While we understand their mechanics, their specific dynamics can depend strongly on the societal setting, and are thus hard to predict.⁷⁷

Modeling the interplay among health opinions that spread through social influence, the pathogens' spread, and policy responses is complex due to the heterogeneous and time-varying nature of these influences. Understanding the combined effect of such mechanisms requires first a deep understanding of each mechanism in isolation. Here, theoreticians need to strike a delicate balance between clarity and simplicity while acknowledging the complexity when translating the principles into real-world settings. A more detailed discussion is provided in [Appendix G](#).

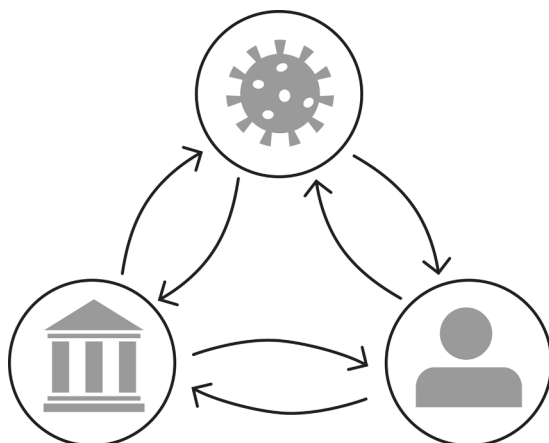


Fig. 10: Epidemics operate as a dynamic feedback system combining the competing dynamics of the pathogen, societies, and policymakers. To assess the impact of public health measures and effectively mitigate the disease, none of these three players may be neglected.

Interplay between mechanisms

As discussed in the previous mechanism ([Feedback between human behavior, spreading dynamics, and policy design](#)), the trajectory of a pandemic emerges from the continuous interaction between pathogen characteristics, immunity, human behavior, and policy responses. Consequently, the mechanisms discussed in this work cannot be regarded as isolated principles with universally fixed effects. Instead, their applicability and effectiveness depend on the epidemiological, social, and institutional context in which they operate. Importantly, this context also includes interactions between the mechanisms themselves, which can reinforce, modify, or attenuate one another.

Several mechanisms describe mathematical or epidemiological relationships under simplifying assumptions, such as homogeneous mixing or stable compliance patterns. However, real-world transmission

dynamics are shaped by heterogeneous contact networks, social inequalities, risk perception, and varying levels of institutional trust. For example, behavioral and solidarity-based processes shape compliance with non-pharmaceutical interventions (NPIs), thereby conditioning the effectiveness of mathematically derived mitigation strategies. Social mechanisms, therefore, do not merely accompany epidemiological mechanisms, but often determine not only whether but also how their theoretically predicted effects can emerge in real-world settings.

The mechanisms compiled in this work should therefore not be viewed as independent, but as interacting. In the following, we discuss several important interactions and limitations that emerge from this interdependence. Importantly, while this section focuses on interactions between mechanisms, our compilation is not exhaustive. Broader societal dimensions—including economic and psychological factors—are likewise part of the same coupled system that shapes pandemic dynamics and decision-making, as discussed below.

Solidarity and the effectiveness of combined NPIs

The effectiveness of combined NPIs ([The effect of combined NPIs can be redundant](#)) depends strongly on the solidarity dynamics ([Solidarity circles change over the course of a pandemic](#)). Mathematical analyses of intervention combinations often implicitly assume stable and homogeneous compliance across the population, overlooking the diverse social and behavioral factors that influence adherence. In practice, however, adherence to policies depends on factors such as trust in institutions, perceived fairness, social support systems, collective identity, and more. Inclusive solidarity can amplify the synergistic effects of combined NPIs by increasing voluntary adherence and mutual reinforcement of protective behaviors. Conversely, a shift in solidarity toward smaller in-groups can weaken compliance. Synergy and redundancy between the same measures can therefore change over time as solidarity circles evolve. This interaction illustrates that the real-world effectiveness of NPIs is not determined solely by their epidemiological properties, but also by the social conditions under which they are implemented.

Airborne transmission changes epidemic dynamics

The quadratic effect of reductions in gathering sizes on expected new infections ([Reducing event size can reduce new infections quadratically](#)) depends on the viability of the following assumption: that the infectious potential of an infected attendee is not diluted as gathering size increases. To the extent that a specific pathogen requires prolonged close contact for transmission, this assumption fails since, at a given time, the average distance between participants at small gatherings is less than that at large gatherings. In biological

terms, this means the effect relies on the pathogen's capacity for airborne spread, enabling transmission over long distances. Airborne transmission may also be a prerequisite for superspreading of respiratory diseases, since superspreading requires infecting distant or fleeting contacts ([Superspreading diseases are highly susceptible to restrictions on non-repeated contacts](#)). In general, epidemic control and management are a matter of choosing the right measure for the right pathogen, and mechanisms [Reducing event size can reduce new infections quadratically](#) and [Superspreading diseases are highly susceptible to restrictions on non-repeated contacts](#) may not apply to respiratory pathogens lacking airborne transmission potential.

Superspreading and contact structure

Repeated contacts and contacts within clustered groups limit the number of susceptible individuals that can be infected, whereas sporadic non-repeated contacts result in a higher number of overall infections and might connect otherwise separated social clusters, thus, creating opportunities for large transmission chains. Consequently, interventions limiting sporadic non-repeated interactions may disproportionately reduce large-scale spread, as explained in section ([Limiting sporadic contacts prevents infections between clusters](#)). As mentioned there, this is especially relevant for diseases with high transmissibility. Similarly, this reasoning can be applied to superspreading pathogens, which are not necessarily extraordinarily transmissible on average, but where transmissibility is highly heterogeneous and the resulting disease dynamics are dominated by a subset of highly infectious individuals ([Superspreading diseases are highly susceptible to restrictions on non-repeated contacts](#)). For these highly infectious individuals, the restriction of sporadic contacts again provides an opportunity to substantially limit the number of infected individuals, thus reducing the effect of superspreading in the population.

Pandemic establishment and limiting sporadic contacts

The usefulness of limiting sporadic contacts ([Limiting sporadic contacts prevents infections between clusters](#)) depends on the probability that an introduced pathogen successfully establishes sustained transmission ([Effective prevention requires monitoring the system rather than the outbreak](#)). During early outbreak phases, when stochastic extinction remains likely, reducing sporadic transmission opportunities may substantially lower the probability of epidemic establishment, particularly for pathogens with reproduction numbers only moderately above the epidemic threshold. For pathogens with high transmissibility, reducing sporadic contacts alone might not lead to extinction but could substantially slow the spread by limiting offspring to a set of static contact partners.

Solidarity circles and event-size reduction

The effectiveness and societal sustainability of event-size reductions ([Reducing event size can reduce new infections quadratically](#)) depend not only on their epidemiological effects, but also on how fairly such measures are perceived across society ([Solidarity circles change over the course of a pandemic](#)). The quadratic reduction mechanism suggests that moderate reductions across many activities may outperform complete shutdowns of selected sectors. However, implementing broad moderate restrictions requires widespread collective acceptance and trust that burdens are distributed fairly. If some groups perceive themselves as disproportionately constrained while others continue social activities relatively unaffected, solidarity may shift toward narrower in-groups, weakening adherence and increasing social fragmentation. Thus, the practical feasibility of broadly distributed attendance reductions depends strongly on inclusive solidarity and equitable policy design.

Desynchronization, solidarity, and the interpretation of low incidence

Another interaction emerges between the desynchronization of epidemic waves across regions ([Desynchronization of waves enables resource sharing](#)) and the fact that under successful pandemic management individuals are unlikely to know someone who is ill ([Your friends are not ill if pandemic control is successful](#)). Effective mitigation and mobility restrictions can desynchronize outbreaks across spatially separated populations. This lowers local prevalence at any given time and thereby creates a visibility gap between objective epidemiological risk and individual experience. As a result, precisely those regions where interventions are most effective may experience the weakest subjective signals of risk, reinforcing the prevention paradox described above. Consequently, interventions may appear exaggerated or irrational from an individual perspective precisely because they are effective at the population level. Thus, desynchronization shapes not only epidemiological burden but also public perceptions of risk and necessity.

A second important interaction arises between this visibility gap and the formation of solidarity circles ([Solidarity circles change over the course of a pandemic](#)). When individuals do not personally observe severe illness within their social networks, the interpretation of this absence depends strongly on prevailing social cohesion, trust, and perceived fairness. In contexts of inclusive solidarity, the lack of visible cases can be understood as a collective success of coordinated action, reinforcing compliance and trust in institutions. By contrast, when solidarity fragments into more homogeneous in-groups, the same absence of illness may be interpreted differently across groups, weakening shared narratives about the necessity and

fairness of interventions. Consequently, populations exposed to similar epidemiological conditions may nonetheless develop conflicting interpretations of risk and policy legitimacy, amplifying polarization.

Desynchronization of waves across regions can further strain solidarity, as geographically uneven burdens may be perceived as unjust, even when they reflect effective coordination at a larger spatial scale. In particular, populations experiencing relatively low case numbers may face greater difficulty relating restrictive measures to their own lived experience when the primary benefits lie in preventing outbreaks elsewhere, or averting future transmission. This mismatch between local experience and system-level necessity can undermine perceptions of fairness and weaken solidarity. Thus, solidarity structures critically mediate whether the invisibility of disease during successful control is interpreted as evidence of effectiveness or as evidence of overreaction, thereby shaping compliance and ultimately influencing epidemic dynamics. Consequently, interventions optimized at the population level may still fail if they are not accompanied by social cohesion, inclusive political measures, and communication strategies capable of explaining their collective rationale.

The effect of exponential growth

The exponential growth dynamics in the early phase of pandemics ([Early and decisive action limits exponential pandemic growth](#)) play a cross-cutting role in several mechanisms discussed in this work. For policy design, exponential growth makes it particularly important to account for the redundancy of measures ([The effect of combined NPIs can be redundant](#)) and behavioral adaptations that influence their effectiveness ([Feedback between human behavior, spreading dynamics, and policy design](#)). If combined interventions are evaluated under the assumption of additive effects, redundancy may result in insufficient mitigation, allowing continued exponential growth in case numbers. Similarly, behavioral responses can either amplify or weaken intervention effects, where even small changes in effective transmission rates can have large consequences due to exponential amplification. Exponential growth, therefore, reinforces the importance of accurately accounting for effects between and on measures. Beyond intervention stringency, exponential growth also highlights the importance of timing in policy responses. Small delays in implementing effective measures, driven by societal responses, can translate into disproportionately large increases in case numbers. In the case of desynchronization of epidemic waves ([Desynchronization of waves enables resource sharing](#)), early exponential growth implies that limiting spatial spread to enable resource sharing is particularly important during the early phase of a pandemic, before outbreaks across regions become synchronized. For

One Health-driven establishment dynamics (*Effective prevention requires monitoring the system rather than the outbreak*), exponential growth is embedded in the underlying transmission processes and interacts with stochastic effects and network structure to determine whether early introductions fade out or develop into sustained outbreaks. Across these mechanisms, exponential growth links early outbreak dynamics to the timing, coordination, and effectiveness of interventions. Lastly, the early exponential growth phase of pandemics creates a fundamental challenge for collective action because infection numbers initially appear small despite rapidly increasing transmission potential. Effective early interventions, therefore, require populations to accept preventive measures before the threat becomes directly visible in everyday life. Inclusive solidarity can facilitate such early action by strengthening willingness to adopt protective behaviors for the benefit of others, including vulnerable groups not yet personally affected (*Solidarity circles change over the course of a pandemic*). Conversely, if solidarity is weak or fragmented, delayed responses become more likely, allowing exponential growth to continue until the epidemic burden becomes immediately visible. Social cohesion, therefore, influences not only compliance with interventions but also the timing at which societies are willing to act.

Economic and psychological factors

Finally, it is important to emphasize that the interactions discussed above are not exhaustive. In addition to interactions between mechanisms, economic and psychological factors are not external side effects of interventions, but integral components of pandemic dynamics. Epidemics and mitigation policies jointly shape labor supply, mobility, mental health, and societal behavior, and these effects feed back into transmission and policy effectiveness.⁷⁸ Mitigation measures do entail direct and indirect costs; however, these must be understood in relation to the counterfactual scenario of uncontrolled transmission, which can generate substantial economic disruption and psychological burden in addition to direct treatment-related costs. This trade-off is also reflected in optimal control analyses, which balance intervention costs with infection-driven societal costs. They suggest that, in stylized settings, suppression strategies are optimal for severe pathogens.²⁰ While such results depend on strong simplifying assumptions and are not intended as empirical prescriptions, they illustrate the broader principle that the cost of inaction may exceed the cost of intervention, and that economic and psychological impacts are inherently intertwined with transmission dynamics rather than external to them. Nonetheless, understanding these basic mechanisms can help understand the effect of interventions.

Outlook

As witnessed during COVID-19 and previous pandemics, crises often catalyze a surge in theoretical knowledge. However, to render this knowledge actionable, we need to translate abstract theory—often accessible only to specialists—into concrete insights for decision-making. Consequently, our aim was to compile basic mechanisms of pandemic spread and mitigation. These mechanisms are grounded in natural laws and the structure of our societies, governing the emergence, unfolding, and control of pandemics. Understanding them is a prerequisite for policy design.

To identify such mechanisms, we adopted a multi-disciplinary approach, as the governing laws emerge at the interface of infectious disease epidemiology and complexity science. These mechanisms are fundamental and, therefore, often not explicitly formulated in study abstracts; moreover, they rarely act in isolation but rather jointly with other effects. Given that this level of abstraction is often considered tacit knowledge within specific fields or is not typically distilled in standard literature, a classical “systematic literature review” would fail to extract it. Hence, our work presents the cumulative experience of a diverse group of experts. This compilation is not exhaustive, but it is intended to grow and to inspire abstraction in how we study pandemics, thereby feeding into the broader discussion on pandemic preparedness.

We can only speculate about when and what kind of pandemic threat will emerge next. However, based on the mechanisms we discuss, specific pillars can support preparedness. First, we can only produce meaningful models and scenarios if we have access to proper data, and the earlier, the better.⁷⁹ This can be achieved by maintaining and adapting robust surveillance and monitoring infrastructure, together with clear rules for distributed data acquisition, access, and use. Second, in the event of a crisis, novel scientific collaboration across disciplines, institutions, and society becomes necessary.⁸⁰ They are facilitated by Open Source platforms and software, rigorous and timely peer review, and FAIR (Findable, Accessible, Interoperable, and Reusable) data principles.⁸¹ Third, strong and independent fundamental research is an immediate asset for pandemic preparedness. We do not know what challenges the next pandemic will bring, but if we have robust, independent fundamental research and a flexible funding and evaluation framework during pandemics, scientists will meet these challenges and work diligently for the benefit of society.

Compiling these mechanics required contributions across multiple disciplines; mitigating pandemics requires the joint effort of even more experts. One method to coordinate and channel scientific expertise into actionable evidence is to form expert groups. The benefits are clear: when functioning well, such groups

provide interactive, transdisciplinary peer review, guide research priorities, and partially protect individual scientists from targeted public attacks. An expert group can compile state-of-the-art evidence alongside quantified uncertainty and distinguish it from unrealistic assumptions across disciplines. However, fundamental questions regarding their governance remain: How are these groups best constituted? Should they be appointed by governments, nominated by independent scientific societies, or self-organize? What is their role in the scientific, political, and public debate? Crucially, the role of science is not to make strong recommendations, but to equip everyone with the best of our current knowledge, both certainties and uncertainties, so that society and politics can jointly reach informed decisions.

Finally, although we have dissected mechanisms in isolation to identify general principles, their combined effects in the real world are as complex as the pandemics they drive. Communicating this non-linear complexity—and the validity and limits of tools such as scenario-based modeling—is challenging by definition. We need to acknowledge the inherent difficulty of this task, particularly amid the current “infodemic” of misinformation,⁸² and never forget that defeating a pandemic requires joint, long-term efforts from across societies.

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Data sharing statement

No original data was produced for this study.

Declaration of interests

The authors declare no competing interests.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.eclim.2026.104101>.

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