https://doi.org/10.1038/s44260-025-00050-2

One pathogen does not an epidemic make: a review of interacting contagions, diseases, beliefs, and stories

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From pathogens and computer viruses to genes and memes, contagion models have found widespread utility across the natural and social sciences. Despite their success and breadth of adoption, the approach and structure of these models remain surprisingly siloed by field. Given the siloed nature of their development and widespread use, one persistent assumption is that a given contagion can be studied in isolation, independently from what else might be spreading in the population. In reality, countless contagions of biological and social nature interact within hosts (interacting with existing beliefs, or the immune system) and across hosts (interacting in the environment, or affecting transmission mechanisms). Additionally, from a modeling perspective, we know that relaxing these assumptions has profound effects on the physics and translational implications of the models. Here, we review mechanisms for interactions in social and biological contagions, as well as the models and frameworks developed to include these interactions in the study of the contagions. We highlight existing problems related to the inference of interactions and to the scalability of mathematical models and identify promising avenues of future inquiries. In doing so, we highlight the need for interdisciplinary efforts under a unified science of contagions and for removing a common dichotomy between social and biological contagions.

The science of contagions

Arguably, no scientific concept encapsulates the human experience as much as contagions. That is in large part because the situations in which people use the word "contagion" are so varied, referring to any process where a property is passed from one or many agents to others. Mathematically, the concept of contagion then inspires different forms of branching processes[1], dynamical systems[2], cascade models[3], and network systems[4]. These are used to model human genealogies[5], teaching[6,7], culture and language[8], viral trends[9,10], scientific ideas[11], innovation[12], rumors[9,13], misinformation[14,15], social movements[16,17], and obviously infectious diseases[2]. Although all of these phenomena interact to shape human life, they are unfortunately often studied in isolation, one at a time. Even simple classifications, such as distinguishing biological and social contagions, may create a false dichotomy and obfuscate their complexity. For instance, epidemics are generally shaped by multiple factors drawn from biological sciences (pathogens, genetics, microbiome) to social sciences (information, culture, behavior) and anywhere in between (nutrition, life history, living environment). All of these factors can affect both the local mechanisms by which a contagion is transmitted and its global, population-level presentation that we get to observe.

How do contagions spread? For biological contagions like infectious diseases, our intuition tends to be that if a pathogen occurs in every positive case and is not found in negative cases, it is causally responsible for the contagion as it gets transmitted from one individual to the next. Memes, as self-replicating cultural elements, might play a similar role in social contagions[18]. This intuition is the basis for the 19th-century postulates of Robert Koch's[19], which established both microbiologic methods and the necessary scientific framework of causality. This framework ushered in the

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golden age of microbial discovery in which many of the pathogens responsible for humanity's worst diseases (tuberculosis, cholera, anthrax, rabies, diphtheria) were identified. While initially useful, the one-pathogen, one-disease model ceases to accurately describe the way in which pathogens interact not only with each other but with the host, environment, and societies in which they exist.

Koch's postulates are too simplistic as they imply that every contagion is caused by a single, sufficient, and necessary pathogen, which is contradicted by empirical evidence[20,21]. For example, many microbiota-related bacterial populations can improve immune defense and prevent infection by intestinal pathogens[22]. Similarly, genetic conditions can spread vertically (from parent to offspring) but also interact with pathogens transmitted horizontally (within a generation), such as resistance conferred against malaria by the gene responsible for sickle haemoglobin[23]. Pathogens might therefore be found in negative cases because some other spreading process is interacting with the contagion. Conversely, one can imagine that pathogens can also be found in negative cases because it has yet to interact with some other synergistic contagion; this is the case, for instance, with opportunistic pathogens[24]. We detail documented biological mechanisms of pathogen interactions further in Box 1.

Similar arguments can explain why the modeling of social contagions should also move away from the assumption that one pathogen equals one contagion[25]. Obviously, no single piece of information, no single meme, and no single idea spreads in a vacuum. Different people can adopt the same ideology for different reasons. Information, ideas, or behaviors are related to each other, and they interact with each other, obfuscating classical notions of causality. For example, can a single piece of information cause anyone to develop an anti-vaccination sentiment? Probably not[26,27], especially since the discourse surrounding epidemics can be quite complicated and nuanced[28].

There is a dire need for unifying the different contagion frameworks and moving beyond the paradigm of "isolated contagion" and the classic distinction between "simple" and "complex" contagions. Indeed, modeling of biological contagions often uses a paradigm called "simple contagions" which assumes a linear relationship between exposure and transmission, while modeling of social processes often assumes more general "complex contagions". These complex contagions take different nonlinear functional forms to relate exposure to transmission[29], and they can also rely on mechanisms from social diffusion[12], sensing[30], adaptation[31], or learning[32]. This conventional approach to modeling biological or social contagions creates a false dichotomy; contagions almost always interact, and these interactions blur the distinction between contagions of different natures (and models thereof). Regardless of its nature and transmission mechanisms, a contagion is often shaped by multiple factors that can be biological, ecological, or social. The effects we wish to consider can be direct interactions between different contagious processes (e.g., two infectious diseases or rumors) or indirect interactions through important covariates (e.g., education or norms). These different types of effects are not always easy to distinguish, given that covariates like education, norms, or culture can themselves be considered as contagious at some level. We provide examples of how contagions interact across scales and scientific domains in Box 2.

The renewed interest in interacting contagions brings more to the table than new keywords like "syndemics" for synergistic epidemics and "infodemics" for epidemics of (mis-)information[33]. It also brings a fresh post-disciplinary perspective on the problem and a call for integrated efforts[34].

In what follows, we do a targeted review of the techniques for modeling interaction contagions in an attempt to highlight some key elements from that literature and map a way forward. In the section "The physics of interacting contagions," we first explain how, following lessons learned from other complex systems[35,36], "more contagions are different contagions." We discuss how the behavior of interacting contagions differs from the conventional wisdom built on models of individual contagions. This makes contagion forecasting, an inherently noisy process[37], even more intractable in the face of unknown interactions. In the section "Modeling social contagions and their interactions," we then review novel empirical and data-driven efforts, mostly coming from interacting cascades of social contagions on social media and in the science of stories. In the section "The spread of beliefs as interacting contagions," we give one particularly potent example of how contagions can interact within hosts using the study and dynamics of beliefs. Finally, in the section "The ecology of interacting contagions," we attempt to outline promising ecological perspectives that might help the science of interacting contagions avoid the trap of high model dimensionality.

Box 1 | Biological interactions within hosts

It is increasingly apparent that interactions between microbes within and across kingdoms play a critical role at the host-pathogen interface. Infection with the hepatitis D virus (HDV), for example, requires co-infection with the hepatitis B virus (HBV) due to its need for HBV-derived replicative machinery to complete its viral lifecycle. Co-infection with these two viruses potentiates one another, leading to more severe clinical disease[197]. Microbial interactions become even richer in the context of infectious disease syndromes (pneumonia, diarrhea, skin and soft tissue infections), which are often polymicrobial or the result of simultaneous or consecutive co-infection with multiple pathogens, as exemplified by post-viral bacterial pneumonia following influenza virus infection[198].

Things grow even more complicated when considering the impact of diverse microbial community structures, the microbiome. Introduction of bacteriophages can directly shape bacterial community structures while commensal bacteria directly and indirectly support or inhibit growth of other pathogens through nutrient competition, iron scavenging, and secretion of antimicrobial molecules (bacteriocins) that directly inhibit the replication of other pathogens, leading to antagonistic microbial dynamics in which the growth of one organism directly inhibits the growth of another[191]. These parasitic microbial interactions in which the growth of one organism reduces that of another are underscored by diseases such as *Clostridium difficile* infection, which is a result of a dysbiosis or imbalance in the gastrointestinal microbiome[193].

Host immune dysfunction can also alter pathogen susceptibility, disease severity, and onward transmission dynamics. Within developed regions, the most common cause of immunosuppression is iatrogenic, resulting directly from the use of immunosuppressing medical therapies[199,200]. Globally, however, the most common cause of immunosuppression is malnutrition, with 230 million children considered nutritionally deficient in 2023[201]. The impact of immunosuppression on the differential spread of infectious diseases across global populations is perhaps best highlighted by HIV, which directly infects key cells of the immune system, resulting in acquired immunodeficiency syndrome (AIDS). The synergistic impact of HIV infection on the spread of other pathogens, notably *Mycobacterium tuberculosis* and *Treponema pallidum* (causative agent of syphilis), results in regional syndemics. The overlapping prevalence of HIV/AIDS and tuberculosis is notable due to the direct immunologic effects of HIV infection, which reduces CD4+ T cell-mediated control of the intracellular bacterium, *M. tuberculosis*, promoting its spread across populations and distinction as the number one cause of death in HIV-infected individuals[202]. In the case of syphilis, HIV transmission is enhanced by the ulcerative lesions occurring during primary infection with *T. pallidum*, and HIV-induced immune suppression in turn enhances syphilitic disease progression and onward transmission of the *T. pallidum* bacterium[189]. The dynamic transkingdom interactions highlighted here illustrate the need to evolve beyond a one-pathogen one-disease model.

Box 2 | Interacting contagions across domains

Epidemics can be the result of multiple interacting contagions involving both the biological and social realms. The paths by which misinformation and disinformation can lead to worse outcomes are complex and many, but include reducing the willingness of an individual to vaccinate, obstructing efforts to contain outbreaks, amplifying political discord, increasing fear and panic, and worsening the misallocation of resources[203]. The interplay between infectious pathogens and social elements leading to the spread of both disease and infodemics is far from new. For example, concern that cow heads might sprout from the sites of inoculation led to vaccine hesitancy, which hindered attempts to control smallpox[204].

More modern examples involving the spread of infectious diseases are also plentiful: many of these involve measles, a disease which spreads so effectively in humans that small drops in a population's vaccine coverage can lead to dramatic outbreaks. The 2019 measles outbreak in the Philippines, which caused over 30,000 cases, was likely sparked by an increase in anti-vaccination sentiments[205] fueled in part by the issues with a tetravalent Dengue vaccine, which increased the risk of severe illness when infected with Dengue in those who had not had Dengue prior to vaccination[206]. Similarly, anti-vaccine sentiment spreading in Samoa after the tragic deaths of two children following nurses administering vaccine with an expired muscle relaxant instead of water[207] precipitated a drop in MMR vaccination coverage from over 75% for the first dose in 2017 to about 40% coverage in 2018, resulting in a measles outbreak that killed over 80 children in 2019[208].

Examples need not be restricted to infectious diseases. For example, multiple stunts and "challenges" have been fueled by memes and social media and can cause a range of adverse health outcomes. Examples include pulmonary damage from consuming spoonfuls of cinnamon in the "Cinnamon Challenge"[209], burns caused by applying salt and ice cubes to the skin in the "Salt and Ice Challenge"[210], and antihistamine overdoses causing deaths in teenagers participating in the "Benadryl Challenge"[211].

While these examples showcase interactions between the biologic and the social leading to worse outcomes, this interaction does not need to be negative: messaging, for example, can help people make decisions that lead to better health outcomes and could make epidemics less likely to occur. While studies have attempted to identify components that may lead to successful and productive public health messaging campaigns[212,213], epidemics are rare enough events that it is difficult to truly evaluate and generalize the requirements for a successful campaign.

Importantly, public health messages and data interact with ongoing epidemics and can contribute to the inaccuracy and inconsistency of models. Recent work studies the resulting stochastic dynamics in the fundamental epidemic reproductive number R_0[37]. If the epidemic is perceived as acute, the public may readily acquiesce to behavioral restrictions, such as isolating, masking, vaccinating, etc., so as to reduce the reproductive number. If the epidemic is perceived as decaying, then the public will want to relax their vigilance. This latter aspect was reflected in the extensive and sometimes vitriolic debate about the efficacy, or even the utility, of various mitigation strategies. This interaction results in a tug-of-war between public-health mandates and social opinions about how to respond to an epidemic, resulting in huge fluctuations in temporal dynamics[37].

The physics of interacting contagions

Mathematical modeling of contagions has a long history going back at least to the 1700s, with the creative work of Daniel Bernoulli[38] among others. In Bernoulli's work, a mathematical system of smallpox dynamics is developed, but already, the text highlights the interaction of the disease with indirect factors such as the age of individuals and the dynamics of inoculation as a method of prevention. Most modern mathematical models borrow a structure and a set of assumptions from a model called Susceptible-Infected-Removed (or susceptible-infectious-recovered, the SIR model) published in 1927 by Kermack and McKendrick[39]. In this model, infected agents transmit the contagion to their susceptible neighbors at a given rate and are removed from the dynamics through death or immunity at some other rate. One can easily generate variants of this model. For example, the SIS process assumes that the disease does not confer long-lasting immunity, such that recovered individuals return directly to the susceptible state. The SI process, in turn, assumes that there is no recovery at all.

Early studies of multiple contagions built on the SIR and SIS foundations to couple the dynamics of contagion with the evolutionary dynamics or cross-immunity between biological pathogens[40,41] or the social process of disease awareness[42]. These new models often assumed that contagions were unaffected by each other [40], or that one was necessary for the other[43], or that they were competing for a chance to spread[41,44–50], or directly in opposition to each other[42,51–54]. These efforts have previously been reviewed in detail[55]. And while there have been studies on a broader definition of interactions between contagions[56–62], the mathematical modeling community has not quite moved beyond the simplest case of two contagions interacting in simple, often symmetric, and deterministic ways. This is due in part to the richness of the behaviors that emerge even in this simple case, and in part because considering more interacting contagions makes the dimensionality of our models grow exponentially.

Importantly, the physics of interacting contagions is more than that of the simple sum of independent contagions, especially when contagions interact synergistically as they spread[63–70]. In a simple contagion model, say the SIR model described above, there is a monotonic relationship between the transmission rate and the final size of the contagion[2]. Interestingly, depending on the density of contacts in the population, there exists a critical value of transmissibility below which the expected size of the contagion is zero, and above which it increases monotonically. But importantly, this phase transition between a contagion-free state and an endemic contagion is typically continuous. That is not the case for synergistic[64] or cooperative[65] contagions, where two spreading processes can increase each other's transmissibility. These models can assume, for example, that a contact between a contagious agent and a susceptible agent transmits at a fixed rate λ, or a higher rate $\lambda' > \lambda$ if a second contagion is involved. This effect might be the same if the second contagion affects the contagious agent (e.g., a superspreader) or the susceptible agent (e.g., increased susceptibility). Regardless of the details, the system can get into a frustrated state where many contagious agents can only transmit the contagion to their susceptible neighbors if a second contagion also reaches them (see Fig. 1A). These frustrated transmissions are similar to latent heat in physical systems, leading to discontinuous phase transitions. Or, in this case, the discontinuous emergence of a large contagion as we tune the transmission rate (see Fig. 1B).

There are other elements of conventional wisdom from simple independent contagions that do not transfer to the dynamics of interacting contagions. Features of the structure of the underlying contact network that tend to slow down contagions, like triangles or any form of clustering, can now hasten the spread of synergistic contagions[64]. The intuition behind this phenomenon is simple. For an independent contagion, the optimal structure on which to spread is a tree-like network where it is impossible to backtrack when starting from a single patient zero. In that case, all connections lead to new, and therefore susceptible, agents. Clustering of connections can instead trap a contagion and cause transmission efforts to be "wasted" on already contagious agents. However, synergistic contagions

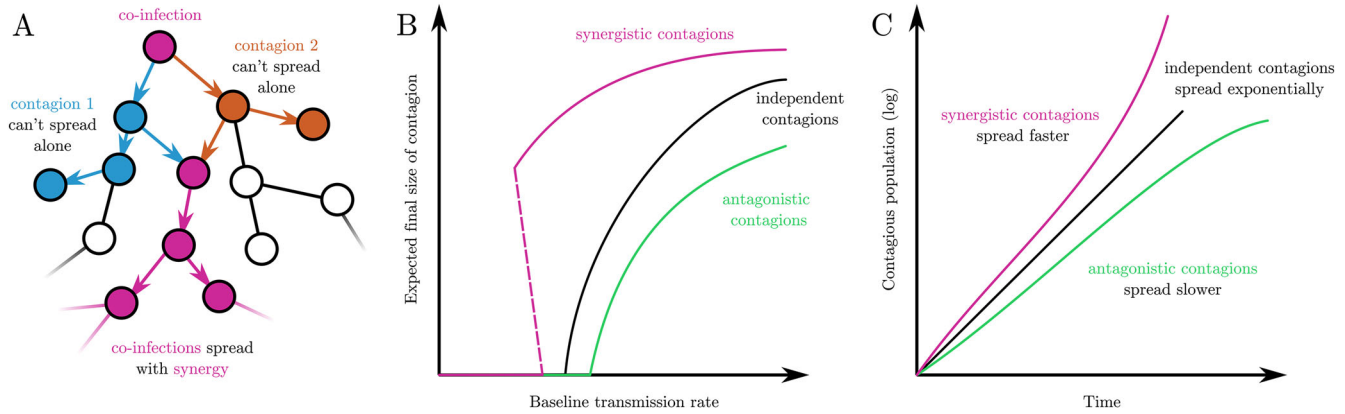


Fig. 1 | Illustration of the dynamics of interacting contagions. A Schematic representation of two synergistic contagions (blue and orange) spreading synergistically through a network. Co-infections, shown in pink, are needed to sustain the contagion and are aided by clustering. **B** Phase transition of various types of contagions. While independent contagions display continuous transitions, synergistic contagions can build up transmission potential, which leads to discontinuous transitions reminiscent of physical systems where latent heat accumulates. **C** Growth rate of synergistic (pink), independent (black), and antagonistic (green) contagions. Synergistic contagions tend to grow superexponentially since they get more likely to interact as they spread further.

Table 1 | Examples of interacting contagions across domains

Interaction	Biological example	Social example	Mixed example	Synonyms
Synergistic	HIV and syphilis[189]	Related memes/ideas[104]	Disease and anti-vaccines[190]	Cooperative
Antagonistic	Bacterial competition[191]	Political opinions[192]	Influenza and vaccines[61]	Competitive/dueling
Parasitic	*C. difficile* and commensal enteric bacteria[193]	Fake news and fact checking[194]	Disease and awareness of the disease[71]	Asymmetric

Directionality and strength of the association are not always easy to assess. For example, vaccination against influenza might remain strong even after or during a weak flu season, but awareness of a new emerging pathogen will only spread if the pathogen does as well.

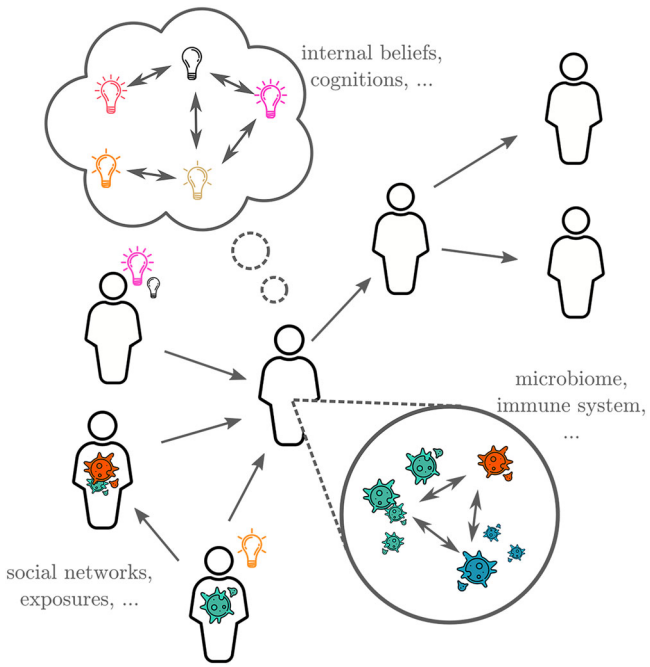


Fig. 2 | Interactions across domains and scales. Biological and social contagions (here represented by colored viruses and different light bulbs, respectively) spread through contact networks and shared physical or digital environments. Contagions can interact directly in the environment or during contact. When an agent is exposed to new viruses or ideas, the potential transmission is mediated through other layers of interactions, now internal. New beliefs or ideas interact with an existing belief system. Likewise, new pathogens interact with an existing microbiome and immune system. These multi-layered interactions can all drive the observed dynamics of contagions[195,196].

benefit from being kept together. On a tree-like network, a co-infected patient zero might transmit one contagion to one branch of the tree and another to a different branch. These contagions might then not interact with one another for a long time and, therefore, not benefit from their synergy. A small amount of clustering helps them stay together.

A similar effect is found in parasitic contagions, where one contagion benefits from the presence of another contagion but hinders its transmission in return. This is the case, for example, of positive messaging around a negative contagion, like the spread of preventive awareness against an infectious disease. The awareness is more likely to spread when the disease is spreading, and individuals exposed to the disease are more likely to be receptive to relevant information. Conversely, the preventive message is likely to reduce the transmission risk of the disease. The awareness is thus a parasitic contagion to the epidemic[71]. With asymmetric interactions, clustering can hurt awareness more than the disease, leading again to a larger epidemic than expected without clustering (Table 1).

Finally, synergistic contagions can also spread superexponentially since the more contagions spread, the more likely they are to interact and benefit from their synergy, thus further accelerating their spread (see Fig. 1C). This dynamic clashes with most mechanistic prediction models[69]. As contagions spread and the number of contagious agents grows, the more likely co-infections become, such that positive interactions become more frequent and important. For a time series of either contagion, this simple statistical effect leads to what looks like an accelerating spread or a transmission rate that increases with time. Altogether, basic intuition built by focusing on a single pathogen might not hold when looking at an ecology of interacting contagions. New modeling approaches are needed. Thankfully, ideas from the social sciences might again come into play.

All of the features described above are also signatures of complex contagions, as introduced in the previous section. In complex contagions, also called frequency-dependent models, transmission depends nonlinearly on the number or frequency of exposures such that the growth rate of a contagion can vary as it spreads[12,29]. Recent results show that synergistic

contagions can be mathematically indistinguishable from these nonlinear effects in complex contagions, such as the psychological phenomenon of social reinforcement[69]. Like peer pressure, this phenomenon means that a second exposure to an idea or behavior through a second source is more effective than the first exposure[29,72]. For example, ten friends telling you to read an article are more likely to convince you than one friend telling you the same thing ten times. That is usually not true for biological contagions, where our models assume a linear relationship between infection rate and exposure regardless of the source. Indeed, researchers often use simple linear contagions for biological applications and complex nonlinear contagions with reinforcement for social applications. Studies suggest the distinction breaks down once contagions interact.

In recent years, modeling studies have also broadened the concept of interaction structure and focused on networks with different (multilayer) interactions[73] or with group (higher-order) interactions[74]. There has therefore been a rapid influx of studies generalizing these previous models. Given that nonlinear group interactions alone create apparent complex contagions, the phenomenology of higher-order and interacting contagions is only richer. Studies have looked at competing contagions in multilayer networks where different contagions spread on different sets of contact[53,54,75,76], through higher-order dynamics[77–79], and even both[80–84]. These extensions are natural, and in fact, some of the early studies on cooperating epidemics also assumed a multilayer structure[85] or a group structure[64].

The rich dynamics of interacting contagions pose an important challenge: Is it possible to measure contagion mechanisms without explicitly controlling for all possible interactions and covariates? Social reinforcement can be defined and measured by the increase in infection probability caused by repeated exposures compared to a null model of equivalent exposures. However, this measurement might be impossible to distinguish from interactions among pathogens. Did someone share a meme because many of their friends liked it, or had they previously shared a related meme? The flip side of this challenge is that measuring social reinforcement can then be used as a proxy for interactions between contagions. Recent work has taken this perspective to justify the need for tractable nonlinear models[86]. New effective models for interacting contagions are needed, especially since we know that countless contagions of biological and social nature interact at all times and that the dimensionality of our current models grows exponentially with the number of contagions involved.

Modeling social contagions and their interactions

Attempts to operationalize and model social contagion processes first appeared in the second half of the 20th century. In the 1950s, Katz and Lazarsfeld[87] published "Personal Influence"[87], a book which would become highly influential about the nature of influence. Their finding were based on their in-the-field study of decision making by women in the city of Decatur, Illinois. Their major contribution was to introduce the "two-step" model of influence, whereby media reached local opinion leaders who then influenced their friends and acquaintances. Though Katz and Lazarfeld were clear that their two-step model was a non-universal approximation of true social influence, the idea that there exists a class of special, influential people—and consequently the importance of identifying and engaging them for the means of large-scale social change to whatever ends—would become a hardened, enduring, and broadly popularized concept[12,88,89]. As the real-data-informed field of network science developed in the late 1990s, the notion of opinion leaders would be challenged with more sophisticated social network models[90]. Nevertheless, the two-step model moved the understanding of media influence beyond the traditional "hypodermic model," revealing and elevating the role of social networks in shaping collective and individual opinions and beliefs.

In the 1960s, early considerations of how to mathematically operationalize social contagion fell to simply porting across the SIR model of mathematical epidemiology[39]. In a 1964 Nature article, Goffman and Newill explicitly cast the spread of an idea as akin to the spread of a disease[91]. It is worth reflecting on the scope and strength of their framing:

> "For example, consider the development of psychoanalysis in the early part of this century. Freud was no less host to the infectious material of the 'disease' of psychoanalysis than the person carrying the organism capable of transmitting a cold, nor is his writing less of a 'vector' carrying the 'infectious material' than the mosquito as a carrier of malaria. Jung might represent an example of acquired resistance to the disease while the resistance of the medical community of Vienna could represent innate immunity. The development of the psychoanalytic movement …was in its way no less an 'epidemic' than the outbreak of influenza in 1917 and 1918.
> One can argue similarly that Darwin and evolution, Cantor and set theory, Newton and mechanics, and so on, were examples of 'epidemics' in the world of scientific thought which were instigated by the introduction of a single infective into a population. The analogy is not restricted to science; for examples such as Christ, Buddha, Moses and Mohammed can be cited in the religious field …"

Of course, social contagions are inherently different from biological contagions as there are no "cultural pathogens" easily identified and cultured[92]. Instead, as recent studies suggest, social contagions are always shaped by other effects such as homophily or cultural[93], human biases and demographic heterogeneity[15], or because of dislike and distrust between subpopulations[94].

Clear movement beyond SIR models began (at least) in the early 1970s, when Schelling introduced his physical, checkerboard-based model of self-organizing neighborhood segregation processes[95]. Schelling operationalized social contagion with the concept of thresholds: Individuals adopt (or reject) a characteristic (action, belief, behavior, etc.) based on the fraction of those around them with that characteristic or some other influential attribute. While simple to run, Schelling's model was non-trivial to address analytically. In 1978, Granovetter showed that, absent any interaction structure, a mean-field threshold model produced informative stories of social contagion[96–99]. Like Schelling's model, a seemingly moderate population could universally adopt a behavior. These models showed mechanistically how collective uniformity could arise not from individual uniformity but rather social following.

It is worth noting that while seemingly distinct, the essential models of biological and social contagion can be reconciled. For example, a generalized contagion model incorporating memory successfully interpolates between SIR-type models and threshold models[100,101].

Abstract modeling of contagions, regardless of their nature, can only get us so far. Turning to data to validate these theories in specific domains is critical. A major, enduring problem with all empirical social network contagion is missing data, either through limited sampling of a social network (hence missing links and interactions) or due to interactions occurring outside of the sphere of observation (e.g., through direct text messages between individuals, or influence from other media across scales).

Modern social media platforms can control and monitor what people see and how they behave with high precision. But the algorithms behind major platforms are proprietary and hidden. Observational analyses of online behavior and properly formalized academic experiments promise an ethical way forward. Because of the massive data on offer, social media provides a unique lab bench to both characterize social contagion and test theoretical models.

As a first simple proxy for social contagion, one can look at cascades of re-sharing of the same content on social media (re-sharing a post, keyword, or a given URL). Under that lens, while co-infection data regarding biological contagions can be rare, social media are essentially a messy soup of countless interaction contagions[102] shaped by each other and by the social networks that support them[103]. Therefore, it is not surprising that online social networks inspired some early studies of multiple spreading processes[104]. These processes can interact by competing or complementing each other[105], and telling these mechanisms apart from noisy data can be subtle but is not impossible[106,107]. For example, Zarezade et al.[107] highlight that incorporating interactions into models enhances their predictive

accuracy of social media cascades, even when accounting for the added model parameters. In their model, a contagion spreads to susceptible individuals at a rate specified by a Hawkes process around each susceptible individual[108], a self-exciting process that relates the transmission rate of a contagion to the sum of recent exposures to the contagion itself *and* to related contagions. Related work focused on competition between memes obtained similar results, showing that the competition for potential "hosts" and their attention is sufficient to qualitatively explain the broad diversity in popularity, lifetime, and activity of memes[109]. In that model, competition between memes occurs through a mechanism of limited attention, through which a susceptible agent is only really exposed to a finite number of memes in its recent memory. The general idea being that signatures of online behavior are a reflection of recent exposure to contagions, both online and in the real world, and both social and biological. In fact, a whole subfield of digital disease surveillance has also looked at the potential of using social media data as a proxy to track the spread of infectious diseases through keywords, posts, and searches related to a disease or its symptoms[110].

Despite this abundance of data, monitoring of social contagions is complicated by the fact that the signature of any contagion can mutate quickly[111]. Early methods often focused on tracking specific hyperlinks or proper names[112,113], which is appropriate for short timescale before new hyperlinks or names start describing the same information. Other approaches tend to aggregate many cascades, such as mixture models or dynamic topic models[114], which by analogy would be similar to tracking respiratory illnesses without tracking individual virus families[115]. To follow a social contagion as it evolves, it is possible to track specific short phrases that are unlikely to mutate[111], but this approach is hard to generalize to visual non-text memes. More recent work has therefore moved to multi-modal deep learning models to identify memes[116], classify them in families[117], and attempt to understand their relations[118] or predict their potential for virality[119].

Beyond the spreading of internet memes and the modeling of social contagions lies a developing, data-driven science of stories and beliefs. That stories matter profoundly to people and societies seems to be both unclear and obvious: Stories are portrayed by some as being just for entertainment, while by others as the core of being human[120–124]. Even so, the centrality of stories has gained ground, as has the conception and possibility of measuring stories through distant reading[125–131,131,132]. Understanding how stories develop, spread, interact, and compete with each other is of utmost importance to understanding social phenomena; from myths and conspiracy theories to hate speech and counter speech[133]. Operationalizing the measurement of stories and interactions between story elements is critical to advancing these aims (see Box 3).

The spread of beliefs as interacting contagions

Interacting contagions in social networks also differ from biological contagions as they often spread in signed social networks that explicitly distinguish between positive links (representing trust and favoritism, generally toward in-group members) and negative links (representing distrust and enmity, generally toward out-group members). Positive links facilitate the spread of contagions within the group of like-minded individuals, while negative links can lead to rejection or counter-adoption of ideas from distrusted sources. This sign-based framework provides essential structure for understanding how contagions compete across partisan divides. Recent dynamical modeling[134] reveals that the relative strength of positive versus negative links determines whether the social network converges toward consensus or diverges toward polarization, explaining the rapid polarization of stance toward masking and lockdowns during the COVID-19 pandemic[135].

While much of the work we have surveyed so far emphasizes the spread of contagions across agents, beliefs rarely spread in isolation from other beliefs and related cognitions such as knowledge, social norms, and emotions[136,137]. Therefore, beliefs and opinions (which we here use somewhat interchangeably[138]) spread not only across social networks but also within internal belief systems (Fig. 2). This interaction of contagions across levels makes the study of beliefs a prominent and powerful example of interacting contagions.

In social networks, the spread of some beliefs is often easier after a group has already been "infected" with a related set of beliefs. During the COVID-19 pandemic, people who grew up in the former East Germany and were thus more accustomed to the idea that the government can enforce certain behaviors were more likely to support the idea of mandatory vaccination[139]. Beliefs about whether abortion should be banned or not are strongly related to prior "infections" of a group by particular political and religious beliefs[140]. Beliefs about what is normal and desired in a group can change in line with shifting perceptions of what the majority of group members believe or do[141], with examples ranging from the support for gay rights[142] to the support for extreme political views[143].

Beliefs also spread within individual minds, affecting existing beliefs and related cognitions and clearing the path for "invasions" of a belief system with other novel beliefs. For example, when one becomes skeptical about the safety of vaccinations, this can open doors to the development of skepticism for those supporting vaccination, such as scientists and the government. This distrust may make one more likely to accept further related beliefs and conspiracies[144]. Emotions can further facilitate belief spread within an individual's mind. Even a temporary "infection" with fear of death can lead one to adopt dislike and prejudice towards beliefs and groups different than one's own[145].

Models of belief dynamics could profit from incorporating the effects of interacting socio-cognitive contagions. Many existing belief dynamics models focus on the spread of only one belief at a time[138,146–148]. To model the spread of several related beliefs or cognitions more broadly, one can proceed in at least two ways[149].

One way to model the dynamics of several beliefs is to assume that each one is affected by a summary of all the others. Many plausible summary measures have been proposed. Some are normative, such as Bayesian reasoning[150,151] or logic[152]. Others are more descriptive, aiming to mimic actual cognitive processes. Examples are averaging strategies[153], frequency-based strategies such as plurality or tallying[154], birth-death dynamics like Moran processes[155], or various non-compensatory strategies such as deciding based on the most important belief or consideration[156].

Another way to model interacting belief contagions is to model the whole network of beliefs. This idea is not new[157], but formal models of such networks that can enable analyses of interacting contagions within belief systems have started to be developed only recently. In these network models, nodes are typically beliefs, and edges represent influence between them[158–161]. However, nodes can also represent concepts, and edges represent beliefs about the relationship between them[162]. Empirical work has been done to assess the assumptions of these models and their predictions, but this is still a developing area of research[163–165].

Modeling interactions as a network of contagions may provide a unified way to explore a wide spectrum of contagion dynamics as emergent behaviors. For example, models of interacting beliefs have been shown to break the dichotomy between simple and complex contagions and integrate both dynamics under a single framework[166]. Linear, simple contagion dynamics occur when the existing belief system is primed to accept a new belief, while complex contagion dynamics occur when a new belief challenges the coherence of the existing belief system, echoing the interacting spreading processes discussed in the section "The physics of interacting contagions."

Recent work has coupled the dynamics of disease spread with a spectrum of internal states that can represent awareness or behavior and can vary according to social contacts through some social sensing process[167,168,169], Incorporating the richness of internal belief interactions is a promising direction for further study of the multiple interactions that occur between and within individuals during social and biological contagions.

The ecology of interacting contagions

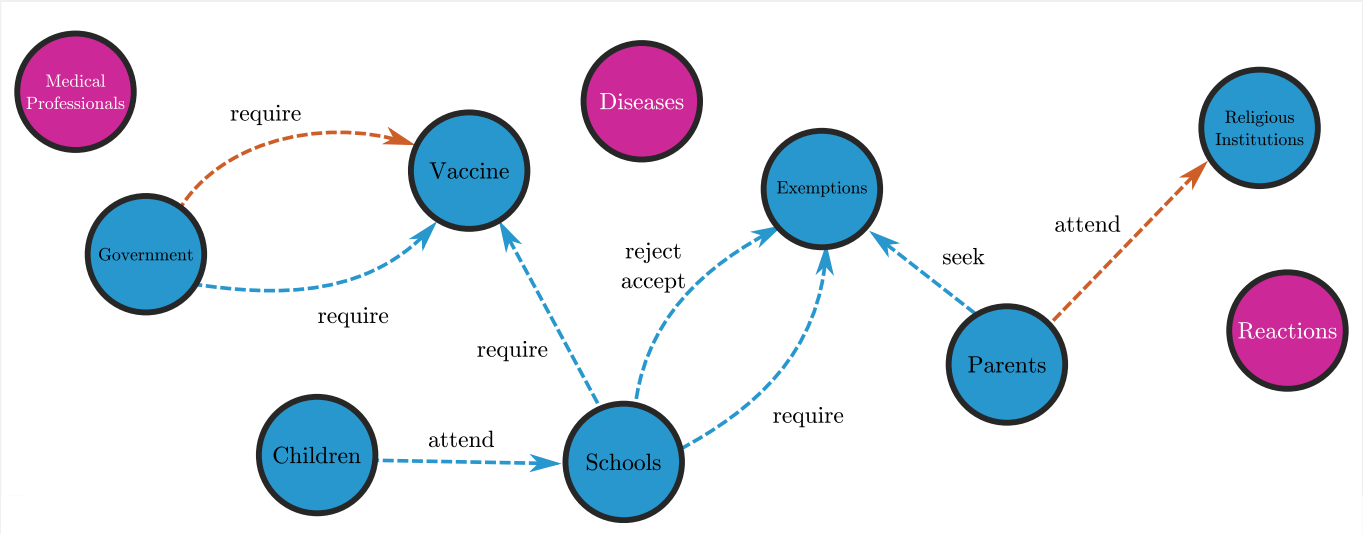
Across biological contagions, abstract models, stories, and beliefs, the previous sections have hinted at two important challenges in the modeling of

Box 3 | Narrative interactions within and across stories

In crisis situations, there is a strong predilection to construct narratives in which some outside force threatens the integrity of an inside group[214,215]. These narratives allow people to collaborate in shaping the story, resulting in a consensus on who is "inside," who is "outside" (and therefore can pose a threat), the range of potential threats, and the possible responses to those threats[216,217]. Evaluative comments, either as framing devices or within the narratives themselves, offer clues to the stance of storytellers and their audiences about the acuity and severity of the threat and endorsement of particular strategies or outcomes[218].

Stories of this nature—and the conversations in which they are embedded—can be modeled as an interactant narrative framework graph, where the nodes consist of actants (individuals, groups, institutions, places) and the edges consist of the relationships between those actants[219,220]. The graph, extracted from conversations, models the range of possible actants and their interactions; and a story, either complete or in part, can be modeled as a directed acyclic graph activating some subset of the nodes and edges[221].

As people react to the unfolding crisis and its attendant threats and disruptors, new actants and relationships are proposed (e.g., by new posts to the forum) and possibly added into the graph, while others are less frequently activated and disappear. Several mechanisms limit the graph from growing too large, including the concept of "tradition dominants," so that new actants who duplicate the role of existing actants in the graph are subsumed into the existing actant's role, and the "law of self-correction" so that variations on assignations to insider/outsider status, the range of threats and possible reactions are aligned with existing conditions[222–224]. These mechanisms and constraints allow new stories to contribute to the evolving conversation, while the constraints on the graph provide support for the group's beliefs and narrative motivation for actions based on those beliefs. Consequently, the storytelling environment not only reacts to changes in external conditions but affects those external conditions through real-world actions while maintaining a degree of narrative stability.



A post about religious exemption to vaccination requirements, "A friend of my daughter got her "shots" for this one and now has multiple health issues. Anxiety attacks, rashes, and a lot of fatigue. My girl is not getting them. No way…" can be represented as an interaction subgraph between concepts of the broader discussion forum, which comprises thousands of interlocking stories and story parts.

interacting contagions. First, the number of states that agents can take grows exponentially with the number of contagions spreading in a population. Therefore, the dimensionality of our models also tends to grow exponentially with the number of contagions we wish to consider[170]. Second, contagions can interact on multiple scales. Interactions can occur internally, within agents, mediated by an agent's immune system (biological contagions) or cognition (social contagions). Interactions can also occur across agents, mediated by the environment, transmission pathways, or local culture and norms.

Despite these challenges, there is an obvious need to consider interactions between large numbers of contagions, in the thousands or more, that shape our everyday lives. Therefore, we need to be able to go beyond individual-based models whose dimensionality grows exponentially and start thinking in terms of an ecosystem of contagions. This call for an ecology of contagion is a recent development[29], and we here outline some promising directions.

There already exist models to infer interactions in large numbers of contagions from detailed time series of social data[106] and biological data[171]. More rarely, we also sometimes have access to co-infection data, often static counts of individuals infected or involved in multiple contagions. From co-infection data, previous studies have built inference frameworks based on permutation tests[172] or joint-species distribution models[173].

Biologically, emerging technologies such as multiplex PCR panels, high-throughput sequencing (metagenomics, metabolomics, single-cell sequencing), and organoid model systems can allow us to begin to better understand the rich interrelated dynamics occurring across microbial communities at pathogen-host interfaces. Integrating these datasets and their insights into contagion models can enhance their accurate reflection of the true biology at play. In the social sciences, it is also a challenge to build broad datasets that include multiple beliefs in a time-resolved manner. Large language models are providing new methods for stance detections and belief quantification that could also resolve interrelated social dynamics[174].

That being said, co-occurrence alone is not evidence of interactions[175], regardless of how many covariates are included in the analyses. The ideal dataset would, of course, be a time series of individual co-infections, where these existing methods could be combined. Despite the scarcity of such data, a significant number of approaches have already been developed[176]. A more spatial and ecological understanding of co-infections could also help

improve inference. This question has been tackled in models[170,177], but separating spatial correlations from actual contagion is not a simple task[178]. For social contagions, surveys can help sort these different effects[92]. For biological contagions, the majority of studies of co-infections rely on observational data or case notes, but a minority of studies attempt to survey specific populations[179].

On the modeling side, it is unclear what the ecology of contagions should look like. Can models of food web stability inspire a new generation of models of contagions? Recent models attempt to study the endogenous emergence of interactions between contagions using a game-theoretic perspective[180], evolutionary models[181], and co-evolutionary interactions with measures to promote or hinder these contagions[182]. These models encourage us to think of interactions as endogenous or emergent features of contagions and not just as fixed parameters or model mechanisms.

The field of ecology itself is challenged by modeling interacting contagions and currently faces significant analytical limitations related to the issues reviewed above. Levins' metapopulation models, for instance, are very similar to standard epidemiological models[183], representing the dynamics of individual patches that could either be empty (susceptible) or occupied (infected) by a given species, and studied for the conditions allowing species persistence at the regional level (outbreaks). Such models have been extended to multiple interacting species[184], but ecologists lost track of the rapidly growing number of potential community states[185]. Solving this modeling challenge may require a fundamental shift in state variables; instead of representing the dynamics of a set of individuals, which could either be susceptible or infected, perhaps a solution would be to represent the dynamics of the growth rates themselves as functions of the entire community of contagious agents. Focusing directly on the contagions rather than on their hosts might help relax assumptions about the binary nature of contagious states or about the different relative timescales of biological, social, and evolutionary contagious forces. With a proper formulation of the dynamical functions, the extensive toolbox of community ecology models could then be used to investigate problems of coexistence, feasibility, stability, and higher-level interactions among contagions.

We thus join our voices to the many recommending ecological models of infectious diseases[186] and contagions[29]. This perspective is necessary to appreciate the intricate and dynamic web of interactions between viruses, animals, parasites, humans, behaviors, and beliefs. Furthermore, under this lens, the study of emerging beliefs, stories, or epidemics can then borrow from known theories regarding invasive species in classic ecological modeling[187].

We end with a question not often posed in the context of these increasingly detailed frameworks. To what end do we pursue these models? The ecological and holistic approach argued above is an increasingly common one, as different fields attempt to produce spatial and population-level understanding in times where social, ecological, and biological variables are in constant flux[188]. Is our goal with these frameworks to actually predict and forecast? Is that truly a litmus test of our understanding of these rich, stochastic, and inherently noisy processes? Even then, what concrete observables are we aiming to predict? New cases of a disease? New believers in a conspiracy theory? Emergence of new contagions or stories? If we adopt an ecological perspective, our goal should probably be to better understand the structure of interactions and to predict their impact on the stability and hierarchy of existing contagions. Importantly, this new objective aims for an ecological science of interacting contagions where we can study contagions as a system in and of themselves, and not just through their individual parts of pathogens and hosts.

Data availability

No datasets were generated or analyzed during the current study.

Received: 27 April 2025; Accepted: 21 July 2025;
Published online: 01 September 2025

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Acknowledgements

This work is based on the workshop "Dynamics of interacting contagions" held at the Santa Fe Institute. The authors acknowledge financial support from the Santa Fe Institute, the Vermont Complex Systems Institute (award #2242829 from the National Science Foundation), and from the Translational Global Infectious Diseases Research Center of Biomedical Research Excellence (award P20GM125498 from the National Institute of General Medical Sciences).

Author contributions

L.H.-D. oversaw the writing. L.H.-D., P.S.D., and M.G. wrote the initial draft. All authors reviewed and edited the manuscript.

Competing interests

L.H.-D. is the Editor-in-Chief of the journal. A.A. is an Associate Editor of the journal. M.G. is a member of the journal's Editorial Board, and the authors declare no competing interests.

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