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Information: a missing component in understanding and mitigating social epidemics

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Social epidemics or behaviorally based non-communicable diseases are becoming an increasingly important problem in developed countries including the United States. It is the aim of our paper to propose a previously understudied aspect of the spread of social epidemics, the role of information in both causing and mitigating social epidemics. In this paper, we ask, can information be harmful, contagious, and a causal factor in social epidemics? In the spread of biological epidemics, the causal agents are biological pathogens such as bacteria or viruses. We propose that in the spread of social epidemics, one of the causal agents is harmful information, which is increasing exponentially in the age of the internet. We ground our idea in the concept of the meme and define the concept of an infopathogen as harmful information that can spread or intensify a social epidemic. Second, we ask, what are the best tools to understand the role of information in the spread of social epidemics? The epidemiological triad that includes a host, agents (and vectors), and the environment is extended into a quad by including information agents. The quad includes the role of information technologies as vectors and the impact of the social environment. The "life cycles" of pathogens in biological epidemics and infopathogens in social epidemics are compared, along with mitigations suggested by the epidemiological quad. Challenges to the theory of infopathogens, including the complexities associated with the spread of memes and the role of behavior in the spread of epidemics are discussed. Implications of the theory including the classification of harmfulness, the freedom of speech, and the treatment of infected individuals are also considered. We believe the application of the epidemiological quad provides insights into social epidemics and potential mitigations. Finally, we stress that infopathogens are only part of social epidemic development; susceptible hosts, a favorable environment, and availability of physical agents are all also required.

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Introduction

In this article, we investigate the idea that information can play an important role in transmitting social epidemics. In developed countries, biological epidemics have become increasingly rare (COVID-19 being a tragic exception); the label "epidemic" is now commonly also used to describe disorders that appear to be increasing in frequency or severity and have high social costs. A social epidemic can be defined as behaviorally based non-communicable disease (Egger et al., 2003). Examples include suicide (Cheng et al., 2014), violence (Gilligan, 1996; Slutkin, 2012), opioid addiction (Williams and Bisaga, 2016), and obesity (Ravussin and Ryan, 2018). Despite efforts to combat these epidemics, many of these epidemics appear to be increasing in developed countries. For example, in the United States, obesity has increased from 30.5% in 2000 to 42.4% of adults in 2018 (Hales et al., 2020), and the overconsumption of oxycodone has increased by 500% from 1999 to 2011 (Kolodny et al., 2015). Suicide is now a leading cause of death in the United States and has increased from 10.7% in 2001 to 14% in 2017 (NIHS, 2019). In each of these cases there are physical agents involved such as junk food for obesity, drugs for opioid addiction and weapons, chemicals or structures for suicide. Additionally, we propose a missing component, harmful information acting as a contagious agent that affects human behavior and spreads these epidemics.

In this article, we ask, can information be harmful, contagious and a causal factor in social epidemics? This is a critical question since there is an unresolved debate about whether social epidemics such as violence should be classified as epidemics (Greene, 2018; Loeffler and Flaxman, 2018; Slutkin et al., 2018b; Williams and Donnelly, 2014). One of the key arguments against their classification as epidemics is that there is no causal agent (Greene, 2018). There is also debate in the literature if social epidemics are contagious, including obesity (Christakis and Fowler, 2013; Cohen-Cole and Fletcher, 2008), violence (McDavid et al., 2011; Williams and Donnelly, 2014) and suicide (Cheng et al., 2014; Mercy et al., 2001). We make the case that information can be infectious and pathogenic (infopathogenic), by citing information compiled from studies on the spread of memes and social epidemics, including violence. We also discuss the role played by the internet, media and social media in acting as a vector for harmful information. Second, we ask, what are the best tools to understand the role of information in the spread of social epidemics? To address this question, we modify the epidemiological triad to include information and demonstrate the use of an analog disease life cycle to explain the spread of social epidemics. Finally, we use the epidemiological quad to generate insights for managing infopathogens and for suggesting potential interventions.

Can information be pathogenic?

Although social epidemics such as violence are age old problems, they have potential to spread and be intensified when vectored by modern information technologies. Digital information is increasing at an exponential rate, tenfold every five years (Koehl, Liu, and Paniccia, 2011) and along with beneficial information comes information that could be harmful. In addition, social media has made harmful content increasingly difficult to detect and monitor (Allcott and Gentzkow, 2017). The potential for increased spread of information pollution (Pandita, 2014) and the potential implications for society requires a fresh look at the role information plays in social epidemics. While information pollution (that is the production of detrimental information) by the internet has been addressed by several authors (Cai and Zhang, 1996; Pandita, 2014), we set out to demonstrate the causality of harmful information as one factor in the spread of social epidemics.

The idea that information can behave like a living organism or gene has been popularized by the concept of the meme. A meme is information that can be copied; an idea, belief, behavior, or style that spreads from person to person within a culture (Blackmore, 2000). The process by which memes survive and change through cultural evolution has been likened to the process of the natural selection of genes in biological evolution (Dawkins, 1976). A proposed model of meme diffusion includes multiple factors such as meme fitness, the individual sources' competence, social network structures, societal, contextual, geospatial and technical factors, and the practical outcomes of meme diffusion (Spitzberg, 2014).

There were several key concerns raised about meme theory. First, there was no ready way of deciding what "information chunks" count as a meme or how they contribute to cultural change (Armitage et al., 2001). Second, unlike genes, ideas rarely copy with high fidelity as they are likely to be modified during transmission through social networks (Armitage et al., 2001). The third issue was that there was no real way to study how memes were selected (Fracchia and Lewontin, 2005). Eventually, the study of memes declined and publication of the Journal of Memetics ended in 2005. Memes would likely have been forgotten had it not been for the rise of the internet. Now, the meme is a widely used term for an idea (usually represented as an image) that spreads quickly (especially on the internet) and as a virus of the mind (Burman, 2012). Importantly, the digitization of memes provided a mechanism to resolve these three objections to the study of memes. Digital memes can be explicitly defined and their reproduction and diffusion through social networks can be quantified and studied (Adar and Adamic, 2005; Gruhl et al., 2004; Weng et al., 2013). Moreover, error-checking mechanisms in DNA replication (Moraes et al., 2012) responsible for the high fidelity replication of genes may have an analog in digital memes: sharing, retweeting, and copying-pasting are all means of replication that have extremely high fidelity that may allow for improved study of memes.

Despite the controversy surrounding memes, the spread of information through social networks (such as memes that go viral) can be understood as information epidemics or contagion (Adar and Adamic, 2005; Gruhl et al., 2004; Hodas and Lerman, 2014; Steeg et al., 2011; Weng et al., 2013) and modeled using the susceptible-infected framework (Kandhway and Kuri, 2016). Obesity, happiness, smoking and cooperation all exhibit evidence of social contagion and can be visualized as networks (Christakis and Fowler, 2013). Another pertinent example is the spread of fake news, which has been studied as if it was an epidemic (Jin et al., 2013; Kucharski, 2016; Tambuscio et al., 2015). If information (including misinformation) can be infectious, then it can also be thought of as pathogenic. Dawkins (1976) noted that a meme essentially parasitizes a brain, turning it into a vehicle for the meme's propagation in just the way that a virus parasitizes the genetic mechanism of a host cell. An infectious agent or a pathogen causes disease or illness to its host, such as an organism or an infectious particle is capable of producing disease in another organism (Anon, 2008). Evidence suggests that observing violence or being impacted by it can alter and dysregulate specific mechanisms and pathways in the brain (Slutkin, 2012), which is akin to disease development. Memes that are absorbed by someone's mind and result in detrimental changes or behavior may be thought of as pathogenic. We build on the concept of the meme to define an infopathogen as harmful information that can spread or intensify a social epidemic. Importantly, while biological pathogens must be transported by physical means, we suggest infopathogens are much more mobile when vectored via internet technologies.

One of the most powerful demonstrations that information can be pathogenic is the understanding and treatment of violence as if it was an epidemic caused by a contagious disease (Slutkin, 2012; Slutkin et al., 2018a). It has long been recognized that violence appears to spread as if it was an epidemic or a contagion (Berkowitz and Macaulay, 1971; Fagan et al., 2007; Loftin, 1986; Papachristos et al., 2015; Patel et al., 2013; National Reserach Council, 1993). Turchin (2012) noted that violence in civil wars or periods of socio-political instability behaves as an epidemic. Violence such as mass killings and school shootings have been shown to be contagious (Towers et al., 2015) as well as violence among adolescents, which is spread through social networks (Bond and Bushman, 2017). The use of the epidemic analogy provides helpful insights. Individuals may be considered infected if they have suffered from violence or have been traumatically threatened (Slutkin, 2012). Violence is spread from person to person by direct victimization, intentional training, or by visual observation, like an infectious epidemic (Slutkin, 2012; Tracy et al., 2016). This understanding led to the development of successful social interventions, similar to the ones deployed for biological epidemics (Slutkin, 2012). The interventions involved the employment of new types of social workers to reduce the expression of symptoms and interrupt the transmission and spread of violence. These community-based interventions focused on behavioral change in individuals who committed or were impacted by violent acts and might be high-risk candidates for committing violent acts in the future.

The proposed classification of violence as an epidemic has been controversial (Greene, 2018; Loeffler and Flaxman, 2018; Slutkin et al., 2018b; Williams and Donnelly, 2014). Although violence can be modeled like an epidemic (Green et al., 2017; Myers, 2000; Wiley et al., 2016), one study demonstrated that gun violence appears to only slowly diffuse over time (Loeffler and Flaxman, 2018). However, once a pathogen is widespread, the spatial-temporal distribution of an epidemic may be more strongly influenced by the spatial properties of the social environment, and the same may be true for violence. Another argument against violence behaving as an epidemic was that there is no "violence bacteria" (Greene, 2018), however, in this paper we argue that harmful information or infopathogens are the infectious agent. A third argument against violence acting like an epidemic is the suggestion that violence (like many other non-communicable diseases (Rea, 2009) is primarily caused by toxic chemicals in the environment (Greene, 2018), an issue that will be discussed below in the context of the epidemiological triad.

In the case of violence and other social epidemics, transmission of the "disease" appears to also happen through the media acting as a vector. Observing violence in media can lead to the perpetuation of violent acts in the community or at home (Huesmann et al., 2003; Hurley, 2004) and exposure to media violence is a risk factor for aggression (Anderson et al., 2015). Cigarette advertising promotes cigarette consumption, but a total ban on cigarette advertising can reduce cigarette consumption (Saffer and Chaloupka, 2000). Suicide appears to spread as an epidemic through imitation (Cheng et al., 2014; Swanson and Colman, 2013), thus raising concerns about childhood exposure to suicide in the media (Howard, 2017). Another well documented example is internet video memes that led to the consumption of laundry detergent pods by adolescents (Murphy, 2019). The introduction of American television programs glamorizing thin body types is associated with the development of eating disorders in Polynesian societies where these disorders were previously unknown (Williams et al., 2006). This last case demonstrates that as humans have enabled the international spread of biological diseases and pests through air and sea transportation (Magarey et al., 2009), human inventions assist the global spread of infopathogens to new social environments. This may be of particular concern in societies without prior experience in mitigating a particular social epidemic. From these examples, we infer that while society has greatly benefited from low cost, highly diverse and universally available print, TV, social, and other media, some of this content has been infopathogenic with detrimental impacts on society and warrants research into potential interventions. We stress that, most memes, like most microorganisms, are not harmful to humans and relatively few memes and microorganisms could be classified as pathogenic, even fewer still cause epidemics. In the next section, we modify the classic epidemiological triad to incorporate information to better understand social epidemics.

The epidemiological quad

The epidemiological triad is a widely used model for infectious diseases and consists of an agent (that is a pathogen and associated vectors), a susceptible host (that is a person), and favorable environment (Gordis, 2009). The classic epidemiological triad (or disease triangle) is a paradigm widely used to explain disease spread, for example in malaria humans are the host, the disease-carrying mosquito is the vector and warm wet weather that creates standing water is the environment. The epidemiological triad has been adapted to behaviorally based Non-Communicable Diseases (NCDs) by including physical agents and vectors in place of biological agents and vectors. Examples include obesity (Egger et al., 2003), motor vehicle injuries (Haddon, 1980; Lett et al., 2002), violence (McDavid et al., 2011) and lung cancer from smoking cigarettes (Egger et al., 2003). In existing literature regarding the triad being applied to behaviorally based NCDs that are social epidemics, the agent is the active cause of the disease, whereas the vector is the carrier of the agent. For example, with lung cancer the carcinogens are the agent and the vector is the cigarette (Egger et al., 2003) or alternatively the cigarette is the agent and manufacturers are the vector (PSU, 2018); with motor vehicle injury, speed is the agent and vehicle is the vector (Haddon, 1980); and with gun violence the perpetrator is the agent (McDavid et al., 2011) and the gun is the vector. In these cases, the host is still human, and the environment is the set of situations that enable the host to interact with the agents and vectors.

While in these examples disease theory is applied to social epidemics, it thus far has been done so inconsistently; labeling the cigarette as either the vector or the agent depending on how the model is applied, as an example. The epidemiological triad provides insight for mechanisms to control diseases; when the triad is inconsistently applied to social epidemics, effective controls cannot be adequately determined and executed. To promote a more universally applicable disease theory to social epidemics, we expand this epidemiological triad to a quad to include an important factor not seen in biological diseases, the role of infopathogens or information as memes that influence human behavior and contribute to the spread and sometimes the intensification of social epidemics (Fig. 1). The utility of the quad for analyzing social epidemics is that "info-pathogenesis" is possible where all four factors occur simultaneously and thus mitigations that reduce this interaction reduce the spread of social epidemics. Mitigations will be discussed later but first each factor is explained in detail.

The infectious agent and vectors. The first part of the quad is the information agents (infopathogens) and associated vectors if applicable, analogous to a pathogen, parasite or virus, and vectors such as a mosquitos and ticks that may carry it. As defined earlier, an infopathogen is harmful information (or memes) that can spread or intensify a social epidemic. The infopathogen and the

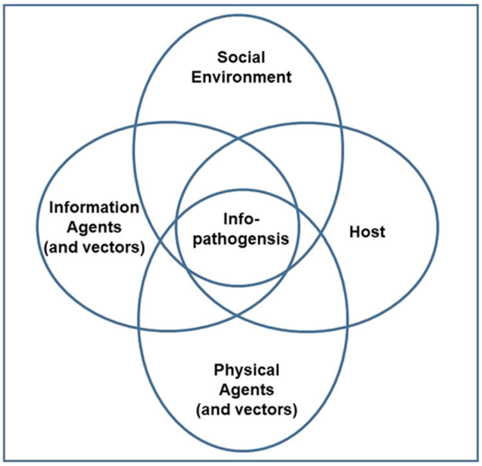

Fig. 1 The epidemiological quad for social epidemics. The quad is modified from the epidemiological triad (Egger et al., 2003) and defines the role of information agents and vectors in addition to physical agents and vectors, the social environment and susceptible hosts as a model for describing how social epidemics spread.

social epidemic is the analog of the pathogen (or virus) and the biological disease. In the same way that biological pathogens can become more or less virulent when exposed to certain selection pressures such as host density (Borovkov et al., 2013), infopathogens and memes also undergo selection for increased reproductive success (Heylighen, 1998). Vectors for infopathogens include media, social media, and advertisers, but they are not always necessary as infopathogens can spread directly from person to person, especially when people share the same belief systems. While there is still a role of personal choice when exhibiting behaviors in a social epidemic (committing acts of violence, overeating unhealthy food, smoking cigarettes, etc.), we assert that infopathogens promote the desire of humans to engage in detrimental behaviors. While humans may influence memes and infopathogens with direct modification in order to influence populations and elicit certain behaviors (propaganda, fake news, etc.), this varied origin makes them no less an infopathogen (in the same way humans could genetically modify biological pathogens for biological warfare).

The social environment. In a communicable disease triad, the environment represents the set of factors that are necessary for disease transmission. Crowded living quarters are more likely to spread human diseases, high humidity and wet soils are more likely to spread fungal diseases within plants. If the conditions of the environment are not suitable for disease transmission, infections are low (if any) and an epidemic does not occur. The same is true for the social environment for infopathogens, which may or may not promote infopathogen transmission and increased host susceptibility. For infopathogens, this is the social environment, which includes the built infrastructure; industrial and occupational structure; labor markets; social and economic processes; wealth; social, human and health services; power relations; government; race relations; social inequality; cultural practices; the arts; religious institutions and practices; and beliefs about place and community (Barnett and Casper, 2001). Social environments are experienced at multiple scales, are dynamic, and change over time as the result of both internal and external forces. It is also well known that the social environment strongly influences the susceptibility of an individual to biological diseases (Farmer, 2001) and infectious diseases disproportionately affect the poor (Bhutta et al., 2014). The same is often true for many social epidemics, for example in Jamaica, criminal violence has roots in poverty and urban decay, political patronage, and more recently organized crime (McDavid et al., 2011).

Power relations includes an individual's ability to have an effect on other individuals actions or on their dispositions to act (French Jr., 1956). Power has the ability to alter the spread of information within networks, an obvious example being state censorship. Thus, an individual's behavioral response is not just a function of an individual's physiological and psychological properties (Menge, 2018) but rather power is a function of network configuration and network composition, including human resources, technologies, and knowledge (Law, 2007). As such, social networks may entail a level of complexity beyond that of biological networks where relationships might be governed primarily by factors such as distance, density, host range and climate.

When developing a model using this infopathogen quad to understand a particular epidemic, the degree to which the different social environmental factors affect the epidemiology of the infopathogen may be very simple or very complicated. It is important to explore and enumerate all mechanisms in the environment, which may contribute to the spread of a social epidemic.

Physical agents. The third part of the quad are physical agents, material things that can be utilized in carrying out the negative behavior that is instigated by the meme, facilitating the spread of a social epidemic including guns (McDavid et al., 2011), cigarettes (Egger et al., 2003), drugs (Kolodny et al., 2015), and highly processed foods (Egger et al., 2003). Physical agents are necessary for symptom expression when infected with an infopathogen, but attributing the spread of social epidemics to physical agents and vectors alone can lead to unsuccessful mitigation strategies. Attempting to eradicate drugs from neighborhoods, illegalizing certain types of firearms, restricting availability of high-sugar drinks at fast food restaurants are attempts to curb social epidemics without addressing the role of harmful information and personal choice in exhibiting expression of social epidemic infections. They are important to consider as part of the disease quad, but must be distinguished from the infectious particle (infopathogen).

The host. The fourth part of the quad is the host (in social epidemics, humans) and their degree of resistance/susceptibility to infopathogens. The degree to which a population of infected individuals expresses symptoms is variable. Just like with biological diseases, most exposed individuals may resist infection, while most susceptible (at-risk) individuals may be asymptomatic, some may be asymptomatic yet contagious (a disease carrier), while other may have symptoms that vary from mild to fatal. A host may be resistant to disease because of past exposure to a similar pathogen or have resistance, which may be a function of age, nutrition or genotype. With biological pathogens, the Susceptible-Exposed-Infected-Recovered framework can describe the epidemiological process following the exposure of an individual to an infectious agent (Hethcote, 2000). Similar stages can also be described for memes. For example, Heylighen (1998) formalized this by describing four stages in meme replication: assimilation, retention, expression and transmission. Assimilation requires the individual or host to respectively notice, understand, and accept the pathogen, analogous to the biological process of infection, which requires complex interactions between host and pathogen (Forst, 2010). Retention requires the host to remember the meme and the longer it is retained the greater the chance of replication. The next stage of meme replication is expression, which requires a conscious decision by the host to communicate the meme. Expression of the meme can be by speech, text, pictures or behavior (Heylighen, 1998). Finally, transmission of memes requires a physical carrier or medium, including those with potential for mass multiplication such as media, including social media. Analogous to the way biological pathogens must reproduce sufficiently in hosts to sustain epidemics, failure at anyone of these steps prevents meme replication and prevents a meme going viral. Having described the epidemiological quad, in the next section we discuss how the disease life cycle can be another tool to understand the role of information in social epidemics.

A comparison of pathogenic and infopathogenic "life cycles"

The spread of social epidemics can be understood in analogous terms of a pathogen life cycle in which infopathogens act as the infectious agents with susceptible and non-susceptible human hosts. The use of biological analogies to describe information is not a new concept. Human interactions with the information economy have been described as an information ecosystem (Davenport and Prusak, 1997; Fidel, 2012). In human epidemiology, disease life cycle or zoonotic cycle diagrams are used to both understand and illustrate how diseases are transmitted and spread. More elegantly, "Zoonotic diagrams function as no less than an epidemiological Rosetta Stone, forging universally recognizable linkages between humans and animals" (Lynteris, 2017). In particular, these diagrams have particular application for public outreach and education, a recent example being the Centers for Disease Control and Prevention's public health communication campaign for Ebola (Lynteris, 2017).

To illustrate this concept, we demonstrate the spread of a fictitious model pathogen (or parasite) that is spread by human to human contact and vectored by mosquitos (Fig. 2A). The disease cycle begins when a susceptible individual is bitten by a mosquito that is carrying the pathogen or comes into contact with an infected individual. The exposed individual may or may not develop disease symptoms after a certain latent period. This infected individual can then spread the disease to mosquitos or other individuals that are not infected. The population of the mosquito vectors is greatly increased when warm, wet weather creates environmental conditions, such as standing water that favors rapid multiplication. Resistant individuals can become susceptible if the pathogen mutates to a new pathotype with increased virulence. This type of life cycle can be described by the SEIR model, with components that include susceptible, exposed, infected and recovered (Hethcote, 2000). The susceptible-infectious modeling approach has already been used to understand the spread of social epidemics (Tracy et al., 2018) including alcohol consumption (Santonja and Shaikhet, 2012), sexually transmitted diseases (Newman, 2002) and opioid addiction (Battista et al., 2017; Befekadu and Zhu, 2018). Included in Susceptible Exposed Infectious Recovered SEIR models is also the concept of an incubation or latent period (Fig. 1) between when an individual is exposed and when symptoms of infection appear (incubation period) or when an individual becomes infectious (latency period).

A comparable figure can be created for an infopathogen, in this case, we use a hypothetical example of violence associated with Neo-Nazi hate groups (Fig. 2B). This begins with the creation of infopathogens, for example posting extracts from Mein Kampf, the biography of Adolph Hitler, on social media as memes. These infopathogens can then be reproduced, resulting in exposure of many thousands of individuals from a single infected individual, since a small fraction of individuals account for most social media posts (Spitzberg, 2014). Susceptible individuals may become infected if the social environment is favorable to the spread of the infopathogen, for example, social circles where racial separatism is encouraged. The spread of the infopathogen can also occur directly through social contact with infected or symptomatic individuals and this might be the most important mechanism for many social epidemics. In the case of violence, this incubation or latent period can be either very short or very long (Slutkin, 2012). Most infected individuals likely never publicly express symptoms that incur social costs, but this might occur if exposed to a trigger, for example a right wing march where anti-social behavior is encouraged. As the cycle of violence continues and more individuals are impacted, the infopathogen may mutate to infect individuals who are previously resistant, for example, non-violent counter-demonstrations becoming violent. Finally, incarceration of individuals in information sterile environments may result in intensification of their symptoms and the development of new content, for example Mein Kampf was written in prison (Heinz, 1934).

Exposure to infopathogenic content such as Mein Kampf memes could also cause an individual to become a pacifist, perhaps something akin to the hypersensitive response in plants, where rapid cell death in the local area of infection protects the rest of the plant. While observing violence in real life or in the media can lead to committing acts of violence, not all who witness the violence in the media commit violence themselves, much in the manner that individuals in a population are naturally variable in resistance or susceptibility to disease. In the next section, we return to the epidemiological quad to better understand how interventions can be deployed against social epidemics.

Interventions using the epidemiological quad

The potential usefulness of the quad is the role it can play in framing and suggesting potential interventions (or mitigations) in four categories: information, social environment, agent and host (Fig. 3). It is important to note that some interventions may benefit the general population, while others are applicable only to exposed or infected individuals (Egger et al., 2003; Slutkin, 2012).

Information interventions. Interventions to slow or stop social epidemics in regards to infopathogens can include manual or automated tagging of potential infopathogens, algorithmically filtering out the most damaging ones, strategic introduction of positive or corrective news to slow or stop the outbreak, and reducing financial incentives that encourage unscrupulous publishers (Nguyen et al., 2012; Pogue, 2017; Weedon et al., 2017). Information interventions are not a new idea (Metzl, 1997) but have been discussed more earnestly for social media since the 2016 US federal election and include diversifying information sources to which a user might be exposed. Pech and Slade (2004) proposed modifying memes that promote terrorism to create forms that inhibit terrorism. A substantial body of research has been developed to combat fake news (Ciampaglia, 2018; Ciampaglia et al., 2015; Tambuscio et al., 2015) and could provide critical insights for combating the spread of infopathogenic content.

Social environment interventions. Social interventions include surveillance, forecasting, policy, legislation, and social change (Egger et al., 2003). There are numerous examples of surveillance and forecasting in human and botanical epidemiology such as monitoring for Zika virus (McGough et al., 2017). In situations where individuals have no choice but to engage in unhealthy behaviors, such as eating fast food in areas that are fresh food deserts (Beaulac et al., 2009), social change to allow individuals to

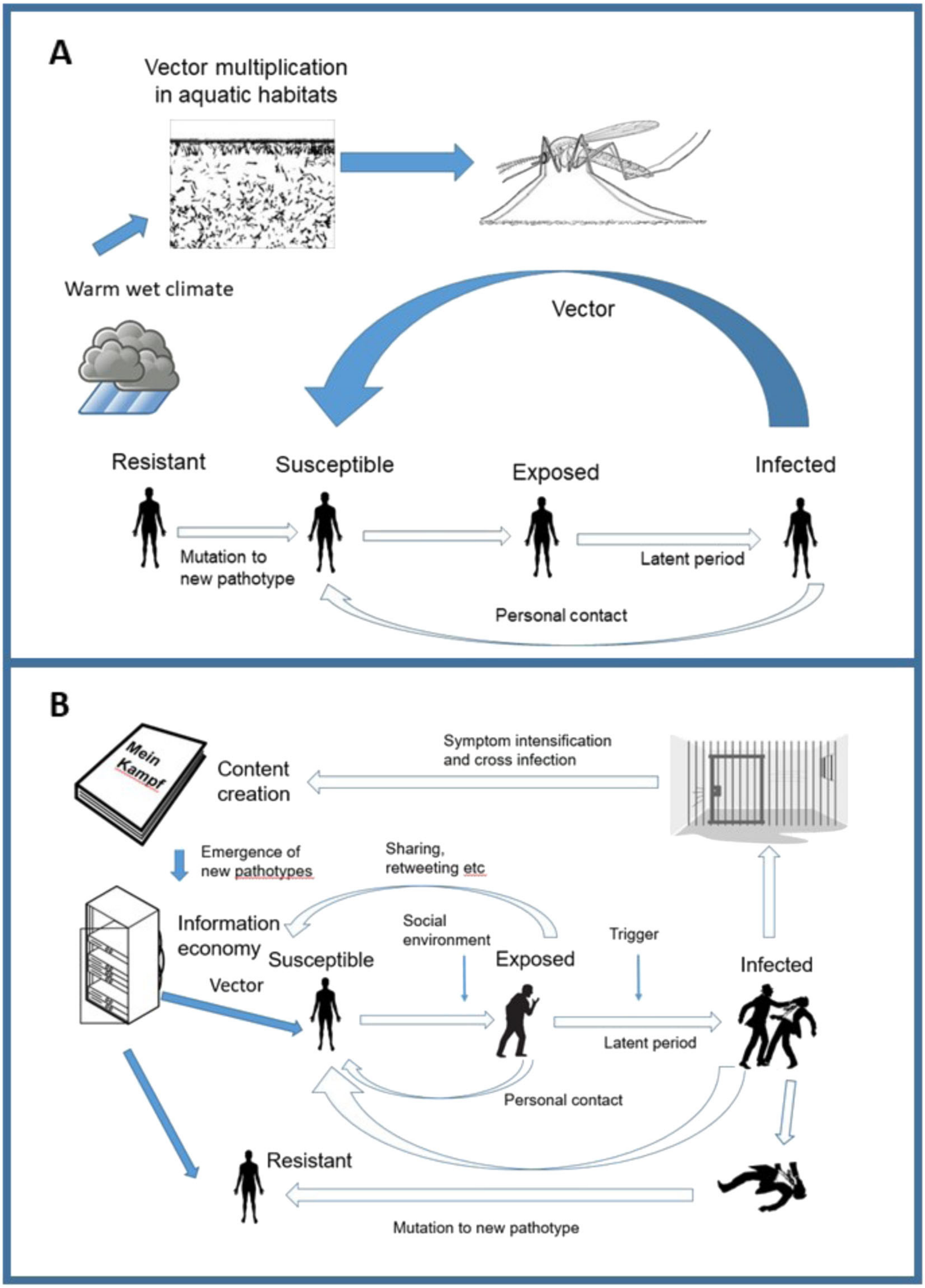

Fig. 2 A comparison of the life cycle of biological pathogens and infopathogen. A A fictitious biological pathogen spread by human contact (hollow arrows) and vectored by mosquitos (solid arrows); and **B** an infopathogen using an example of violence associated with Fascism and Neo-Nazis, spread by human contact (hollow arrows), and vectored by information technology (solid arrows).

exhibit behavior based on host-centered interventions is necessary. Incorporation of concepts suggested by this biological analogy into a meme diffusion framework (Spitzberg, 2014) could potentially lead to a model with explanatory or predictive power for forecasting changes in social epidemics or for testing the efficacy of potential interventions. Policy and legislation changes can influence the price and/or availability of many of the physical agents (e.g., guns, drugs, sugary beverages, and cigarettes) that are required for a social epidemic to occur.

Physical interventions. Engineering and technology can also impact social epidemics, for example, seat belts reduce injury in the case of collisions, filters reduce toxins ingested from cigarettes and safety locks reduce negligent discharge of guns resulting in

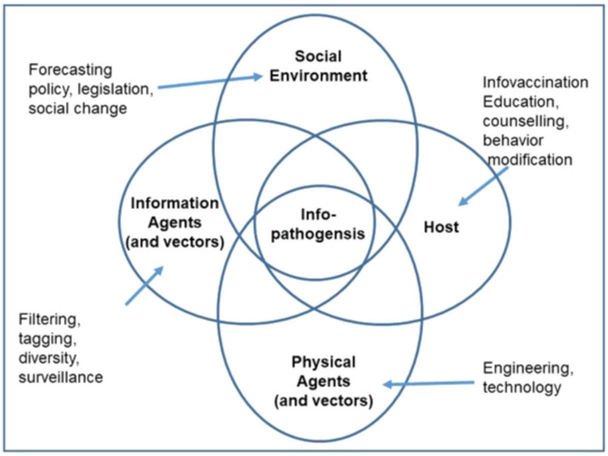

Fig. 3 Interventions for social epidemics suggested by the epidemiological quad. Potential interventions to reduce the spread of social epidemics are classified according to their impact on information agents, physical agents, the social environment, and susceptible hosts.

accidental injury. Importantly, establishing a causal role for information in the spread of social epidemics does not negate the need for the appropriate control and regulation of the physical agents that also cause social epidemics. However, it is important to make the distinction here, that in some cases physical agents can easily be substituted when controls are attempted that reduce their availability. In cultures where guns have been removed, other weapons such as knives may take their place to continue perpetration of violence when interventions on the physical agent is the only action taken (Squires, 2009) Complete prohibition of some items do not stop the epidemic, but rather result in a loss of control (alcohol prohibition in the United States, or the current war on drugs (Gray, 2010)).

Host interventions. Host interventions include infovaccination, education, counseling, and behavior modification. In human epidemiology, vaccination (the inoculation of uninfected individuals with a safe form of the pathogen that leads to immunity or resistance against a disease) is one of the most important tools to prevent epidemics (Plotkin and Plotkin, 2004). Evidence suggests that "infovaccination", more commonly known as cognitive immunity (Roozenbeek and van der Linden, 2019) or inoculation (Cook et al., 2017; Van der Linden et al., 2017) also works against infopathogens. The idea of inducing resistance to persuasion is not new and has been around since the 1960s (McGuire, 1964) A classic example might be anti-smoking advertisements that contain shocking and graphic images, designed to prevent youth from smoking (Wakefield et al., 2003). Another example is pre-emptive inoculation against misinformation about climate change (Cook et al., 2017; Van der Linden et al., 2017). These approaches may work well for a specific infopathogen but there could also be approaches that infer resistance to multiple infopathogens. For example, Roozenbeek and van der Linden (2019) developed an interactive online game to confer cognitive immunity through exposing participants to common types of misinformation.

Additional insights on the mitigation of infopathogens might also be gained from interventions used for the mitigations of viruses. For example, when immunizations for biological diseases are applied at large scale, or when large numbers of individuals recover, it can lead to a condition known as herd immunity where susceptible individuals are protected by the inability of the pathogen to spread because of a large proportion of resistant individuals (Anderson and May, 1985). The techniques used to develop new vaccines such as inactivation, encapsulation or genetic engineering (Plotkin and Plotkin, 2004) may provide insights for the development of info-vaccines. As has already been mentioned, other insights might also be gained from the studies of the spread of epidemics, especially those that study the immunization of complex networks (Pastor-Satorras and Vespignani, 2002).

Interventions can be used to encourage infected and exposed individuals to not express symptoms, for example by offering alternative role models to Adolf Hitler and alternative social events to right wing hate marches. Institutions and organizations may provide a key role in suppressing the negative impacts of infopathogens by providing positive information alternatives. Examples of host interventions include anti-drunk driving campaigns (Cismaru et al., 2009) and the "play 60" movement to reduce childhood obesity (Bai et al., 2015). Prochaska et al. (1992) identified five stages for quitting an addiction: pre-contemplation, contemplation, preparation, action and maintenance, stages that could be analogous to the SEIR model. It is known that at any one time in at-risk populations, 80% of the population are in the pre-contemplative or contemplative stages and interventions can be matched to the stage that individuals are in (Prochaska and Velicer, 1997).

A combination of intervention tactics are likely to be more successful than individual ones (McDavid et al., 2011). This has been exemplified numerous times in agro-ecological systems for example, in response to the development of resistance to pesticides that were initially highly efficacious, such as DDT (Forgash, 1984). This resistance led to the development of integrated pest management, which relies upon a combination of strategies, such as cultural practices, biological controls and monitoring, and is more sustainable in the long term (Kogan, 1998).

Discussion

In this discussion, we explore challenges to the theory of infopathogens. First, we demonstrate that the biological analog can explain the behavior of information spread in networks. Second, we discuss how human (or host) behavior influences the spread of both biological and social epidemics. Third, we discuss challenges to the theory in terms of the subjective nature of harm and the protection of the right to freedom of speech. Finally, we discuss the most important implication of the theory, the rehabilitation of infected individuals.

First, our theory can be falsifiable if the spread of harmful information through human and computer networks is not consistent with the biological analog of infectious diseases. For example, cognitive limits that restrict the spread of memes, has been seen as evidence that a meme is not a virus (Hodas and Lerman, 2014; Lerman, 2016). However, cognitive limits can be seen as analogous to the biological principle of host resistance. Some individuals are biologically resistant and do not transmit a pathogen while other susceptible individuals might be super-spreaders (Hawley and Altizer, 2011), and others fall in between these extremes of symptom expression. Another resistance related observation is that individuals that are exposed to a meme repeatedly are less likely to transmit it (Steeg et al., 2011). The biological analogy also explains the high transmission rates of memes by poorly connected individuals (Hodas and Lerman, 2014), analogous to the increased disease susceptibility observed in isolated populations, for example, tuberculosis (Hurtado et al., 2003). Likewise, while the spread of infectious agents (including information) can be explained by simple contagion, the spread of human behaviors (including those associated with social epidemics) are spread by complex contagion (Centola and Macy, 2007). This has been observed for the ice bucket challenge as well as for certain biological diseases associated with the failure to vaccinate (Campbell and Salathé, 2013).

Second, the importance of human behavior in the spread of social epidemics also suggests that a biological analog is not appropriate. For example, social media users actively seek out information and make a conscious decision to propagate it (Hodas and Lerman, 2014), sometimes with damaging consequences. However, the choices, knowledge, and behavior of individuals and groups also influences the rate of spread of biological pathogens (Tanaka et al., 2002). Some pathogens will also cause a change in behavior in their hosts, an example being hantaviruses in rodents (Klein et al., 2004). Biological pathogens are spread by vectors, which actively seek out hosts and hosts who actively seek out resources, such as water. A classic case of the later is the outbreak of cholera from the use of the Broad Street pump where individuals did not have the epidemiological knowledge of water borne diseases to inform their behavioral choices (Paneth, 2004). With the recent outbreak of COVID-19 social distancing is an important behavior for controlling the epidemic (Anderson et al., 2020), yet many individuals still chose to ignore these guidelines and some became infected because of this choice. Likewise, many individuals' behavioral choices are influenced by the information they consume, which can render them more vulnerable to social epidemics (Anderson et al., 2015; Howard, 2017; Huesmann et al., 2003; Hurley, 2004; Murphy, 2019; Saffer and Chaloupka, 2000). Furthermore, the media and information consumption choices made by individuals are likely heavily influenced by their social environment, an example being echo chambers and filter bubbles caused by ideological polarization (Spohr, 2017). Importantly, these choices are constrained by algorithmic ranking and evidence suggests that minimal nudging from a central node can change users' viewpoints (Perra and Rocha, 2019).

Another issue raised by the theory of infopathogens is that the classification of information as harmful can be quite subjective or open to debate. One of the best examples is video games; despite anecdotal evidence suggesting their harm, a widely cited recent study suggests there are no linkages to violent behavior (Markey et al., 2015) indicating a misapplication of a meme (video game violence) to an epidemic (physical violence). Something that is considered to be a social epidemic now, for example teen smoking, might once have been considered harmless. In biology the distinction between pathogenic and non-pathogenic microbes has not always been clear. Over a hundred and fifty years of microbiological research has helped us learn what microbes are beneficial and which ones are harmful. Many microbes are beneficial, including microbes involved in digestion and even some viruses are beneficial to humans by increasing host resistance to harmful bacteria (Roossinck, 2015). Recent controversies and the rise of the anti-vaccination movement (Kata, 2012) suggest that scientific evidence is not universally accepted by the public even regarding childhood diseases that have historically killed thousands. Unfortunately, our understanding of the harm and benefits of information have not kept up with the exponential spread of the internet. In the end, if the meme is, at current, perceived as being an infopathogen, the epidemiological infopathogen quad provides a framework for understanding and remedying the perceived epidemic.

Historically, society has relied upon the "marketplace of ideas" analogy, allowing for selection of good ideas and rejection of bad ones (Gordon, 1997), but this analogy relies upon a rational human mind, which may be overly optimistic (Ingber, 1984). The theory that the best ideas will win out clearly does not apply to the fields of advertising and entertainment where harmful and intellectually questionable content may be delivered if it results in more views or clicks (Hurley, 2004), nor with memes purposefully created and spread to elicit a certain behavior—propaganda and fake news, for example. The issue is not what information content is harmful but that more effective tools for protecting individuals from harm should be developed. This implicitly recognizes that content that may be harmful to one individual but not to another, since individuals likely vary in their susceptibility to infopathogens even more so than they do to biological pathogens. We implicitly recognize that the concept of an infopathogen does not negate the personal responsibility of individuals who ultimately make the choice to engage in behaviors that may be detrimental to themselves or others.

One concern presented by the concept of an infopathogen is that it could be used as excuse to enforce censorship and limit free speech. We argue that direct censorship is akin to biological eradications—a technique that is often difficult and somewhat rare as its success is unlikely. Like biological pathogens in the natural environment, infopathogens in the information economy are ubiquitous and difficult to eradicate. Once invasive pests and pathogens have become widely established, eradication attempts usually fail (Pluess et al., 2012). Similarly, attempts to limit or eradicate infopathogenic content of popular memes is not a productive strategy as new sources of an infopathogen can quickly appear from other online sources or an infopathogen can quickly mutate to a new form. A classic biological example of mutation pressure is herbicide-resistant weeds that appeared following widespread herbicide use in crops that were genetically engineered to be resistant to herbicide (Gould et al., 2018). Pressure also exists for content producers who will look for new ways to deliver or disguise their messages following censorship. Instead, multiple intervention strategies provide a more practical approach to limiting the harm caused by infopathogens.

Finally, one of the most important implications of this theory is that individuals who perpetuate social epidemics such as violent offenders should be treated as infected individuals. This idea is supported by the observation that repeat offending is socially and spatially contagious (Mennis and Harris, 2011). The idea of treating offenders as infected individuals does not negate the role of the criminal justice system, but suggests that interventions as part of rehabilitation can be deployed to break the epidemic cycle. Education practices and reducing social isolation in some prison systems such as Norway confirms this approach can be successful (Bhuller et al., 2020; Langelid, 1999). We also wish to be clear that the identification of a role for information in causing social epidemics does not diminish the importance of susceptible hosts, physical agents or social environments in any way. Any public health interventions should involve all these aspects in a systems approach.

Conclusions and next steps

In conclusion, we have developed a novel framework for improving the understanding and the mitigation of social epidemics:

1. Infopathogens: Information has been shown to be contagious (Jin et al., 2013; Kucharski, 2016; Tambuscio et al., 2015) and play a role in social epidemics (Christakis and Fowler, 2013). However, this paper is the first to clearly define the role information plays as causal agent in social epidemics by defining the term infopathogen. This definition can help resolve the scientific debate over the status of violence as a contagious disease (Greene, 2018; Slutkin et al., 2018b) by suggesting infopathogens as the missing causal agent (Greene, 2018).
2. The epidemiological quad: This paper is the first to expand the epidemiological triad to a quad by including information agents and vectors (information technologies and media). The epidemiological quad provides an improved tool for the macro-analysis of social epidemics, including the role of information and potential mitigations. In

particular, the epidemiological quad could provide guidance for the confusion over how violence should be approached in public health policy (Williams and Donnelly, 2014).

3. Infopathogenic life cycle: Another novel contribution of this paper is to compare the life cycles of biological pathogens and infopathogens. The use of life cycle diagrams and associated modeling is critical to understanding the spread of epidemics, including social epidemics. In particular, life cycle diagrams (or zoonotic cycle) have functioned as "pilots of human mastery" over biological epidemics (Lynteris, 2017), suggesting that these diagrams could also be critical for mastering social epidemics.

We recognize that more research is needed to continue to develop and test this framework. The utility of a framework for describing infopathogens will be verified if additional mitigations for social epidemics can be identified through the comparative analysis of social and biological epidemics, especially those caused by viruses. Two of the most promising areas for research are the development of vaccines (Plotkin and Plotkin, 2004) and the spread of epidemics through networks (Pastor-Satorras and Vespignani, 2002). A second test of the utility of the framework will be if the epidemiological quad proves to be useful as an educational, outreach and management tool for social epidemics. One approach is to combine the quad with the Public Health Approach as suggested by Lett et al. (2002) for the triad. The Public Health Approach is a methodology consisting of four hierarchical levels; surveillance, risk factor identification, intervention evaluation and program implementation. The utility of the quad can be tested by identifying the contribution of information agents to the spread of epidemics and their potential mitigations in each of these four levels.

Finally, in developed countries, we believe that the impact of infopathogens on social stability will become one of the most important issues for the twenty-first century. This is because the intended and unintended consequences of harmful information on human behavior will become increasingly harder to mitigate, even as our information technologies increasingly regulate and control other aspects of the social environment. In addition, the efficiency and diversity of the internet and social media makes it possible for just a single infected individual to transmit infopathogens to large numbers of people. Human society may become more vulnerable to infopathogenic content as people increasingly rely on intelligent decision support systems and less on individual common sense and judgment (for example, overreliance on instructions from a faulty GPS system). Moreover, our own human judgment may spur us to share lies at a higher rate than truth (Vosoughi et al., 2018) increasing the potential for the spread of harmful information. We hope this article will spur cooperation between the humanities and sciences to study the influence of harmful information on the spread of social epidemics.

Data availability

No data was generated for this paper.

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Competing interests

The authors declare no competing interests.

Additional information

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