# DM Kant Aff

## FW

#### I affirm the resolution. Resolved: In a democracy, a free press ought to prioritize objectivity over advocacy.

Emotions Or, xx-xx-xxxx, "Objectivity," Vocabulary, <https://www.vocabulary.com/dictionary/objectivity> //Memorial Ma

#### Vocabulary.com defines objectivity as - Objectivity is a noun that means a lack of bias, judgment, or prejudice.

#### Morality is a derived a priori -

#### 1)External Worlds Fallacy- only internal knowledge can be trusted. All external experience is corrupted- we could be dreaming, hallucinating, or being tortured by a demon. Therefore, it shows us what we perceive as existing, not what actually is existing. Thus, knowledge must be derived internally for a moral theory.

#### 2) Constitutivism- empirical knowledge is gained through experience. Experience isn’t constitutive to a subject, it comes from a posteriori knowledge through observation and isn’t part of a subject.

#### 3)Is Ought Fallacy- experience only tells us what is since we can only perceive what is, not what ought to be. But it’s impossible to derive an ought from descriptive premises, so there needs to be additional a priori premises to make a moral theory.

#### 4)Uncertainty- inability to know others’ experience make empiricism unreliable for ethics. Outweighs since it would be escapable since people could say they don’t experience the same. Hijacks roles of the ballot since only a solid basis is enough to convince people to follow their theory.

#### This means practical reason-

#### 1)Constitutivism- the only thing constitutive to subjectivity is reason. I could be a brain in a vat but so all other forms of knowledge are unreliable and doubtful. The only thing I know is that I am doubting and reasoning. Thus, the only thing constitutive is reason. Anything else could be an illusion.

#### 2) Infinite Regress- We can ask why for any other framework but to ask why for reasons concedes the authority of reasons which means they are inescapable and binding. And, bindingness outweighs because binding theories are the only ones that can guide action which is the only purpose of ethics.

#### 3)Action Theory- any action can be divided into infinite parts. Any other theory is incoherent because there are infinite ends to look to. Prefer reason because it’s the only thing unifying all those actions.

#### That necessitates universalizable ends - it doesn’t make sense to say 2+2=4 for me but not for anyone else. This necessitates universal maxims- individuals can’t be exempt from maxims that apply to others.

#### Thus the standard is consistency with universalizable maxims

#### Prefer Additionally

#### 1] Performativity: when you enter debate, you presume that you will be free to set and pursue ends in the round because of a system of reciprocally enforced constraints. This means denial of my framework is impossible and all objections should be ignored on face because responding to my framework requires my framework to do so.

#### 2] Ethical frameworks are topicality interpretations of the word ought so they must be theoretically justified. Prefer on resource disparities—focusing on evidence and statistics privileges debaters with the most preround prep excluding lone-wolfs who lack huge evidence files. A debater under my framework can easily be won without any prep since minimal evidence is required. That controls the internal link to other voters because a pre-req to debating is access to the activity.

#### 3] Isolating unconditional worth within the other is uniquely liberatory and the basis from which other theories begin, so my offense turns and outweighs yours.

(solves for Kritiks bc it says everyone is a practical reasoner so we treat everyone equally, meaning we don’t violate K)

Farr 02 [Arnold Farr(African American Professor of philosophy at University of Kentucky, focusing on German idealism, philosophy of race, postmodernism, psychoanalysis, and liberation philosophy). “Can a Philosophy of Race Afford to Abandon the Kantian Categorical Imperative?” JOURNAL of SOCIAL PHILOSOPHY. Vol. 33, No. 1. Spring 2002.]

Whereas most criticisms are aimed at the formulation of universal law and the formula of autonomy, our analysis here will focus on the formula of an end in itself and the formula of the kingdom of ends, since we have already addressed the problem of universality. The latter will be discussed ﬁrst. At issue here is what Kant means by “kingdom of ends.” Kant writes: “By ‘kingdom’ I understand a systematic union of different rational beings through common laws.”32 The above passage indicates that Kant recognizes different, perhaps different kinds, of rational beings; however, the problem for most critics of Kant lies in the assumption that Kant suggests that the “kingdom of ends” requires that we abstract from personal differences and content of private ends. The Kantian conception of rational beings requires such an abstraction. Some feminists and philosophers of race have found this abstract notion of rational beings problematic because they take it to mean that rationality is necessarily white, male, and European.33 Hence, the systematic union of rational beings can mean only the systematic union of white, European males. I ﬁnd this interpretation of Kant’s moral theory quite puzzling. Surely another interpretation is available. That is, the implication that in Kant’s philosophy, rationality can only apply to white, European males does not seem to be the only alternative. The problem seems to lie in the requirement of abstraction. There are two ways of looking at the abstraction requirement that I think are faithful to Kant’s text and that overcome the criticisms of this requirement. First, the abstraction requirement may be best understood as a demand for intersubjectivity or recognition. Second, it may be understood as an attempt to avoid ethical egoism in determining maxims for our actions. It is unfortunate that Kant never worked out a theory of intersubjectivity, as did his successors Fichte and Hegel. However, this is not to say that there is not in Kant’s philosophy a tacit theory of intersubjectivity or recognition. The abstraction requirement simply demands that in the midst of our concrete differences we recognize ourselves in the other and the other in ourselves. That is, we recognize in others the humanity that we have in common. Recognition of our common humanity is at the same time recognition of rationality in the other. We recognize in the other the capacity for selfdetermination and the capacity to legislate for a kingdom of ends. This brings us to the second interpretation of the abstraction requirement. To avoid ethical egoism one must abstract from (think beyond) one’s own personal interest and subjective maxims. That is, the categorical imperative requires that I recognize that I am a member of the realm of rational beings. Hence, I organize my maxims in consideration of other rational beings. Under such a principle other people cannot be treated merely as a means for my end but must be treated as ends in themselves. The merit of the categorical imperative for a philosophy of race is that it contravenes racist ideology to the extent that racist ideology is based on the use of persons of a different race as a means to an end rather than as ends in themselves. Embedded in the formulation of an end in itself and the formula of the kingdom of ends is the recognition of the common hope for humanity. That is, maxims ought to be chosen on the basis of an ideal, a hope for the amelioration of humanity. This ideal or ethical commonwealth (as Kant calls it in the Religion) is the kingdom of ends.34 Although the merits of Kant’s moral theory may be recognizable at this point, we are still in a bit of a bind. It still seems problematic that the moral theory of a racist is essentially an antiracist theory. Further, what shall we do with Henry Louis Gates’s suggestion that we use the Observations on the Feeling of the Beautiful and Sublime to deconstruct the Grounding? What I have tried to suggest is that instead of abandoning the categorical imperative we should attempt to deepen our understanding of it and its place in Kant’s critical philosophy. A deeper reading of the Grounding and Kant’s philosophy in general may produce the deconstruction35 suggested by Gates. However, a text is not necessarily deconstructed by reading it against another. Texts often deconstruct themselves if read properly. To be sure, the best way to understand a text is to read it in context. Hence, if the Grounding is read within the context of the critical philosophy, the tools for a deconstruction of the text are provided by its context and the tensions within the text. Gates is right to suggest that the Grounding must be deconstructed. However, this deconstruction requires much more than reading the Observations on the Feeling of the Beautiful and Sublime against the Grounding. It requires a complete engagement with the critical philosophy. Such an engagement discloses some of Kant’s very signiﬁcant claims about humanity and the practical role of reason. With this disclosure, deconstruction of the Grounding can begin. What deconstruction will reveal is not necessarily the inconsistency of Kant’s moral philosophy or the racist or sexist nature of the categorical imperative, but rather, it will disclose the disunity between Kant’s theory and his own feelings about blacks and women. Although the theory is consistent and emancipatory and should apply to all persons, Kant the man has his own personal and moral problems. Although Kant’s attitude toward people of African descent was deplorable, it would be equally deplorable to reject the categorical imperative without ﬁrst exploring its emancipatory potential.

#### 4] Ideal Theory Good – a] end point – we’d constantly be fixing injustices as a precondition to ethical action so we never get to the bottom of what is actually ethical b] relevance – every society has different injustices that occur – the resolution is a universal values statement which means you cannot universalize any theory under nonideal theory.

## 1AC – Offense

#### 1] Non-objective reporting is a form of lying.

Michael **Koliska** and Linda **Steiner, 15** [Michael Koliska, (Dr. Michael Koliska is an assistant professor in the Communication, Culture, and Technology master’s program at Georgetown University. His primary research focuses on the practices, performances and effects of authenticity, accountability and transparency on trust in both “traditional” and computational journalism. Specifically, he explores how technology alters the newsroom sociology such as production and accountability processes that influence public perception of journalism’s legitimacy. Dr. Koliska’s work on these issues has appeared in the International Journal of Communication, Journalism Studies, Journalism Practice, Digital Journalism, Journalism, Journal of Media Ethics and others.) Linda Steiner, (Linda Claire Steiner is a professor at Philip Merrill College of Journalism, University of Maryland. She is also the editor-in-chief of the journal Journalism & Communication Monographs, and sits on the editorial board of Critical Studies in Media Communication.)]. " TRANSPARENCY AND TRUST IN JOURNALISM: AN EXAMINATION OF VALUES, PRACTICES AND EFFECTS " Accessed 3-1-2022. https://drum.lib.umd.edu/bitstream/handle/1903/17031/Koliska\_umd\_0117E\_16478.pdf?sequence=1 //duongie + Xu

More responsibility toward the public and a climate “of public hostility to the media” led to a reevaluation of the media codes of ethics (including the movie, radio and television industry) between the 1930s and early 1950s (Siebert et al., 1956, p. 86). For the news media this meant specifically the development of accurate and objective reporting. The Hutchins Commission (CFP, 1947) suggested that for a free, democratic society “the first requirement is that the media should be accurate” (p. 21). The commission stressed the fact that media “should not lie” and that it is the media’s responsibility to provide the context of facts, without which it may be misleading or untrue (p. 21). According to the Commission: “It is no longer enough to report the fact truthfully. It is now necessary to report the truth about the fact” (p. 22). In that respect, accuracy in reporting arose to be central to the self-regulation efforts of news media organizations in order for them to be publicly accountable.

#### That’s immoral – 2 warrants.

MCAE ND [MARKKULA CENTER FOR APPLIED ETHICS (explores ethical issues in corporate governance, global business, leadership, executive compensation, and other areas of business ethics at Santa Clara University). “Lying”. No Date. Accessed 3/1/2022. <https://www.scu.edu/ethics/ethics-resources/ethical-decision-making/lying/#:~:text=The%20philosopher%20Immanuel%20Kant%20said,that%20he%20called%20human%20dignity>. //Xu]

The philosopher Immanuel Kant said that lying was always morally wrong. He argued that all persons are born with an "intrinsic worth" that he called human dignity. This dignity derives from the fact that humans are uniquely rational agents, capable of freely making their own decisions, setting their own goals, and guiding their conduct by reason. To be human, said Kant, is to have the rational power of free choice; to be ethical, he continued, is to respect that power in oneself and others. Lies are morally wrong, then, for two reasons. First, lying corrupts the most important quality of my being human: my ability to make free, rational choices. Each lie I tell contradicts the part of me that gives me moral worth. Second, my lies rob others of their freedom to choose rationally. When my lie leads people to decide other than they would had they known the truth, I have harmed their human dignity and autonomy. Kant believed that to value ourselves and others as ends instead of means, we have perfect duties (i.e., no exceptions) to avoid damaging, interfering with, or misusing the ability to make free decisions; in other words - no lying.

#### 2] Lexico ND defines advocacy[[1]](#footnote-1) as “Public support for or recommendation of a particular cause or policy” but that definitionally isn’t universalizable since it only cares about one cause and disrespects the ends of the agents that it disagrees with.

### 1AC: Pandemics- preempt to util

#### Advantage 2 is Pandemics:

#### Objective Media Coverage is key to combat Vaccine Disinformation BUT Advocacy creates polarization that hardens misinformation.

Sullivan 21 Margaret Sullivan 3-7-2021 "The media plays a crucial role in battling vaccine misinformation. But here’s what not to do." <https://www.washingtonpost.com/lifestyle/media/vaccine-misinformation-media/2021/03/05/fd01a0ba-7dbd-11eb-a976-c028a4215c78_story.html> (Education: Georgetown University; Northwestern University's Medill School of Journalism)//Elmer

There are all sorts of ways to counter reluctance to get the coronavirus vaccine. There’s leading by example. There’s guilt. And there’s pure charm. Dolly Parton went the latter route last week as she got her first shot, wearing a sparkly blue cold-shoulder dress for her Instagram PSA and crooning “Vaccine” to the tune of her signature “Jolene.” Anthony S. Fauci made an argument both moral and scientific, reflective of his Jesuit education. “Think about your societal obligation,” he told members of the military, about a third of whom reportedly don’t want the vaccine. He added: “Like it or not, you’re propagating this outbreak.” And Boston Marathon director Dave McGillivray chose to inspire, explaining to the Wall Street Journal how he took the logistics expertise he would have deployed for this year’s canceled race and reapplied it to organizing vaccinations in Massachusetts instead. Despite all this high-level persuasion, a big chunk of Americans — about 3 in 10 — remain hesitant, according to a new Pew Research survey. And like Parton, Fauci and McGillivray, the news media has a role to play — not in outright advocacy, but in relentlessly providing accurate, nuanced information and answering questions straightforwardly. “There is a lot to be said for honestly reporting as much context as possible and knowing the terrain into which your sound bites and headlines will play,” said Emily Bell, director of the Tow Center for Digital Journalism at Columbia University. Although Bell is eager to see more people move past their concerns and get the vaccine, she told me she doesn’t believe in downplaying the numbers on negative reactions to shots: “All you are doing is reinforcing the narrative of the ‘wellness bloggers’ that Big Pharma is hiding something.” And what journalists shouldn’t concentrate on, according to one misinformation expert I talked to, is spending too much energy debunking myths. Some of the most popular myths: That tech mogul Bill Gates is secretly implanting microchips in people’s arms. That the vaccine causes the disease. That there are toxic levels of mercury in the doses. That flu shots protect against covid-19, so the newer vaccine is unnecessary. But even though such notions are incorrect and damagingly so, “the media should not be playing Whack-a-Mole by debunking every obscure rumor,” said Claire Wardle, founder of First Draft, a nonprofit that fights online misinformation. “The more you say some outrageous thing is not true — ‘No, Bill Gates is not microchipping you!’ — the more you give people the key words” that will send them down the social media rabbit hole of misinformation, she told me. “You’re giving it oxygen.” Instead, like Bell, she believes it’s all about relentlessly educating the public by answering reasonable questions with as much expertise as can be mustered. Local reporters — who tend to be relatively well-trusted — are especially important in this effort, providing basic information, and pointing readers or viewers to credible public-health sources. Sadly, there are far fewer of these reporters than when the pandemic began. At their best, local news organizations also provide important watchdog coverage, as the Boston Globe did Friday in an investigative report about Massachusetts Gov. Charlie Baker’s (R) administration disastrously pivoting to privatize vaccine distribution, with private entities awarded no-bid contracts “to undertake perhaps one of the state’s most pressing, ambitious initiatives in modern times.” The media’s performance, to date, has been far from perfect. Early on, the overemphasis of allergic reactions — without enough context — set a bad standard. And some experts think the media coverage has been too pessimistic overall. “The public has been offered a lot of misguided fretting over new virus variants, subjected to misleading debates about the inferiority of certain vaccines, and presented with long lists of things vaccinated people still cannot do, while media outlets wonder whether the pandemic will ever end,” sociologist Zeynep Tufekci wrote in the Atlantic. The joy of vax: The people giving the shots are seeing hope, and it’s contagious Still, there’s evidence that some people are changing their minds. The number of those who don’t intend to get the vaccine has come down from about 40 percent a few months ago to about 30 percent now, according to the new Pew numbers. Vaccine coverage still has room for improvement. “What the public needs to hear,” Tufekci wrote, “. . . is that based on existing data, we expect them to work fairly well — but we’ll learn more about precisely how effective they’ll be over time, and that tweaks may make them even better.” Before last year’s election, the reality-based media — to its everlasting credit — got across the idea that election night probably wouldn’t provide the answer to who won the presidency, that it might take weeks to count the vote. The media succeeded by repeating this message over many weeks, basing their accounts on credible experts, and warning about misinformation campaigns. When the pandemic-hampered vote count did indeed take several days, most news consumers were prepared to recognize this as acceptable, and far less likely to buy into the lie that the election had been stolen. Call it a victory, rare enough these days, for good information over bad. Vaccine coverage — with its life-or-death implications — is even more consequential. We need to get it right.

#### Credible News Distribution is key to vaccine adoption – it’s the only way to end Pandemics.

Harmon 21 Gerald Harmon 9-27-2021 "Defeating misinformation is key in ending the pandemic" <https://www.ama-assn.org/about/leadership/defeating-misinformation-key-ending-pandemic> (Gerald E. Harmon, MD, a family medicine specialist having practiced for more than 30 years in coastal South Carolina, became 176th president of the American Medical Association in June 2021. He was first elected to the AMA Board of Trustees in June 2013 and elected board chair in 2018. In addition, Dr. Harmon also served as the secretary of the AMA in 2016.)//Elmer

As we confront yet another major surge in COVID-19 cases and hospitalizations across the country, we are once more fighting a two-pronged war: against the virus and against rampant misinformation. The evidence around vaccination is abundantly clear. Vaccines are by far the best way for your patients to protect themselves and their loved ones from severe complications of COVID-19. But you wouldn’t know it if you were a regular viewer of some popular TV networks, or received your news from agenda-driven websites that traffic in half-truths and outright lies about the virus. Whatever their reasons, the result of this misinformation crusade is doubt, confusion and division at a time when our public response to this pandemic must be unified and resolute. This sobering reality has been made clear by the Centers for Disease Control and Prevention: Roughly 99% of deaths linked to COVID-19 in this wave—and the vast majority of those with severe symptoms that require hospitalization—have come among patients who were not fully vaccinated. The Food and Drug Administration’s recent approval of the Pfizer-BioNTech vaccine against COVID-19 is not only a landmark event in science and medicine; it is an opportunity to set the record straight. Vaccines for COVID-19 are safe. They are effective. And they are our best chance to bring this pandemic to an end. But vaccines alone won’t save us. Now, more than ever before, the public needs honest and clear communication about the importance of vaccines, vaccine science, and the crucial role they have in protecting public health. Obligations of responsible media Entities of public trust in society play an important role as credible sources for information at all times, but particularly during a public health crisis. Given their reach and influence, news organizations carry tremendous responsibility. They must help viewers and readers separate the facts from fiction, and proven treatments from potentially dangerous poisons. As physicians, and in an effort to ease the tremendous pressure on our nation’s health system, the AMA urges the cooperation of media outlets—TV, print and online—to tell the truth about the safety and efficacy of these COVID-19 vaccines, the rigorous research and review process behind them, and to be voices for science and evidence for their audiences. Reporting on unproven and potentially dangerous treatments for this virus, including ivermectin, hydroxychloroquine and other treatments that have not been scientifically validated, confuses the public and puts lives at even greater risk. As fall proceeds, the ongoing tragedy of the COVID-19 pandemic in our country is only intensified by the fact that science has given us the means to bring this dark chapter to a close. Vaccination is our only way out this pandemic—but that exit will remain blocked until the vast majority of those who are eligible to receive the vaccination do so. It is clear that some media outlets and personalities continue to foster hesitancy and resistance to COVID-19 vaccinations by framing the issue solely in terms of infringement upon civil liberties or personal freedom, and those voices that are then amplified through social media and other online channels.

#### Best studies conclude aff – misinformation independently causes disease spread, but “immunization” against “fake news” solves.

Brainard & Hunter ’19 [Julii Brainard – Dr, Senior Research Associate, Norwich Medical School Honorary Research Fellow, Norwich Medical School Member, Epidemiology and Public Health Member, Public Health and Health Services Research, Paul Hunter - Professor in Medicine, Norwich Medical School Member, Water Security Research Centre Member, Epidemiology and Public Health Member, Public Health and Health Services Research, “Misinformation making a disease outbreak worse: outcomes compared for influenza, monkeypox, and norovirus”, 11-12-2019, https://journals.sagepub.com/doi/full/10.1177/0037549719885021]//pranav

No previous studies have integrated information spread with disease spread to the level of sophistication that we have done. Prior models often considered information spread in disease outbreak development, but information awareness was typically equally available to all agents, and benign at worst. Thus, information spread in the models nearly always led to greater protective measures (such as increasing vaccine uptake or decreasing contact rates41–50). Most previous similar disease and awareness spread models had awareness increases that could only happen following physical contact or as a result of global conditions.42,45,48,50–55 Our modeling is unusual because information spread was individual and separated from the physical interactions that could transmit disease. Our model is unique and original in attempting to consider the potentially deleterious role of information sharing with stochastic and individually assigned elements. The need for research such as ours has been recognized before.17,56 More sophisticated information sharing networks than we tried to create could make these models more credible. There exist more sophisticated models on rumor spread that we could possibly replicate for the information spreading process,57–59 and simultaneously merge with existing sophisticated disease spread models. More ambitious models than ours would describe more agents and more complicated movement patterns, such as including flight as a behavior option. Many rumor spreading models have borrowed ideas and methods from epidemiological models,60,61 but not many (if any) previous models have integrated both rumor and disease spread as separate but interacting processes into one unified probabilistic model. This study describes the spread of three viral diseases; misinformation affecting the spread of bacterial diseases could be modeled equally well. The ideas could be applied to non-communicable diseases and health outcomes, but it would be necessary to change the time scale to be much longer to model chronic and lifestyle diseases and how their incidence might change in response to circulating misinformation. A much longer time scale would mean incorporating many other lifestyle factors into the models. Model construction relied heavily on a small number of existing studies about such factors as number of contact rates, social contacts (i.e.., Dunbar numbers), how much bad or good advice can change behavior, and the propensity to believe in misinformation (the finding that on average, British people believe in 38.9% of conspiracy theories that they are exposed to). More reliably estimating any of these and many of the other factors would also increase the credibility of our results. Our threshold for a “worse” outbreak situation was r0 being 40% worse or the number of generations of disease transmission increased from 4 to 7; these thresholds were decided for convenience in this set of demonstration models. Given our definition of stage 2 as an outbreak “made worse by circulating misinformation,” stage 3.1 modeling concluded for all three diseases that a ratio of about 60:40 good:bad advice circulating would reduce the stage 2 conditions to those of stage 1. The models also suggested that “immunizing” about 20% of the population against misinformation was likely to revert stage 2 to stage 1 conditions (for all diseases, stage 3.3). Since these apparent consistencies could be artefacts of shared model design, tests to explore the true consistency of these findings for multiple diseases would be worthwhile. It is possible that more sophisticated, detailed, or larger models or more flexible modeling software62 would facilitate better insights into risk distributions and behavior choices. There is uncertainty in the reliability of these findings because the models are experimental and have not been tested in real world situations. There is a general lack of reliable quantification for how much misinformation spread impacts real life risk-taking behavior with regard to communicable diseases. 5. Conclusions We applied three stages of modeling (1 = no misinformation spread, 2 = misinformation making outbreaks worse, and 3 = strategies to reduce the influence of misinformation). Our modeling approach and design is adaptable to many different types of diseases. Controlling spread of misinformation or susceptibility to it could reduce communicable disease burdens. Our stage 3.1 modeling found that a ratio of about 60:40 good:bad circulating advice reduced stage 2 conditions to those of stage 1 in three types of disease. “Immunizing” about 20% of the population against misinformation (stage 3.3) was likely to revert stage 2 to stage 1 conditions (for all diseases). The feasibility of implementing these types of strategies (“immunization” or changing the proportions of types of advice in circulation) should be explored. The efficacy of implementing such strategies to fight “fake news” needs to be tested in real world settings, with costs and benefits ideally compared with real world disease reduction.

#### Pandemics risk extinction - simulations, empirics, and surging connectivity prove.

Kim 21, Kiseong, et al. "Network Analysis to Identify the Risk of Epidemic Spreading." Applied Sciences 11.7 (2021): 2997. (Department of Bio and Brain Engineering, KAIST; R&D Center)//Re-cut by Elmer

Several epidemics, such as the Black Death and the Spanish flu, have threatened human life throughout history; however, it is unclear if humans will remain safe from the sudden and fast spread of epidemic diseases. Moreover, the transmission characteristics of epidemics remain undiscovered. In this study, we present the results of an epidemic simulation experiment revealing the relationship between epidemic parameters and pandemic risk. To analyze the time-dependent risk and impact of epidemics, we considered two parameters for infectious diseases: the recovery time from infection and the transmission rate of the disease. Based on the epidemic simulation, we identified two important aspects of human safety with regard to the threat of a pandemic. First, humans should be safe if the fatality rate is below 100%. Second, even when the fatality rate is 100%, humans would be safe if the average degree of human social networks is below a threshold value. Nevertheless, certain diseases can potentially infect all nodes in the human social networks, and these diseases cause a pandemic when the average degree is larger than the threshold value. These results indicated that certain infectious diseases lead to human extinction and can be prevented by minimizing human contact. 1. Introduction The emergence of a pandemic is one of the various scenarios frequently discussed as a human extinction event, and it is listed as one of the global catastrophic risks in studies regarding the future [1,2,3]. In particular, several pandemics, such as the Black Death [4,5], Spanish flu [6], and those caused by smallpox [7], severe acute respiratory syndrome (SARS) [8], and Ebola [9], have affected a large population throughout history. The risk of pandemics increases with an increase in population mobility between cities, nations, and continents, thereby threatening humankind [10,11,12]. It is essential to analyze the epidemic spread in society to minimize the damage from epidemic disasters; however, extinctive epidemic spreading experiments have limitations in real-world situations, as they predict stochastic effects on the spread without considering the structure of human society. Network-based approaches have been proposed to overcome these limitations and perform epidemic spreading simulations by considering the network structure of numerous real-world connections [13,14,15]. These methods use various models of epidemic spreading, such as the susceptible–infectious–susceptible (SIS) [16,17,18], susceptible–infectious–recovered (SIR) [19,20,21], and Watts threshold models [22]. While these methods are mathematically convenient, they are epidemiologically unrealistic for various infections because they require exponentially distributed incubation and infectious periods [23,24,25]. Moreover, previous epidemic studies did not perform quantitative assessment of the pandemic risk depending on the network connectivity in individuals and fatality rate of various diseases [26]. In the present study, we applied an SIR epidemic model to a scale-free network with Monte Carlo simulation to identify the quantitative relationship between infectious diseases and human existence. Our fundamental hypothesis states that when the epidemic spreads to all nodes of the network and the fatality rate is 100%, it can increase the pandemic risk. To address this, we initially constructed a scale-free network to simulate a society. Moreover, for the epidemic spreading simulation, an SIR model was applied to the network to describe the immune state of an individual after infection. From the simulation study, we found that the mean degree of a scale-free network was an essential factor in determining whether epidemics threaten humans. This approach provides important insights into epidemic spreading analysis by investigating the relationship between epidemic and scale-free network parameters. Furthermore, it highlights the necessity of determining information flow during an epidemic. 2. Materials and Methods We designed an epidemic simulation process to identify the relationship between pandemic risk and network parameters. This study was performed in four steps (Figure 1): (i) generating a scale-free network model to reflect real-world conditions; (ii) applying an SIR model to the scale-free network for epidemic spreading simulations; (iii) adapting the Monte Carlo method to reflect the stochastic process in the node status of the SIR model; and (iv) iteratively performing simulation for every parameter set and analyzing the results. We have provided the source code and sample results of epidemic simulation in Supplementary Materials. Figure 1. Overview of epidemic simulation process based on the Monte Carlo method. (A) We generated scale-free networks for a fixed population (N = 1,000,000) and various node degrees (k = 2, 5, 7, and 10). (B) Epidemic spreading was simulated by applying a susceptible–infectious–recovered (SIR) model to the scale-free network. We set the epidemic parameters, β and γd. β represents the spreading rate of epidemics, and γd is the reciprocal of γ and reflects the time interval between infection and recovery. Randomly, 0.05% of nodes were initially infected. (C) We adapted the Monte Carlo method to determine the status of the transition from the infection node to immunization node. Repeated simulations were performed until a steady state was achieved. (D) For every parameter set, 10,000 simulations were performed. 2.1. Network Generation Based on a Scale-Free Model We constructed a network model for the epidemic spreading simulation (Figure 1). The nodes and edges of the network represent people in the society and their physical contacts, respectively. We used a scale-free network model, which follows the preferential attachment property observed in numerous real-world networks, such as social networks, physical systems, and economic networks [27,28,29]. In the scale-free network, when a node is added to the network, its likelihood of connecting to existing nodes increases with an increase in the node’s degree. Hub nodes, which lead to fast and vast spreading of epidemics, exist. Two characteristic parameters, including N and k, affect the form of scale-free networks. The parameter N denotes all nodes in the network. In the real world, N indicates the whole population size. The parameter k is the average degree of the network, which determines the degree of the newly attached node for each step during network generation. Following the characteristics of the network model, we generated scale-free networks representing human contacts for epidemic spread. The scale-free network was generated by the Barabasi–Albert graph distribution, in which the network is constructed from a cycle graph with three vertices, followed by the addition of k edges at each construction step [30]. The k edges are randomly attached to the vertex based on the degree distribution of the vertex. After network generation, we investigated the degree distribution properties of the network (Figure 2). The results indicate that the degree distributions have similar tendency for networks with varying number of nodes and edges. This study constructed scale-free networks with the largest number of nodes considering computational complexity (N = 1,000,000). Figure 2. Degree distribution of the scale-free network. We analyzed the degree distribution of the network based on the number of nodes (N) and mean degree (k). 2.2. Epidemic Spreading Based on the SIR Model For the epidemic spreading simulations, we applied an SIR model to the generated scale-free network. The classical SIR model can be expressed by the following nonlinear differential equations [21]: where S, I, and R represent susceptible, infected, and recovered compartments, respectively, in the whole population. S represents people who have not been infected yet but can be infected in future. I represents infected people who can spread the epidemic to susceptible people through physical contact. R denotes people who have recovered or died from the epidemic and who no longer participate in the epidemic spreading process. The sum of the S, I, and R values represents the whole population size N. Epidemics have two parameters in the SIR model, transmission rate (β) and recovery rate (γ), which arise from the basic reproduction number R0 (Figure 1B). The basic reproduction number is the number of infections caused by one infective node [31,32,33]. If the R0 is more than 1, the infection can spread in a population, whereas if R0 is less than 1, the infection cannot spread. We express the basic reproduction number as R0 = β/γ, where β represents the spreading rate of epidemics between infective nodes and adjacent susceptible nodes and γ represents the probability of recovery from infection [34]. We mainly used γd, which is the reciprocal of γ and reflects the time interval between infection and recovery. 2.3. Investigation of Epidemic Status Based on the Monte Carlo Method The epidemic simulation was performed for a time series event by constructing epidemic status matrix (z) to represent the status of the nth node at time step t. For each node, the value of epidemic status matrix at time step t can be 0, 1, or 2, indicating that a node is susceptible, infective, or recovered, respectively. We initially (t = 0) set every value of epidemic status matrix to 0 because all nodes are susceptible before the epidemic spreads. At the initial infection stage, randomly selected 0.05% of nodes were infected. At every time period, we performed immunization and observed the infection stages (Figure 3). At the immunization stage, we identified infective nodes and determined whether these nodes would be recovered in the next time step. To calculate the transition probability of infected and recovered phenomena, the Monte Carlo method was applied [35,36]. When infection and recovery parameters are provided, it is possible to investigate whether a node transitions from an epidemic state to another state. To accomplish this, we compared the method revealing the change in each population in every compartment over time (Figure 4). The final steady state of the epidemic spreading simulation model indicates the total number of casualties of the epidemic who either are dead or have recovered from the disease. Infective nodes at time t (zn [t] = 1) are transformed to recovered nodes at time t + 1 (zn [t + 1] = 2) when 1/γd is larger than a random real number between 0 and 1. We determined whether the neighbor nodes of the infection node would be infected by identifying susceptible nodes adjacent to the infective nodes at time t (zn [t] = 0, with the adjacent infective node) (Figure 5). When β is larger than a random real number between 0 and 1, a susceptible node becomes an infective node at time t + 1 (zn [t + 1] = 1); this scenario represents epidemic spread. For each time step, we recorded the number of susceptible, infective, and recovered nodes during epidemic spread. 2.4. Simulation Parameters We carried out simulation trials for various mean degrees of networks (k = 2, 5, 7, and 10). Each network considered the following epidemic parameters: β ranges from 0.05 to 0.95 and γd ranges from 1 to 10. The Monte Carlo model was repeatedly simulated to observe saturation of the recovery process. Considering that the simulation pipeline contains random processes such as initial infection and Monte Carlo trials, we performed the simulation iteratively until the status of nodes remained unchanged. After simulation, time series data from every simulation were interpolated in the time domain. The fatality rate determines the ratio of deceased and recovered individuals in the final population [37,38,39]. If the fatality rate is below 100%, the recovered population contains both dead and recovered individuals. Such a situation does not always cause a pandemic. In this simulation, we assumed a 100% fatality rate. To accomplish this, we enumerated the recovered nodes as dead for considering the pandemic risk. 3. Results Through our method, we obtained epidemic spreading data with various network and epidemic parameter sets. In the present study, we focused on the case where the epidemic infects all nodes and defined this phenomenon as “extinctive spread”. Diseases causing extinctive spread are potential candidates of high pandemic risk. In the real world, extinctive spreading indicates that the disease will infect every person in the society. From the simulation data, we calculated the extinctive spread score by dividing the total number of simulation trials by the number of extinctive spread cases. Thereafter, we identified that the number of extinctive spread cases is mainly influenced by spreading speed, which is determined by β, γd, and k (Figure 6). The extinctive spread region (brown area in Figure 6) is expanded as the value of mean degree of network (k) is increased, thereby indicating that the area of extinctive spread becomes noticeably wider in a dense network than in a sparse network. Thus, the more contact between people, the higher the risk of epidemics. Moreover, high γd and high β cause extinctive spread across a large region, indicating that the high spreading rate and short time interval between infection and recovery are risk factors of epidemic diseases. In contrast, the infective nodes recover before they transmit the disease to their neighbors in low β and low γd scenarios, thus disconnecting the network and preventing extinctive spread. This occurs because the infective nodes need more time to transmit the disease in low β and high γd scenarios. Therefore, the disease begins to subside due to a lack of new infective nodes. Furthermore, we investigated the range of β and γd for existing epidemics of the common cold [40,41] and fatal diseases, namely, cholera [42,43], Marburg [44,45], Ebola (Congo and Uganda) [46,47,48,49], SARS [50], and MERS [51] (Table 1). We selected diseases with relatively well-known epidemic parameters, such as average duration of infection and basic number of reproductions from previous studies. Transmission rates were calculated using the mean duration of infectious periods and basic reproduction numbers of the epidemics. Different studies reveal multiple values of infectious period and transmission rate for some of these diseases; we considered these values separately [40,41,42,43,46,47,48,49]. For example, the infectious period of a common cold is from 3 to 7 days and that of Ebola is 6.5 days. Next, we placed the possible regions of these epidemics as a disease band for various k values (colored lines in Figure 6). When k > 5, fatal diseases have an opportunity to cause a pandemic. Even when k = 5, diseases such as cholera and Ebola (Congo) can be threatening in regions of low γd and high, thus demonstrating that the knowledge of network parameters of the society and the characteristics of epidemic diseases can aid in quantifying the risk of epidemics. 4. Discussion Many previous studies have made stochastic SIR models to analyze the dynamics or stability of epidemic diseases. They investigated the distribution of susceptible, infected, and removed populations for specific epidemic disease spreading, such as cholera, SARS, Marburg, and MERS, based on mathematical modelling [52,53,54,55]. However, they did not conduct a quantitative assessment of pandemic risk taking into account physical contact between people. To solve this limitation, we performed epidemic spreading simulations by applying an SIR model to scale-free networks with Monte Carlo simulation. In the simulation, we consider various connectivity and disease characteristics on scale-free networks. For each network and epidemic parameter set, the probability of extinctive spread was calculated. The results revealed that certain infectious diseases can lead to extinction. Moreover, even if the disease band extends over the extinctive spread regions, it does not indicate that human extinction results from the disease, as the fatality rate is below 100%; however, in the case of 100% fatality, the disease can cause a human extinction event. The risk of infectious disease is influenced by the network structure. A dense network has a higher risk of spreading infectious disease than a sparse network, as we observed in the extinctive spreading maps. According to our results, when the average degree of human social networks is below the risk threshold, i.e., less than 4 in this study, human society is safe from an extinctive outbreak based on our knowledge regarding the epidemic parameters of the infectious disease. Nevertheless, in other cases, human extinction is possible. For example, if the population is 1,000,000 and there are 4 or more instances of physical contact between people, human extinction events may occur, depending on the fatality rate of the epidemics. Hence, physical contact between people is closely related to an extinction event of infectious diseases. Eventually, from a public health perspective, lowering the average contact level of society is an appropriate way to increase the robustness of strategies against the occurrence of extinction. In the real world, reducing network density can be accomplished by epidemic prevention activity, such as isolation and quarantine treatment. This action prevents epidemic risk to the society, thereby avoiding human extinction. Additional considerations may improve our analysis. First, large population size and various proportions of initial infective nodes were not considered in the experiments. We have confirmed that the result was consistent when the proportion of initial infective nodes was 0.05% of the total population; however, this can vary depending on the distinct proportion of initial infective nodes in a different population. To achieve robust results, we need to perform additional experiments for various parameters; however, we could not address this issue due to computational complexity. Second, we did not consider numerous known epidemic diseases. We calculated the transmission rates of epidemic diseases using the known infectious periods and reproduction numbers of the epidemics from evidence in the literature. In the present study, we only considered five epidemic diseases, since the information on infectious periods and reproduction numbers of diseases was mostly unavailable for other epidemic diseases. Third, this study only considers the SIR model on scale-free networks in epidemic simulation. Since the dynamics of epidemic diseases can be varied in different models or networks, it is important to experiment in various simulation environments to confirm the robustness of the results. Nevertheless, these limitations can be considered in future experiments or using improved computational methods. With these further improvements, our approach can be used as a computational tool to analyze the risk of epidemic diseases. 5. Conclusions In this study, we analyzed the risk of epidemic diseases by creating an epidemic simulation on a scale-free network. Based on the simulation results for various epidemic parameters, we confirmed that certain infectious diseases can lead to extinction and can be prevented by minimizing human contact. We believe that identifying potential candidate diseases that may lead to human extinction is crucial in addressing epidemic prevention activities such as quarantine.

1. Lexico Dictionary (Lexico.com is a new collaboration between Dictionary.com and Oxford University Press (OUP) to help users worldwide with everyday language challenges. Lexico is powered by Oxford’s free English and Spanish dictionaries and features multi-language dictionary, thesaurus, and translation content). No Date. Accessed 3/3/2022. <https://www.lexico.com/en/definition/advocacy> //Xu] [↑](#footnote-ref-1)