TITLE
Centering human cognition in epidemiological models

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KEYWORDS
behavior, behavioral modeling, cognition, feedback, perceived risk, infectious disease, epidemiology

Viral infectious disease poses a threat to the health of individuals and the functioning of society. Projecting infection dynamics and assessing the efficacy of potential interventions is key to controlling the spread of disease and minimizing its impacts. Mathematical models of infectious disease, known as epidemiological models (EMs), are an important tool for projecting the spread of infectious disease and for examining the efficacy of proposed interventions. EMs have

traditionally assumed that individuals occupy a small set of states with respect to disease, e.g. susceptible to disease (S), infectious (I), or recovered (R), with transitions between states governed by treating individuals as gas-like particles colliding with each other—this forms the foundation of the classic SIR model. Variations on the SIR model have introduced additional states to account for specific disease stages or interventions, for example, asymptomatic stages, quarantines, or more detailed descriptions of how people interact with each other [1–4].

The COVID-19 pandemic illustrated the inability of many EMs to capture the disease case trajectories observed in populations [5,6]. This failure stems, in part, from a lack of attention to human behavior in EMs. Behavior strongly mediates the spread of infectious disease and can introduce non-linear effects, context dependence, delays, etc. When individuals decide to adopt protective behaviors such as isolation, social distancing, hand washing, or use of masks, they reduce the transmission of virus. Decisions to be vaccinated or to use antiviral drugs can reduce both viral transmission and health impacts in infected individuals. Behavioral decisions are related to perceptions of risk from both the disease and the protective behaviors themselves, and are also influenced by socio-cultural context and governmental policy. For example, perceived social norms (How are people around me behaving?) signal how people should behave while policy can facilitate what behaviors people can engage in (e.g., Are vaccines available? Are they subsidized?). Behavioral adoption influences the spread of the behavior itself in addition to mediating the spread and severity of disease, modifying risk perceptions, and the socio-behavioral context. Infectious disease is not primarily a biophysical phenomenon but rather a dynamic, coupled human-pathogen system dominated by socio-behavioral processes. Skillful projections and understanding of disease dynamics depend on the accurate representation of both system components—the human socio-behavioral system and the biophysical system [7–9].

Many EMs incorporate some representation of human behavior [10–17]. Some models use behavioral proxies to reduce contact rates in response to increasing disease prevalence, encoding the implicit assumption that people adopt protective behaviors with increasing disease prevalence [18,19]. In other models, cognitive states are represented as discrete switches (e.g., aware/unaware) that diffuse through populations to determine behavior [20]. Behavior has also been represented as a continuous trait that is mediated by individual beliefs and social structure [21]. Others have used game-theoretic approaches for economic optima [10–17] or evolutionary game theory to model social learning with imitation dynamics [16,22,23]. Algorithmic rules are used to map disease state to contact rates and disease transmission in agent-based models that can incorporate structured networks of interactions between individuals [24–26]. While this variety of approaches all incorporate some representation of behavioral response, they do not treat cognition and behavior as separate processes with distinct temporal dynamics. In addition, these modeling approaches often utilize frameworks that conflict with the structure of traditional EMs based on compartmental models and this can limit their utilization.

The structure of models reflects the biases and expertise of those that develop them. EMs, largely developed by mathematicians and biologists, have focused on the mathematical representation of underlying biological processes of disease spread, leaving the human behavioral component of the human-pathogen system underdeveloped. EMs would benefit from increased focus on the socio-behavioral processes, the explicit incorporation of social and psychological theory into the representation of human behavior, and from direct collaborations with social and behavioral scientists throughout the process of model development and analysis [27]. We also believe that this can be done within the compartmental structure of traditional EMs.

Behavioral scientists have developed a large set of theories to describe human cognitive and behavioral processes [28–33]. Cognition describes how humans process and interpret information to form behavioral intentions. Cognitive states are unobservable, nonlinear, interact with the environment, and are dependent on past states. Cognition and behavior operate on different timescales: Cognitive states evolve continuously in response to information about disease prevalence and virulence as well as the social and cultural context, and these states in turn mediate the discrete behavioral choices that drive disease transmission. By separating cognition from behavior, CEMs enable modeling of interventions at different time scales: information-based interventions (e.g., public health messaging) that shape cognitive states and resource-based interventions (e.g., vaccine availability) that directly constrain or enable behavioral options. We refer to EMs that represent cognitive states as continuously evolving state variables, alongside traditional disease compartments, as CEMs.

A key uncertainty is identifying which cognitive processes from the broad set of theory should be included in CEMs and how to represent them in models. There is a rich body of theory on cognition, but no single dominant framework for representing cognition. Rather than advocating for any particular theory, we distill from this literature a set of cognitive processes that mediate behavioral responses to disease. We frame human cognition fundamentally as a mechanism of receiving and processing information. Cognition determines the perception of risk and the formation of a behavioral intention that is mapped against the set of possible behaviors, leading to the adoption of a behavior. We briefly describe the processes we believe are important to represent in CEMs.

Perceived risk is the individual's continuously evolving internal representation of the threat posed by a disease or a protective behavior and is formed by processing available information with existing beliefs. Information provides cues on risks and how to behave from multiple sources, including direct experience (e.g., perceived prevalence and/or virulence of a disease), observations of how others behave (e.g., social influence), and guidance from authorities (e.g., government policy). Information processing mechanisms include biased assimilation, which reflects how individuals give greater weight to information that aligns with their prior beliefs or trusted sources, and habituation, in which the weighting of information decays over time as individuals become desensitized or adjust their expectations in light of recent experience.

Perceived risk is shaped by social influence and culture (e.g., which information sources are trusted), and in turn shapes behavioral intention.

Social influence describes how the behaviors of others affect an individual's behavior and includes perceived social norms and social learning. Perceived social norms constrain behavior to be similar to others' behaviors, while social learning expands the set of accepted behaviors as individuals observe and adopt others' efficacious behaviors. Perceived risk together with social influence drive behavioral intention, which is the mental commitment to act in a particular way. Social influence both mediates perceived risk and provides an independent driver of behavioral intention, and is mediated by culture.

Culture is a population-level trait that mediates the processing of information as well as the mapping of behavioral intention to behavior. Material culture, for example, constrains or facilitates behavioral choices through resource availability (e.g., are vaccines or masks available?) while non-material culture (e.g., social norms, values, and policies) mediates information flow and processing and constrains the range of acceptable behaviors (e.g., vaccination, mask wearing). Culture mediates information processing (which sources are trusted, how information is weighted) and constrains the mapping from intention to behavior, while itself slowly evolving as aggregated individual behaviors change population-level norms.

Learning describes how individuals adjust their cognitive states and behavior in light of continuously evolving information on disease prevalence and intervention outcomes. Learning updates perceived risk, social influence, and the formation of behavioral intentions, closing the feedback loop between disease and cognition.

These processes are dynamic and linked through feedback loops: Socio-behavioral processes drive disease spread, and prevalence of disease drives the socio-behavioral processes. Individual cognitive processes aggregate to population-level patterns, changing culture. These processes thus operate across social scales from individuals to populations and on distinct timescales: perceived risk shifts in days, behavioral intention in days to weeks, and cultural norms in months to years, producing the delayed and heterogeneous responses that distinguish CEMs from instantaneous-response models.

Cognitive processes can be modeled at the individual level or as the aggregate behavior of individuals. Agent-based models explicitly represent individual heterogeneity in cognitive states, allowing emergent population-level dynamics to arise from individuals. Alternatively, compartmental models treat populations as homogeneous in combined cognitive-epidemiological states (e.g., susceptible individuals with high vs. low perceived risk). Compartmental models can also represent distributions of cognitive states using mean-field approximations where population-level averages are represented by state variables (e.g., a population-level perceived risk) that evolve alongside traditional disease compartments.

Behavioral data to construct and parameterize CEMs is limited and often does not capture demographic and cultural heterogeneity. Cognitive processes cannot be directly observed but are inferred through surveys, behavioral experiments, and by comparing observed behavior with individuals' information environments. Data limitations do not, however, negate the utility of CEMs in understanding, exploring, and generating testable hypotheses about the interactions between disease and human behavior.

The representation of cognitive processes in epidemiological models will allow for more realistic behavioral dynamics, improve predictive skill, enhance policy relevance, and ultimately provide deeper insights into how behavior and disease interact and co-evolve over time. A traditional EM might project high uptake of vaccines as an instantaneous response to increasing disease prevalence, while a CEM would capture how cognitive states evolve over time to produce delayed, heterogeneous behavioral responses to government policy, trust in authorities and other socio-behavioral factors. This separation of cognition from behavior allows CEMs to distinguish between information-based interventions that shift cognitive states and resource-based interventions that facilitate behaviors, and provide insight into why different intervention strategies succeed or fail and how interventions might be optimally timed and targeted.

Although our focus here is infectious disease, the framework we propose is broadly applicable to coupled human-natural systems. A recent system dynamics implementation of this framework in the climate domain treats perceived risk, social influence, and culture as interacting state variables that mediate opinion change [34], demonstrating that cognitive processes can be operationalized within a compartmental modeling framework and applied across a range of contested-behavior settings.

The representation of cognitive processes that underlie behaviors will initially add model complexity and uncertainty, but will ultimately improve our understanding and forecasts of disease dynamics. CEMs will also improve our understanding of human behavior, which is increasingly important in a world where our most pressing challenges from climate change to biodiversity loss to pandemics fundamentally depend on effectively representing human behavior.

Author Contributions. Brian Beckage: Conceptualization, Writing – original draft, Writing – review & editing, Funding acquisition. Louis J. Gross: Conceptualization, Writing – review & editing. Ari Freedman: Writing – review & editing. Laurent Hébert-Dufresne: Writing – review & editing. Katherine Lacasse: Conceptualization, Writing – review & editing. Suzanne Lenhart: Conceptualization, Writing – review & editing. Chadi M. Saad-Roy: Conceptualization, Writing – review & editing. Charles Sims: Writing – review & editing.

Funding. We acknowledge the support of the National Science Foundation through Award Number 2436120 to the University of Vermont. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Prior Presentation. This article has not been previously presented, published, or otherwise disseminated.

Disclosure Statement. The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

Use of AI. We used Claude Opus 4.7 (Anthropic) to search for and summarize research papers, provide feedback on manuscript drafts, and assist with spelling and grammar.

References.

1. Hethcote HW. The Mathematics of Infectious Diseases. SIAM Rev. 2000;42:599–653. doi:10.1137/S0036144500371907

2. Giordano G, Blanchini F, Bruno R, Colaneri P, Di Filippo A, Di Matteo A, et al. Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy. Nat Med. 2020;26:855–60. doi:10.1038/s41591-020-0883-7

3. Chichakly K. Behavioral Implications in COVID-19 Spread and Vaccinations. Systems. 2021;9:72. doi:10.3390/systems9040072

4. Wormser GP, Pourbohloul B. Modeling Infectious Diseases in Humans and Animals By Matthew James Keeling and Pejman Rohani Princeton, NJ: Princeton University Press, 2008. 408 pp. 2008.

5. Bergstrom CT, Hanage WP. Human behavior and disease dynamics. Proc Natl Acad Sci. 2024;121:e2317211120. doi:10.1073/pnas.2317211120

6. Gozzi N, Perra N, Vespignani A. Comparative evaluation of behavioral epidemic models using COVID-19 data. Proc Natl Acad Sci. 2025;122:e2421993122. doi:10.1073/pnas.2421993122

7. Wang Z, Andrews MA, Wu ZX, Wang L, Bauch CT. Coupled disease–behavior dynamics on complex networks: A review. Phys Life Rev. 2015;15:1–29. doi:10.1016/j.plrev.2015.07.006

8. Verelst F, Willem L, Beutels P. Behavioural change models for infectious disease transmission: a systematic review (2010–2015). J R Soc Interface. 2016;13:20160820. doi:10.1098/rsif.2016.0820

9. Bedson J, Skrip LA, Pedi D, Abramowitz S, Carter S, Jalloh MF, et al. A review and agenda for integrated disease models including social and behavioural factors. Nat Hum Behav. 2021;5:834–46. doi:10.1038/s41562-021-01136-2

10. Tanaka MM, Kumm J, Feldman MW. Coevolution of Pathogens and Cultural Practices: A New Look at Behavioral Heterogeneity in Epidemics. Theor Popul Biol. 2002;62:111–9. doi:10.1006/tpbi.2002.1585

11. Bauch CT, Earn DJD. Vaccination and the theory of games. Proc Natl Acad Sci. 2004;101:13391–4. doi:10.1073/pnas.0403823101

12. Bauch CT. Imitation dynamics predict vaccinating behaviour. Proc R Soc B Biol Sci. 2005;272:1669–75. doi:10.1098/rspb.2005.3153

13. Reluga TC. Game Theory of Social Distancing in Response to an Epidemic. Bergstrom CT, editor. PLoS Comput Biol. 2010;6:e1000793. doi:10.1371/journal.pcbi.1000793

14. Fenichel EP, Castillo-Chavez C, Ceddia MG, Chowell G, Parra PAG, Hickling GJ, et al. Adaptive human behavior in epidemiological models. Proc Natl Acad Sci. 2011;108:6306–11. doi:10.1073/pnas.1011250108

15. Traulsen A, Levin SA, Saad-Roy CM. Individual costs and societal benefits of interventions during the COVID-19 pandemic. Proc Natl Acad Sci. 2023;120:e2303546120. doi:10.1073/pnas.2303546120

16. Saad-Roy CM, Traulsen A. Dynamics in a behavioral–epidemiological model for individual adherence to a nonpharmaceutical intervention. Proc Natl Acad Sci. 2023;120:e2311584120. doi:10.1073/pnas.2311584120

17. Glaubitz A, Fu F. Social dilemma of nonpharmaceutical interventions: Determinants of dynamic compliance and behavioral shifts. Proc Natl Acad Sci. 2024;121:e2407308121. doi:10.1073/pnas.2407308121

18. Funk S, Salathé M, Jansen VAA. Modelling the influence of human behaviour on the spread of infectious diseases: a review. J R Soc Interface. 2010;7:1247–56. doi:10.1098/rsif.2010.0142

19. Perra N, Balcan D, Gonçalves B, Vespignani A. Towards a Characterization of Behavior-Disease Models. Viboud C, editor. PLoS ONE. 2011;6:e23084. doi:10.1371/journal.pone.0023084

20. Funk S, Gilad E, Watkins C, Jansen VAA. The spread of awareness and its impact on epidemic outbreaks. Proc Natl Acad Sci. 2009;106:6872–7. doi:10.1073/pnas.0810762106

21. O'Gara D, Kasman M, Hébert-Dufresne L, Hammond RA. Adaptive behaviour during epidemics: a social risk appraisal approach to modelling dynamics. J R Soc Interface. 2025;22:20240363. doi:10.1098/rsif.2024.0363

22. Tanimoto J. Sociophysics Approach to Epidemics [Internet]. Vol. 23. Singapore: Springer Singapore; 2021 [cited 2026 Feb 8]. (Evolutionary Economics and Social Complexity Science). Available from: https://link.springer.com/10.1007/978-981-33-6481-3 doi:10.1007/978-981-33-6481-3

23. Flores LS, Azevedo-Lopes AD, Saad-Roy CM, Traulsen A. Seasonal social dilemmas. Npj Complex. 2025;2:17. doi:10.1038/s44260-025-00035-1

24. Epstein JM. Agent zero: toward neurocognitive foundations for generative social science. Princeton (N.J.): Princeton University Press; 2013. (Princeton studies in complexity).

25. Braun B, Taraktaş B, Beckage B, Molofsky J. Simulating phase transitions and control measures for network epidemics caused by infections with presymptomatic, asymptomatic, and symptomatic stages. Rizzo A, editor. PLOS ONE. 2020;15:e0238412. doi:10.1371/journal.pone.0238412

26. Zhang X, Wang J, Yu C, Fei J, Luo T, Cao Z. Agent-Based Modeling of Epidemics: Approaches, Applications, and Future Directions. Technologies. 2025;13:272. doi:10.3390/technologies13070272

27. Shin YA, Lacasse K, Gross LJ, Beckage B. How coupled is coupled human-natural systems research? Ecol Soc. 2022;27:art4. doi:10.5751/ES-13228-270304

28. Ling M, Kothe EJ, Mullan BA. Predicting intention to receive a seasonal influenza vaccination using Protection Motivation Theory. Soc Sci Med. 2019;233:87–92. doi:10.1016/j.socscimed.2019.06.002

29. Wong LP, Alias H, Wong PF, Lee HY, AbuBakar S. The use of the health belief model to assess predictors of intent to receive the COVID-19 vaccine and willingness to pay. Hum Vaccines Immunother. 2020;16:2204–14. doi:10.1080/21645515.2020.1790279

30. Fan CW, Chen IH, Ko NY, Yen CF, Lin CY, Griffiths MD, et al. Extended theory of planned behavior in explaining the intention to COVID-19 vaccination uptake among mainland Chinese university students: an online survey study. Hum Vaccines Immunother. 2021;17:3413–20. doi:10.1080/21645515.2021.1933687

31. Griffin B, Conner M, Norman P. Applying an extended protection motivation theory to predict Covid-19 vaccination intentions and uptake in 50–64 year olds in the UK. Soc Sci Med. 2022;298:114819. doi:10.1016/j.socscimed.2022.114819

32. Eyster HN, Satterfield T, Chan KMA. Why People Do What They Do: An Interdisciplinary Synthesis of Human Action Theories. Annu Rev Environ Resour. 2022;47:annurev-environ-020422-125351. doi:10.1146/annurev-environ-020422-125351

33. Hayden J. Introduction to health behavior theory. Fourth edition. Burlington, MA: Jones & Bartlett Learning; 2023. 1 p.

34. Shin YA, Constantino SM, Beckage B, Lacasse K. Climate change and opinion dynamics models: Linking individual, social, and institutional level changes. Curr Opin Behav Sci. 2025;64:101528. doi:10.1016/j.cobeha.2025.101528