HPLS (2021) 43:47
https://doi.org/10.1007/s40656-021-00407-5

NOTES & COMMENTS

# What are the COVID-19 models modeling (philosophically speaking)?

**Jonathan Fuller**[1]

Received: 30 December 2020 / Accepted: 16 March 2021


**Abstract** COVID-19 epidemic models raise important questions for science and philosophy of science. Here I provide a brief preliminary exploration of three: what kinds of predictions do epidemic models make, are they causal models, and how do different kinds of epidemic models differ in terms of what they represent?

**Keywords** COVID-19 · Epidemics · Compartment models

During the COVID-19 pandemic, philosophers and non-philosophers have been deluged with model predictions taking the form of monstrous numbers, curves, or circular tumors expanding on world maps with the growth in COVID-19 cases and deaths (Ferguson et al., 2020; Walker et al., 2020; IHME 2020).[1] These predictions have foretold a grim future and inspired severe criticism when they appeared to misfire (Ioannidis et al., 2020). They have also been used to justify dramatic public health policies. However, they are not fully understood by experts or non-experts, including many historians and philosophers (for brief overviews of epidemic modeling, see: Adams, 2020; Tolles & Luong, 2020). Philosophers have seldom studied epidemiology, and infectious disease epidemiology even more seldomly. Yet epidemic modeling raises fascinating and pressing questions for philosophy and public health. Foremost among them is the (deceptively) simple question: what are COVID-19 epidemic models modeling, anyway?

[1] This note belongs to the Topical Collection "Seeing Clearly Through COVID-19: Current and future questions for the history and philosophy of the life sciences", edited by G. Boniolo and L. Onaga.

✉ Jonathan Fuller
JPF53@pitt.edu

[1] Department of History and Philosophy of Science, University of Pittsburgh, Pittsburgh, PA, USA

Published online: 26 March 2021

While there are many facets to this question, here I will explore three: what kinds of predictions do epidemic models make, are they causal models, and how do different kinds of epidemic models differ in terms of what they represent? I will not pretend to offer satisfying answers to these questions here but will explore their motivation and contours and will offer some preliminary directions for future work in philosophy of science. Getting clearer on these questions and their answers could help modelers and policy-makers better understand the assumptions built into the structure of these models and their predictions as well as the limited perspective any epidemic model comprises.

Starting with the first question, to get clearer on the kinds of predictions that epidemic models like the ones used in the COVID-19 pandemic make, we can analyze a distinction sometimes invoked in infectious disease modeling between model forecasts and model projections. Model forecasts are often understood as model predictions about what will actually occur, while model projections are understood as model predictions about what would occur under some hypothetical scenario (Adams, 2020). Schroeder (2021) understands the distinction this way: forecasts are unconditional predictions (e.g. '2 million deaths *will* occur'), while projections are conditional predictions (e.g. '2 million deaths *would* occur *if* no infection control measures were taken'). For instance, in March 2020 Imperial College London modelers predicted that over 2,000,000 people in the U.S., 500,000 in the U.K. and 40,000,000 worldwide would die in an unmitigated COVID-19 pandemic, but that far fewer would die if a strategy of aggressive viral suppression were implemented (Ferguson et al., 2020; Walker et al., 2020). Meanwhile, in April 2020 modelers at the Institute for Health Metrics and Evaluation (IHME) at the University of Washington predicted that 60,000 Americans would die by the end of May 2020 (IHME 2020).

Given that the Imperial College modelers initially made multiple predictions predicated on different levels of policy implementation largely before countries like the U.S. and U.K. had implemented viral suppression policies, it seems reasonable to view their predictions as hedged conditional projections. In comparison, because the IHME modelers made a single prediction after most U.S. states had gone down a particular policy path and implemented 'lockdowns', it seems reasonable to view their predictions as more definite unconditional forecasts. While accepting the distinction between forecasts and projections, Schroeder (2021) casts doubt on the foregoing interpretation of the IHME model and argues from clues such as the heading of the model's website in April ("COVID-19 projections assuming full social distancing through May 2020" (IHME 2020)) that the IHME model also provided projections. However, considering that epidemic models (and scientific models generally) always make assumptions that are built into the structure of the model or the values of the parameters imputed, we should wonder whether epidemic models *ever* make unconditional predictions and worry about the cogency of the model forecast/model projection distinction. It may be, for instance, that models are best viewed as making predictions conditional on certain assumptions (after all, a model does not know whether its assumptions obtain), and that the forecast/projection distinction is best invoked at the stage when model-users use a model's outputs to make predictions, to which those users may or may not attach an antecedent condition.

Next, are epidemic models *causal* models? At this point, we should distinguish among three widely used types of COVID-19 models: compartment models, microsimulation models, and curve-fitting approaches. Compartment models divide a population into compartments corresponding to the infection status (e.g. susceptible vs. infectious) of individuals within them and track changes in the size of compartments over time. Microsimulation models assign an infection status to all individuals in a population and simulate the interactions and changes in status of individuals over time. Lastly, curve-fitting approaches (roughly speaking) extrapolate an infection curve from other population(s) and locate the current population at a point in the curve based on its trajectory until now. The Imperial College team used both a compartment model (Walker et al. 2020) and a microsimulation model (Ferguson et al. 2020) in March, while the IHME team used a curve-fitting approach in April (IHME 2020). I have been using these three models as examples because they are widely known and influential, but the details of their structure will not concern us; for our purposes, they incorporate the main features of the three model types. I will now focus on compartment models.

Compartment models have a strong claim to being considered causal models because their structure is derived from the mechanistic theory of how infections spread among individuals: individuals transition from being susceptible to being infectious to being recovered from infection as they become infected with a pathogen and then overcome their infection (or die) (Fuller, 2020). Moreover, modelers manipulate variables within a compartment model in order to estimate the effectiveness of a certain change in behavior for preventing infection. That is how the Imperial College modelers inferred that 38,700,000 lives would be saved by a viral suppression strategy compared to an unmitigated pandemic scenario (Walker et al., 2020). Presumably, only causal models would license such an estimation of a causal effect. Meyer (2020) argues that dynamical models more generally license causal explanations when their variables satisfy Woodward's (2003) manipulability criterion.

By estimating the effectiveness of policies or behavior changes, compartment models are plausibly being used to make causal inferences. However, these estimations simply involve manipulating model parameters and comparing what falls out of the model under different values, and 'causal inference' is typically thought to combine causal information with non-causal information to infer a novel causal conclusion. Thus, the idea that compartment models are causal models may be in tension with the idea that on their own they can do causal inference. If they are purely causal models, then we may intuitively think that we cannot infer new causal knowledge simply by manipulating them; any causal conclusions we derive must in a sense already be contained within the model. While we can hang on to the commitment that compartment models are causal models by accepting that manipulating parameters generates causal predictions and retrodictions rather than so-called causal inferences, it may be difficult to shake the intuition that we learn about novel causal relationships (including their quantitative strength) by tweaking model parameters.

Finally, how do different COVID-19 epidemic models differ in terms of what they represent? A microsimulation model and a compartment model can both be

used to model the same epidemic, as with the two models that the Imperial College team used in March 2020 to make predictions about the U.S. and U.K. (Ferguson et al., 2020; Walker et al., 2020). Are these models modeling different phenomena, or the same phenomenon but from a different vantage point – is a kind of perspectivalism (Massimi, 2018) the correct attitude to take towards these models? In other words, if we take these models to be causal, then do they model different causes or the same causes but from different perspectives? Consider that a microsimulation model represents the process of infection at the level of particular individuals, while a compartment model abstracts away from individuals to represent the process at the level of compartments. Therefore, prima facie these models represent the same phenomenon, differing in the level of abstraction of model variables. Microsimulation models take an individual perspective, while compartment models take a population perspective.

In order to better understand the population perspective on epidemics afforded by compartment models, we can turn to the epidemiologist Jeffrey Rose, who developed many of the conceptual foundations for the contemporary population perspective in epidemiology. However, Rose (2001) distinguished between the causes of cases and the causes of incidence in a population, where the causes of cases are responsible for differences in disease outcomes (e.g. infection) among individuals while the causes of incidence are responsible for differences in incidence (of e.g. infection) among populations. Rose seems to suggest that the causes of cases are distinct from the causes of incidence. If compartment models represent the causes of incidence and microsimulation models represent the causes of cases, then Rose's dictum might imply that these models represent *different* phenomena. However, the meaning of Rose's important ideas has not yet been sufficiently probed by philosophers (though see Fuller (forthcoming) for a contrastive causal explanationist reinterpretation of Rose's principle and its application to COVID-19).

The COVID-19 pandemic has presented an extreme challenge to scientists and policy-makers. One way or another, scientists and policy-makers have responded. Philosophers of science should now respond to the philosophical challenges posed by the pandemic, including those raised by COVID-19 epidemic models.

## References

Adams, J. (2020). What are COVID-19 models modeling? *The Society Pages*. April 8, 2020. https://thesocietypages.org/specials/what-are-covid-19-models-modeling/. Accessed December 29, 2020.

Ferguson, N., et al. (2020). *Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand*. Imperial College London. March 16, 2020. https://www.imperial.ac.uk/media/imperial-college/medicine/sph/ide/gida-fellowships/Imperial-College-COVID19-NPI-modelling-16-03-2020.pdf. Accessed December 29, 2020.

Fuller, J. (forthcoming). Epidemics from the population perspective. *Philosophy of Science*.

Fuller, J. (2020). What's missing in pandemic models. *Nautilus*. May 6, 2020. http://nautil.us/issue/84/outbreak/whats-missing-in-pandemic-models. Accessed December 29, 2020.

IHME. (2020). COVID-19 projections assuming full social distancing through May 2020. Version of 8 April 2020. University of Washington. https://web.archive.org/web/20200408105853if_/https://covid19.healthdata.org/united-states-of-america. Accessed December 29, 2020.

Ioannidis, J. P. A., Cripps, S., & Tanner, M. A. (2020). Forecasting for COVID-19 has failed. *International Journal of Forecasting*. https://doi.org/10.1016/j.ijforecast.2020.08.004.

Massimi, M. (2018). Perspectivism. In J. Saatsi (Ed.), *The Routledge Handbook of Scientific Realism.* (pp. 164–175). Routledge.

Meyer, R. (2020). The non-mechanistic option: Defending dynamical explanations. *The British Journal for the Philosophy of Science, 71*(3), 959–985.

Rose, G. (2001). Sick individuals and sick populations. *International Journal of Epidemiology, 30*(3), 427–432. https://doi.org/10.1093/ije/30.3.427.

Schroeder, A. (2021). How to interpret COVID-19 predictions: reassessing the IHME's model. *Philosophy of Medicine, 2*(1), 1–7.

Tolles, J., & Luong, T. (2020). Modeling epidemics with compartmental models. *JAMA, 323*(24), 2515–2516. https://doi.org/10.1001/jama.2020.8420.

Walker, P. G. T., et al. (2020). The global impact of COVID-19 and strategies for mitigation and suppression. Imperial College London. 26 March 2020. https://www.imperial.ac.uk/media/imperial-college/medicine/mrc-gida/2020-03-26-COVID19-Report-12.pdf. Accessed December 29, 2020.

Woodward, J. (2003). *Making things happen: A theory of causal explanation*. Oxford University Press.