

*Are COVID-19 Mortality Statistics Valid?*

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Statistics attributing mortality to the COVID-19 virus as a cause or causally contributing factor presuppose adequate answers to the following questions:

1. What causality concepts were used to generate COVID-19 mortality statistics and what do those concepts mean?
2. Were COVID-19 mortality statistics compiled using equivalent applicable meanings of “cause” or “causally contributing factor”?
3. How is it to be determined whether COVID-19 mortality statistics were compiled using equivalent applicable meanings of “cause” or “causally contributing factor”?
4. If the answer to question 2 is “no,” “not entirely” or “not sure,” what should be inferred about the validity of COVID-19 mortality statistics?
5. If there is no practical way of answering question 3, what should be inferred about the validity of COVID-19 mortality statistics?

This article provides a measure of clarity from a philosophical point of view regarding question 1, the generalized version of which, “what is causality?” has been with us since Aristotle. Conceptual clarity—one of the goals of analytic philosophy—is especially critical now because statistical data have been used to estimate the seriousness of COVID-19 as a threat to public health, project the virus’ likely course and impact, and implement measures intended to cope with it such as lockdowns, social distancing, masking and vaccination.

Question 2 raises an issue of methodology that comes up routinely any time data sets from a variety of sources are aggregated to provide an overview of a situation, across countries or

in countries taken singly. If an equivalence class is to result, as it must, equivalent properties must define membership in the sets to be aggregated. I will leave it to those who have access to the requisite data to address the issue and inform the public. If they can answer question 2, presumably they are also in a position to answer question 3, as they probably have access to operational protocols along with the data they generated.

As to question 4, if its antecedent (the “if” part) is true, there is a danger that the fallacy of equivocation has been committed, in which case it is legitimate to question the validity of statistics attributing mortality to COVID-19 in a causally relevant sense. The same point applies if the antecedent of question 5 is true. The significance of “applicable meanings” in questions 2 and 3 will become apparent shortly.

## Causality Concepts

I note first that causality in the present context is a relationship between events, not things. When it is claimed that the COVID-19 virus caused this or that condition in an organism, this is shorthand for an event involving the COVID-19 virus causing that condition. How to describe these events in biological terms I am not competent to say. But it must be done; otherwise, causal talk lacks meaning. Thus, in the definitions below, C and E designate events. (It’s a complicated question what an event is; use intuition for the time being or research the philosophical literature, which is extensive—see Sosa bibliography.)

With that proviso understood, four senses of “cause” matter in the present context, which must be distinguished and applied only if applicable (“=df” is the symbol for definition.)

**Causality 1:** C caused E =df (i) C and E both occurred and (ii) C is sufficient for E. Here we are saying that E must occur if C occurs. Thus, if a certain temperature range is reached in the right circumstances, we can infer that there will be a fire. This sense of “cause” predicts the future, inferring effect from cause.

**Causality 2:** C caused E =df (i) C and E both occurred and (ii) C is necessary for E. Here we are saying that without C, E cannot occur. Thus, if there was a fire, we can infer that oxygen must have been present. This sense of “cause” retrodicts the past, inferring cause from effect. (Science must predict as well as retrodict, e.g., the next solar eclipse as well as past ones.)

**Causality 3:** C caused E = (i) C and E both occurred and (ii) all other things being equal (*ceteris paribus*), C is sufficient for E.

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For present purposes, I will assert without argument that Causality 1 and 2 apply in physics and chemistry (the “hard sciences”) but not biology, psychology and economics,

where Causality 3 and 4 apply.

The key difference between Causality 1 and 2, on one hand, and Causality 3 and 4, on the other, is in the laws of nature on the basis of which explanations, predictions and retrodictions can be made (see Armstrong [9].) In physics and chemistry, strong universal correlations hold between events—e.g., Newton’s Laws and Boyle’s Law—so we are on firm ground when it comes to explaining, predicting and retrodicting. Everywhere else, laws (if any) are *ceteris paribus*, or CP-laws, which allow exceptions. Various factors that can “perturb” a relationship between events must be kept constant to apply CP-laws, which is not always easy to do, not even in the laboratory. Explanation and prediction based on CP-laws are subject to hedging of one sort or another and are less reliable. How much less reliable is hard to say; likewise the reliability of results based on CP-laws. Thus, stimulus-response laws in psychology are CP-laws at best, as are supply-demand laws in economics. Godfrey-Smith notes [7, p. 23] that laws in biology are at best CP-laws.

To put all this in the context of questions 2-5, COVID-19 mortality statistics may apply only Causality 3 or 4, not Causality 1 or 2. I will leave it to others to determine if the latter is taking place. If it is, statistical results are invalid.

- It would be helpful to know which of Causality 3 or 4 is being applied to generate COVID-19 mortality statistics and how it is done. Mixing them up is a bad idea. Causality 4 would list the virus as one of several causal factors and thus would yield an aggregated estimate not necessarily consistent with one applying Causality 3.
- If some other senses of “cause” or “causally contributing factor” were applied to generate COVID-19 mortality statistics, they should be identified and it should be explained why statistics are valid. Mill’s Five Methods (Hurley [7], Ch. 10) offer several options for testing causality, though not all are applicable to COVID-19.

## Causality is Not Constant Conjunction

If you live on a farm and want to wake up at the crack of dawn for a busy day ahead, there’s no need to set your alarm clock. Rocky the Rooster will take care of it. A healthy, mature bird will crow as the sun begins to rise.

Rocky is a counterexample to an idea that has a good deal of intuitive appeal: To know whether event C causes event E, all we have to do is observe that the two events follow each other with a high degree of regularity. Right?

Wrong! Roosters don’t cause the sun to rise any more than night causes day just because one regularly follows the other. There’s more to causation than constant conjunction. The

\$50,000 question is, what more is there?

Here's one way to think about it. Suppose you get change for a purchase in dimes, seven of them. You put the coins in your pocket. You don't have any other coins in your pocket, so we have the true generalization—which is a statement of the form “All A are B”—that all the coins in your pocket are dimes. So, is it true that if a quarter were to be a coin in your pocket along with the dimes, it would also be a dime? Of course not! Quarters don't become dimes because they're in a pocket full of dimes.

The issue is whether a generalization has predictive force. Cause-effect generalizations do, supporting counterfactuals—statements of the form “if X were to occur, Y would occur” or “if X had not occurred, Y would not have occurred” (see Lewis [2].) The one about Rocky and the sunrise obviously doesn't. “If Rocky hadn't crowed, the sun would not have risen” is absurd.

So, “A and B,” however regular, does not mean “A caused B,” regardless of one's analysis of “cause.” Thus, COVID-19 mortality statistics may not conflate “the COVID-19 was virus present in the deceased” (died with the virus) and “death was caused by the COVID-19 virus” (died from the virus.) Whether COVID-19 mortality statistics respect this distinction, and do so as rigorously as they should, is not for me to say. I hope so; otherwise, the validity of these statistics is in doubt.

## A Final Thought

One of the key considerations noted above, that the fallacy of equivocation is committed unless criteria for determining causality are consistent over an aggregated data set, also apply to tests used to determine the presence of COVID-19 in an organism.

The fallacy can (and must) be avoided by standardizing COVID-19 test methodology and test result analysis. Some, such as body temperature and blood oxygen level, are relatively straightforward. The more common one, the polymerase chain reaction (PCR) test, is not. PCR amplifies a DNA segment in a collected sample, so consistency applies to two test components: (a) sample collection methodology, and (b) the cycle threshold used to amplify the DNA segment. It would be helpful to know what is scientifically optimal for (a) and (b) and whether applicable criteria are met consistently for both.

## References

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