Why correlation is not even part of causation

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Hume’s analysis of causation

The philosopher David Hume offered an analysis of causation that has deeply influenced our theoretical causal thinking. According to this analysis, causation is tightly linked to perfect correlation: whenever the cause occurred, the event would typically follow.

One type of event is always followed by another type of event. Example: Hume’s billiard balls. What we can observe here, according to Hume, is the following:

a. correlation (A and B both occur)
b. succession (A occurs before B)
c. contiguity (contact between A and B)

Causation is nothing but correlation

This is what we can call a regularity view on causation. The cause is constantly conjoined or correlated with its effect, but there is nothing more connecting them.

Causation thus becomes an unobservable relation between two observable events, A and B. If occurrences are the only things that can be observed, and therefore verified or falsified, we easily end up in scepticism about causation: causation is nothing more than correlation.

Might correlation be enough for causation? Statistical correlation is often used as an indication of causation. Examples: Happier without kids. Married men live longer.

Causation is correlation plus something more

Philosophers have been criticising Hume, arguing that correlation is not sufficient for causation: we need something more that ties the cause and its effect together, for instance some necessary connection between them.

Such a connexion wou’d amount to a demonstration, and wou’d imply the absolute impossibility for the one object not to follow, or to be conceiv’d not to follow upon the other (Hume, A Treatise of Human Nature, 1739: 161-2)

What has been largely neglected, however, is that correlation is not even necessary for causation. Most, if not all, cases of causation is less than perfect correlation: smoking causes cancer, birth control pills cause thrombosis.

It seems to be the rule rather than the exception that causation is interfered with by counteracting causes. Any causal process can be counteracted, interfered with or even prevented.

The problem of causal interference

Most philosophical theories of causation are informed by theoretical physics. This area is characterised by its extensive use of idealised models and theoretical abstractions. Any causal interaction is typically considered within a clearly defined and closed system.

In both science and philosophy we look for law-like behaviour and universal truths, similar to what can be found in theoretical physics. Any disturbance to this law-like behaviour or exceptions to universal truths is treated as noise, or even as irrelevant.

In science we try to shield off any interfering factor in experiments or by theoretical abstraction in models. The aim is to limit and control the number of causal factors to find perfect correlations or laws.

In philosophy we try to avoid causal interference by making the cause so big that it includes everything needed to produce the effect and excludes all interferers. The aim is to find a cause that is sufficient for the effect.

If a cause is not sufficient for the effect, we need to make it bigger. But the bigger the cause gets, the less likely it is that we get a recurrence.

A solution is to stipulate some ideal conditions under which the effect would occur: A causes B, ceteris paribus. If we have the cause but not the effect, then clearly the conditions were not ideal.

Perfect correlation of cause and effect seems to be a result of theoretical abstraction, not observation. In the real world idealised situations do not exist, and it is impossible to abstract away from all interfering factors.

So while theoretical physics seems perfect for a theory of causation that embraces law-like regularities, the macroscopic world with which we are familiar seem to require something radically different.

In this paper I present an alternative that would fit better to applied sciences: a dispositional theory of causation. On this account, causal interference is not something that is treated as a problem, but rather is embraced as an essential part of causation.

Tendencies, not laws

Instead of strict laws of nature, governing the behaviour of objects, a dispositionalist emphasises that things have real causal powers. They behave the way they do in virtue of their own dispositional properties, not because of external laws (Stephen Mumford, Laws in Nature, Routledge 2004).

Typical examples of dispositions are fragility, solubility and fertility. A disposition can have various intensities.

Something can be more or less fragile, soluble or fertile.

Dispositions bring with them real potencies or tendencies. This is a modality that is short of necessity but more than pure possibility. Dispositions tend towards their manifestations only. Dispositionality is the modality needed for causation (Mumford and Anjum, Getting Causes from Powers, Oxford University Press 2011, ch. 8).

A cause is something that tends towards its effect without guaranteeing it: smoking tends towards cancer, but not everyone who smokes gets cancer. A tendency can be counteracted by other tendencies: a healthy diet tends away from cancer.
Less than necessity, stronger than probability

Many philosophers admit of a type of causation that is short of necessity. All laws that give less than perfect regularities would then be probabilistic laws. The first is often associated with deterministic causation and the second with indeterministic causation.

With tendencies there is a better alternative that keeps the question of determinism or indeterminism separated from causation. Example: Half of all smokers will die from it. This is a statistical fact and less than a perfect correlation. For any two smokers there is the possibility that one dies from smoking while the other doesn’t.

This is explained without statistics or probability. There might be a difference in their diet, lifestyles or biological dispositions. Contrast this with cases of genuine chance as some believe coin tosses and dice rolls to be.

Causation itself is neither law-like nor probabilistic. It is about real tendencies, and such tendencies can hold universally even if they are not manifested in every case.

In order to deny a causal connection, one can therefore not look for counterexamples. Most people who smoke don’t get cancer. Instead one must deny the tendency itself; that there is such a disposition of smoking.

Whether a tendency will manifest will depend on its intensity and other factors involved. This is because of causal complexity, context sensitivity, and the possibility of causal interference.

A dispositional theory of causation

Modality: Dispositionality or tendency is a modality stronger than pure contingency but weaker than necessity. It is more than just a possibility that a fat rich diet is linked to diabetes 2, but it is also less than necessity: not all people who have a fat rich diet develop diabetes.

Complexity: Causation is typically complex: the effect is produced by many causal factors. Example: a number of causal factors can produce high blood pressure and often there will be a combination of factors: smoking, stress, alcohol, salty diet, physical inactivity, diabetes, obesity, genetic pre-dispositions.

Context-sensitivity: For any two people who smoke the outcome might differ depending on contextual factors. A smoker might have an otherwise healthy lifestyle that keeps the blood pressure down. Causal production is thus a highly context dependent matter.

Subtractive interference: A doctor might advise the patience with high blood pressure to quit smoking and drinking.

Additive interference: If subtractive interference is not sufficient or possible, the doctor might subscribe medication to lower the blood pressure.

Threshold: Causal production occurs when causes accumulate and reach a certain threshold effect. This might be a stage of the causal process at which something new happens (water starts boiling), or in which we have an interest (reaching a particular room temperature).

Non-linearity: Causes compose in a number of ways, not just additively. One such example is clonidine and betablockers, two medications that each disposed towards lowering blood pressure. Taken in combination, however, they tend to increase blood pressure in 6 of 10 cases.

No necessity: Cases of additive prevention show that causes do not necessitate their effects, at least not in the sense that ‘whenever the cause occurs, the effect must occur’. Something could always have been added that prevented the effect from occurring.

Induction: Our causal inferences are fallible: we can never know in advance whether a cause will be prevented from producing the effect. What we can predict is whether there will be a tendency towards an effect, but not whether the effect will actually occur.

Perfect correlation is not causation

If causes are tendencies, then we should not expect to get the effect every time we have the cause. In fact, we should expect causal interference and less than perfect correlation.

Rather than grounding causation in perfect correlations, as philosophers have done since Hume, we should take the opposite view: a perfect correlation would be indicative of something else than causation.

Cases of perfect correlation are identity, classification and claims of essence. An extra chromosome does not cause Down Syndrome; it is a definition of the syndrome. Being a whale does not cause it to be a mammal; it is classified as a mammal.

Humans are mortal and might be essentially so. If we managed to find a “cure” for mortality, the correlation between humans and mortality would then be unaffected: whatever is not mortal would then not be classified as a human.

Identity and classification also give us perfect correlations because they – totally unlike causation – are insensitive to contextual changes.

Understanding causation by way of failure

Causes no more than tend or dispose towards their effects, and this has implications also for how we come to learn about causal connections.

We do not learn about causation by observing that the cause is constantly conjoined with the effect. On the contrary; in order to understand causation, we need to understand how things causally interact, including what could counteract and prevent the effect from occurring.

Constant conjunction does not give us an understanding of underlying causal mechanisms. If a lamp lit up every time we pressed a certain light switch, and it didn’t matter whether the electricity was switched off, the bulb was broken or the wire butts where sticking out of the wall, we might start questioning whether pressing the light switch was actually the cause of the lamp getting lit.

This is because causation is essentially sensitive to contextual changes. Demanding constant conjunction for causation does not give us an idea of interrelatedness, but rather forces us to deny causal relation whenever the effect does not follow the cause.

I suggest instead that it is when causation fails that we learn how this object and its properties interacts and relates to other objects and properties.

To understand how things causally relate seems also to be an important motivation behind the experimental method. We want to map the causal landscape and separate causally relevant factors from all the causally unrelated factors.

It is when causation fails that we get an idea of relevant causal factors and thus expand our causal knowledge.