

The psychology of time and causality

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Abstract

In this paper I present a brief overview of the relationship between time and causality in human experience. More specifically, I will outline that humans use temporal contiguity as a cue towards causality, and that in turn impressions of causality can shape perceptions of what is contiguous.

1. David Hume's heritage

Causality pervades nearly every aspect of human experience. Based on causal understanding, we decide which foods we like and which ones make us ill, who is our friend and who is our enemy, which tool is working best, and where to invest our resources. In other words, causal knowledge enables us to predict and control our environment. The ability to acquire such knowledge thus is of central significance to human intelligence. However, despite the all-pervading nature of causality, it cannot actually be perceived directly. While we can observe that ingesting large amounts of alcohol tend to be followed by headaches, and that ingestion of aspirin may be followed by a relief of these headaches, we cannot actually perceive the causal relations at work directly. David Hume [1] observed that causal relations cannot be directly appraised by the sensory system. There is no causality sense'. Yet, our senses are the ultimate source of our experience. Consequently, according to Hume, causality must be construed in the mind, by transforming essentially non-causal sensory input into representations of causal relations. This empiricist stance still dominates cognitive science approaches to causality today. Hume identified three critical empirical cues that may facilitate causal learning or attribution:

- temporal priority: the cause has to happen before the effect,
- constant conjunction (also referred to as contingency): the cause and the effect have to co-occur together repeatedly and reliably,

- contiguity in space and time: the co-occurrence of cause and effect needs to be close in space and time.

The first of these cues is almost universally accepted and seldom subjects to empirical investigation. The second cue – contingency – has received a lot of attention from learning theorists and psychologists. In fact, the majority of the literature on human causal learning focuses on the question of how statistical patterns of contingency are transformed into internal representations of causal strength, structure, or power (see [2, 3] for an overview). In the following section, I will review key psychological evidence for the third cue.

Before going there, however, it may be worthwhile to take a detour and provide some clarification regarding the relation between contingency, correlation and causality. Virtually every introductory text to statistics admonishes readers that correlation does not imply causation, yet Hume’s second cue precisely states that from the regular conjunction of the candidate cause with its effect we form the mental abstraction that the former causes the latter. So how do we make the mental leap from covariation to causation, and ensure that we only identify genuine causes and leave spurious relations alone? While we sometimes indeed draw false conclusions, plenty of anecdotal evidence suggests that the human mind is exquisitely able to distinguish genuine from spurious causes. How come, for example, that we do not think that the rooster’s regular crowing in the morning causes the sun to rise?

This is a deep philosophical and psychological question that is well outside the scope of this article, and indeed has led to a divergence within cognitive psychology between the empiricists, who subscribed to Hume’s heritage and so-called causal power theorists, *e.g.*, Refs. [4, 5], who follow in the footsteps of Immanuel Kant [6]. According to power theorists, people recognize a causal relation when they are aware of a *causal power, force, or propensity* that has the capacity to bring about the effect. We don’t think that the rooster’s crowing causes the sun to rise because we do not know of a mechanism whereby the rooster or its crow could lift the sun over the horizon. It does not take much, however, to see that the power view suffers from circularity: In order to identify a causal relationship, we first need to know that there is causal power. How to overcome this obstacle without falling into the equally unattractive trap of endorsing every correlation that we encounter as causal?

Patricia Cheng [7] offered an elegant solution: Her suggestion is that reasoners are able to assess covariations at the level where the contrast is maximal, and that this ability to strive for maximum explanatory coherence enables us to identify genuine causal relations. An example would be the relationship between *smoking* and lung cancer. Yes, there is a correlation between them, and now (after many years of toil) we have come to understand that the former indeed causes the latter. And we have asked the question about smoking and lung cancer. We could just as well have categorized the cause at a different level of abstraction. For example, we could have only asked whether smoking Marlboro cigarettes

causes cancer. This would be an example of a narrower definition of the cause—smoking other brands of cigarettes would then constitute the absence of the cause, and the contrast (i.e. the difference between the likelihood of the effect in the presence of the cause minus the likelihood of the effect in the absence of the cause) would therefore be smaller than in our original definition. Likewise, had we considered the inhalation of fumes as the cause, then the cause would now be defined more broadly, and many non-carcinogenic fumes (like steam) would be included, again lowering the contrast. Lien and Cheng have shown that people automatically “zoom in” on that level of abstraction where the explanatory contrast is the greatest, and this ability is what enables us to distinguish genuine from spurious causes (see also [8]).

2. The role of temporal contiguity in causal learning

But now, let us return to the relation between time and causality. The first systematic investigation of temporal contiguity in causal learning was conducted in 1987 by Shanks, Pearson, and Dickinson [9]. Shanks et al. presented participants with an instrumental learning task: they had to find out to what extent pressing a key on the computer made a triangle on a computer screen light up. The apparatus was programmed with a contingency of 75%, i.e. three out of four key presses produced the target effect. Shanks et al. reported that people’s ability to correctly identify this relation decreased rapidly when a delay between action and outcome was inserted. More specifically, delays of four seconds or more meant that subjects could no longer distinguish contingent conditions—those where three out of four actions produced an outcome—from non-contingency conditions—where their actions had no consequences and outcomes occurred seemingly at random. While this result fits with similar findings obtained from non-human animals, it is considerably at odds with our common sense understanding of human intelligence: We certainly seem to be able to acquire, appraise, and exploit long-term causal relations. How else would doing agriculture be possible, for example?

One solution to this paradox was proposed by Buehner and May [10, 11, 12], who demonstrated intact causal learning over a four second delay in a paradigm modeled on Shanks et al. [9]. Unlike Shanks et al., however, they provided their participants with a rationale for why the effect may sometimes be delayed. Thus knowledge of a plausible mechanism, and the timeframe this entails, enables reasoners to bridge temporal gaps. However, and this will prove important later on, the default assumption is that causal relations are immediate. In other words—everything else being equal, contiguous causal relations are much easier to learn than non-contiguous ones. This effect is even true in cases where the cause-effect contingency is not experienced in real time, but can be directly read off from a table. Greville and Buehner [13] found that even when statistical relations are presented in plain sight to participants, they will take contiguity information into account, and combine both sources of information to form causal judgments.

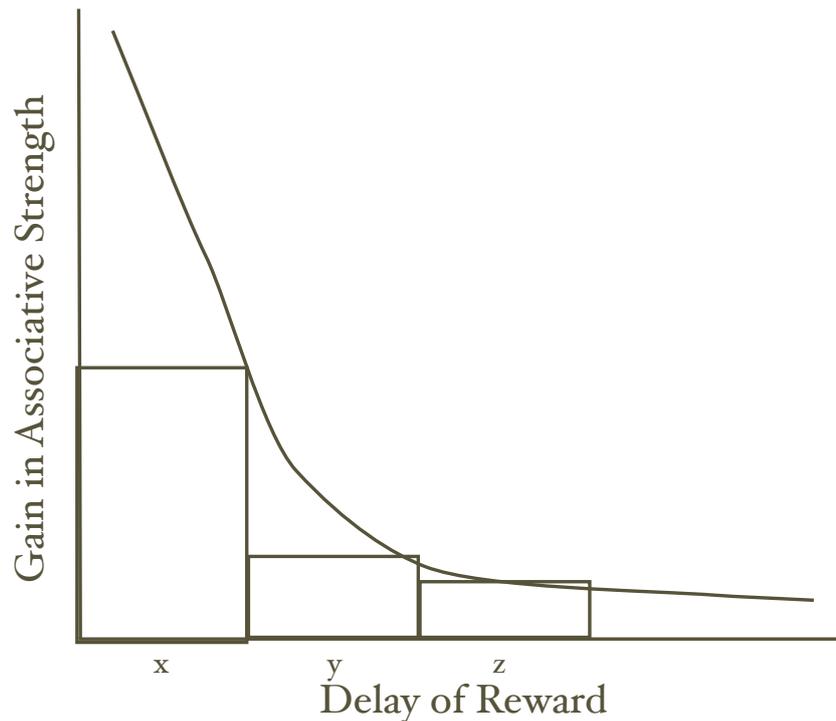


Figure 1: A hypothetical example of a discounting function. For the purposes of causal or associative learning, the subjective value (*SV*) plotted on the *y* axis translates directly to gains in associative strength.

When we learn about delayed causal relations, there is likely to be some variation in the extent of the delay. In other words, a learning experience may consist of multiple cause-effect intervals, and not only can the average of this interval vary between different causal relations, but also the variance of the (sampled) cause-effect intervals. How might (the extent) of such variance influence causal learning? Or, to put the question more formally: if we were to compare two sets of experiences with the same average cause-effect delay, one where this delay is constant, and one where it varies around the average, which of them might afford better causal learning? Looking towards the literature of animal and associative learning, one might expect that the variable interval sample would afford better learning. This is because of the negatively accelerated discounting curve which models the value of rewards over time (see Fig. 1). Reward learning usually always involves discounting, such that —everything else being equal— immediate rewards are preferred over delayed rewards of the same magnitude: We all would rather have \$100 today than \$100 in four weeks. The exact nature of the discounting curve is a function both of the nature of the reward (consumable rewards like food, drink, or drugs lose their value faster than non-consumable rewards like money), and individual differences (see [14, 15] for more detailed discussions).

Be that as it may, the general shape of the discounting curve implies that a combination of early and late rewards carries higher utility or value than accruing repeated instances of a middling reward. In Fig. 1,

$$x + z = 2 * y, \tag{1.1}$$

indicating that the average delay is identical, yet when considering the subjective value (SV),

$$SV(x) + SV(z) > 2 * SV(y) . \quad (1.2)$$

From this perspective then, variable cause-effect intervals might afford better (i.e. faster, or stronger) learning. Considering the symbolic nature of a causal relation, however, one might make the opposite prediction: Most natural causal mechanisms involve a pretty constant delay (consider the regularity of cosmological patterns, biological cycles, etc). From a Bayesian perspective, constant cause-effect intervals would dramatically improve causal learning. A constant delay is extremely unlikely to be produced by chance, and thus is a clear signal of some meaningful relation between the two events of interest. In contrast, a statistically reliable pattern involving variable delays will be much harder to identify. Greville and Buehner found exactly that [16]: Instrumental causal relations involving constant delays afforded better causal learning than relations with variable delays averaging around an equivalent mean.

3. The role of causality in appraising temporal and spatial contiguity

The previous section illustrated how human causal learning neatly follows the bottom-up principles laid out in David Hume's empiricism: The human sensory system appraises the hard facts about the presence and absence of objects and events, as well as the temporal (and spatial) patterns between them, and derives a mental construct of causality from this. A little over a decade ago, work from Patrick Haggard's lab [17] strongly challenged this position. Haggard et al. asked their participants to report the time of either their own action (key press) or an external event (beep) with reference to a fast moving clock projected onto the computer screen. Once they established a participant's baseline judgment error using this method, they changed the procedure so that the participant's key press resulted in the beep after a short delay. Again, participants had to report either the time of their key press or the time when they heard the tone. Haggard et al. found systematic shifts in participants' judgment errors: relative to the single-event judgment errors derived at baseline, now participants reported the time of their action to be later, whereas they reported the time of the outcome (i.e. the beep) as earlier. In other words, the action and its outcome mutually attracted each other in time.

Subsequent work revealed that this temporal binding only occurs when the action is in fact a causal instrumental action [18] and that the cause need not be a human action machine causation is just as effective [19]. At the same time, the effect has been demonstrated within a range of experimental paradigms and methods [20, 21, 22, 23, 24], for example see [25]. The causal binding between cause and effect occurs only in the temporal domain, but also in the spatial domain: stimuli involved in simulations of collision events are judged to be closer in space when they were linked by a causal collision compared to other, non-causal, displays [26].

Causal binding in time and space actually follows from a Bayesian interpretation of Hume's principles: if it is the case that temporal or spatial contiguity increase the likelihood that we form a causal connection between two events, then it is also true that once we have formed such a connection, it is also more likely that cause and effect are relatively contiguous in space-time. Because human time perception is inherently ambiguous and noisy, the mind can attempt to resolve some of this ambiguity by drawing on higher level knowledge of causality between the constituent events. The relationship between time and causality—at least in the world of human experience—thus is a two-way street. Not only do we build causal representations based on our empirical observations of contiguity between events, but our perception of what is contiguous is in turn shaped by the representations of causality these observations have helped to create.

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Volume 7
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