Temporal Components in Judgements of Singular Causation

Ignacio Acosta Hernandez (ignacio.acosta@mail.utoronto.ca)

Afnan Rahman (afnan.rahman@mail.utoronto.ca)
Department of Cognitive Science
University of Toronto

Abstract

Making causal inferences among events is a core cognitive process of human mind and depends largely on not only the causual strengths of the potential causes of the target outcome, but also the temporal information of the causes. To determine whether two co-occurring events are causally connected or they are mere coincidence, and to investigate how human infers singular causation in different conditions of causal strengths and temporal relations, a computational model was built from ground up following the methods proposed in Stephan, Mayrhofer and Waldmann (2020). By combining these factors the model is able to accurately mimic the human causal rating observed from three different experiments. The paper provides a step-by-step breakdown of the model development and the mentioned paper replication. Plots and visualizations are included in the appendix.

Keywords: General Causation, Singular Causation, Temporal Order, Onset, Causal Latency, Causal Strength, Gamma Distribution

Introduction

The ability to draw a causal relationship between two events is at the core of everyday decision-making. Deciding whether two events occurring together is merely a coincidence or whether there is a causal relationship between them, is crucial for understanding how the world works. Causal relationships can be categorized into two: general causation, and singular causation. As the names imply, general causation is a general understanding about the causal relationship between two events. For example we might say that, in general, smoking causes cancer. Singular causation, on the other hand, is the understanding of a causal relationship between two particular events in a single instance. For example, drawing the conclusion that John has cancer because he smoked and not because, say, he works with toxic fumes.

The concept of singular causation is derived from the idea of general causation. A singular co-occurrence of events c and e of the general factors C and E need to be measured while assessing singular causation, even if there is a defined general causal /co-incidental relation between C and E (i.e., $C \rightarrow E$) (Stephan, Mayrhofer, and Waldmann, 2020).

Singular causation's application is critical not only in our everyday decision-making but also crucial in areas like medical diagnosis, criminal investigations and engineering design. One real-life scenario: let's say a cognitive science student RSVP at a Women in Cognitive Science Conference. However, a few days before the conference they find out that it is postponed (event e) and they also learn that the keynote speaker had cancelled (event c). How can we infer whether these two events are causally related, in other words, whether the panel was postponed because of the keynote speaker's cancellation? Or were there some other reasons? The two factors that can help answer these questions are: Were there any other reasons that may have caused the conference to postpone? If yes, were they more important than the speaker's attendance? What is the temporal order of these causes? Did they occur before or after the speaker cancelled? So the list of questions goes on. As such, making accurate causal judgments can be difficult, as events often have multiple potential causes and the temporal order of events can be ambiguous.

Past research that investigates the computational approach to determine singular causation for two simultaneous events, mainly focuses on causal selection. It is the human tendency to focus on a subset of potential causes when trying to explain an observed effect (Phillips, Luguri and Knobe, 2015), even if multiple factors may have contributed to that effect. This can lead to human errors or biases in causal judgments, as people may overlook important causes or even assign causality to factors that are not in turn responsible for the effect. Most research, however, fails to consolidate the two main factors: causal strength (the probability of the target cause causing the target outcome) and temporal information of the two events.

Stephan, Mayrhofer, and Waldmann (2020), which we partly replicate here, investigate how the causal links in singular cases are created. It explores how people reason singular causation in situations where they have limited information about the potential causes and their temporal relation. The computational model proposed in the paper builds on the power PC framework of causal attribution proposed by Cheng and Novick (2005) and its extension by Stephan and Waldmann (2018) and integrates causal strength and temporal information to successfully assess singular causation relations. The Bayesian network lays the groundwork of the causal reasoning (Pearl, 1985). The model suggests that two types of information are required to infer singular causation between two co-occurring events: (a) information about the causal strengths of the potential causes, and (b) information about temporal features of the potential causes and about their temporal relation. The two temporal features that were integrated into the model are (a) the onset times of the potential causes, and (b) the causal latencies: the time it takes the

causes to show their effects. For example, when taking an aspirin pill, the moment in time that you take it is the causal onset, whereas the time it takes to generate the effect of reducing pain is the causal latency.

In this project, we have replicated the paper to build the suggested model from the ground up and tested the model's performance using the behavioural data from the first three experiments mentioned in the paper. This is a slight deviation from our initial project proposal as we are no longer considering the uncertainty-accounting model (UAM) of Marchant et al. (2023).

Materials and Methods

The analysis was conducted where a target effect E can be produced by two potential causes: either by the target cause C or by an alternative cause A. By following the steps mentioned in the paper, we have built the model from scratch and have tested the experiment data from the first three experiments to replicate the same results as the paper. The link to the project GitHub can be found here.

The methods can be divided into three sections: a) The derivation of the generalized standard model that calculates the probability that c caused e on a singular occasion on which c, e, and a potential alternative cause a are known to have occurred (considering the causal preemption between the two causes), b) Testing the model with behavioural data.

The Derivation of the Generalized Model:

The general causal relationship between three variables: C, A, and E, can be illustrated through a common-effect causal Bayes net, as seen below in figure 1, with the assumption that C and A are independent causes of E. Here, the variables b_C and b_A are the base rates of the two causes (C and A), while w_C and w_A denote the causal strengths of C and A, respectively. The influences of A and C are combined according to a noisy OR-gate.

Figure 1: General Bayesian Net

The causal Bayes net framework and rational models of causal induction can help derive the causal strength as the probability with which a cause generates the effect E without any influence from the other causes of E. In other words, the causal strength of a cause denotes how likely it is to produce the effect, all other things being equal.

The standard power model of the causal attribution model (Cheng and Novick, 2005) mainly focuses on the causal strengths of the potential causes to decide whether a particular cause was the actual cause. According to the standard model, $P(c \rightarrow e|c,a,e)$ is the probability that c caused e on a singular occasion on which c, e, and a potential alternative cause a are known to all have occurred together:

$$P(c \rightarrow e|c,a,e) = \frac{w_C}{(w_C + w_A) - w_C \cdot w_A}$$

Here, the target cause's causal strength, w_C, is normalized by the probability of the effect in the presence of its potential causes, $(w_C + w_A) - w_C \cdot w_A = P(e|c,a,e)$. It is sensible to describe this probability this way as it is the union of both potential causes, minus their intersection. The standard model is able to accurately explains the notion that as the target cause's (c) causal strength increases, the probability of c being the singular cause of an effect also increases (Stephan and Waldmann, 2018). However, the model fails to explain causal preemption. Causal preemption occurs when an alternative cause is able to generate its effect faster than the target cause. Two factors can make this happen: causal onset and causal latency. Causal preemption by an alternative cause might occur, for example, if the alternative cause occurred later than the target cause but was also faster acting so that it generated the effect first.

In order to account for causal preemption, Stephan and Waldmann (2018) have suggested a generalized model of the standard model:

$$P(c \rightarrow e|c,a,e) = \frac{w_C - w_c \cdot w_A \cdot \alpha}{(w_C + w_A) - w_C \cdot w_A}$$

Here the new parameter,α, adds a weight to the intersection $w_C \cdot w_A$. And it represents the probability that the alternative cause preempts the target cause:

$$\alpha = P(t_{a\rightarrow e} + \Delta t < t_{c\rightarrow e}|e,c,a)$$

Here $\Delta t = onset(a) - onset(c)$. Thus if the alternative cause occurs first, $\Delta t < 0$, and if both causal latencies are equal and fixed, $\alpha = 1$. Which in this case means that the alternative cause will always preempt the target cause becaue it happened earlier and they both had the same latency. However, Stephan et al. (2020), argue that causal latencies are not fixed and instead follow a gamma distribution. For this cases, α can be calculated with the following Montecarlo Sampling procedure:

1. Sample N pairs of ($t_{c\rightarrow e}$, $t_{a\rightarrow e}$) from causal latency distributions.
2. Calculate $t_{a\rightarrow e} + \Delta t$ for all pairs
3. Count the pairs in which $t_{a\rightarrow e} + \Delta t < t_{c\rightarrow e}$
4. Divide this count by N

Model Testing:

The data analysis of the first three experiments described in Stephan et al. (2020) were replicated to test different combinations of causal strengths and temporal factors (onset and causal latency) on the model. Participants were presented with a cover story about attacking barbarians. Two different watchtowers were to send pigeons to the palace when barbarians attacked. The subjects were then tasked to answer

the causal query of which tower caused the alert of the attack given that both towers sent their pigeons (two co-occurring possible causes for a particular event). The causal onset is represented as the time it takes for a watchtower to react, the causal latency as the time it takes for a pigeon to arrive from the tower to the palace, and the causal strength as the probability that the pigeon gets lost in the way. There was a learning phase before the query to learn the onset, latency and strength, and the query was based on an 11 point confidence rating, as to mimic a probability rating. The different variables were manipulated in different ways in the experiments and they are described below:

Experiment 1 The first experiment aimed to test the hypothesis that humans use temporal information when making judgments about singular causation. For this reason, in this experiment, causal latencies were assumed to be fixed (do not follow a gamma distribution). And the causal strengths were assumed to be deterministic ($w_A = 1$ and $w_C = 1$). Then, the values for causal latency and causal onset were manipulated in different conditions. Where all conditions have a 'short' cause that is the fastest acting one, and a 'long' cause which is the slower acting one.

- Condition 1
  Set the causal onset different and the latencies the same with the short cause having an onset of 800ms and latency of 800ms. And the long cause having an onset of 1600ms and a latency of 800ms.
- Condition 2
  Set the causal onset the same and the latencies different with the short cause having an onset of 800ms and latency of 800ms. And the long cause having an onset of 800ms and a latency of 1600ms.
- Condition 3
  Set the causal onset different and the latencies different with the short cause having an onset of 1600ms and latency of 800ms. And the long cause having an onset of 800ms and a latency of 2400ms.
- Condition 4
  Set the causal onset different and the latencies different with the short cause having an onset of 800ms and latency of 1600ms. And the long cause having an onset of 2400ms and a latency of 800ms.

Experiment 2 The goal of this experiment was to discover whether humans are sensitive to probabilistic causal latencies. In this experiment, causal latency was assumed to be probabilistic and follow a gamma distribution. Like Experiment 1, the causal strengths in this experiment were assumed to be deterministic ($w_A = 1$ and $w_C = 1$) and the onset was assumed to be equal in all conditions. The model was tested upon five conditions where pairs of gamma distributions were compared. As the value of α is dependent on the specific gamma distribution, varying the shape and scale of the distribution amounts to varying the value of α, which is really the variable of interest here.

- $\alpha = 0.01$
  Target cause's causal latency $\sim$ *Gamma*$(10.00, 100)$ and alternative cause causal latency $\sim$ *Gamma*$(30.00, 100)$.
- $\alpha = 0.25$
  Target cause's causal latency $\sim$ *Gamma*$(17.77, 100)$ and alternative cause causal latency $\sim$ *Gamma*$(22.00, 100)$.
- $\alpha = 0.50$
  Target cause's causal latency $\sim$ *Gamma*$(20.00, 100)$ and alternative cause causal latency $\sim$ *Gamma*$(20.00, 100)$.
- $\alpha = 0.75$
  Target cause's causal latency $\sim$ *Gamma*$(22.00, 100)$ and alternative cause causal latency $\sim$ *Gamma*$(17.77, 100)$.
- $\alpha = 0.99$
  Target cause's causal latency $\sim$ *Gamma*$(30.00, 100)$ and alternative cause causal latency $\sim$ *Gamma*$(10.00, 100)$.

Experiment 3 The previous two experiments aimed to test the role of the temporal factors when making judgments of singular causation. For this reason, causal strength was fixed as deterministic. The current experiment aims to understand the relationship between causal strength and causal latency and so these two factors are varied in 4 different conditions.For this experiment, causal onset is assumed to be the same in all conditions, so it should have no effect when exploring the interaction between latency and casual strength. Additionally, causal strength is varied across conditions but in every condition the target cause and the alternative cause strength's are equal. We say that the target cause is strong when it has causal strength 0.83 and weak when it has causal strength 0.5. We say that the target cause is long when the causal latency $\sim$ *Gamma*$(30, 100)$ and the alternative cause causal latency $\sim$ *Gamma*$(10, 100)$. Inversely, we say that the target cause is short when the causal latency of the target cause $\sim$ *Gamma*$(10, 100)$ and the alternative cause causal latency $\sim$ *Gamma*$(30, 100)$. Thus we set up 4 conditions on the target cause: 1. Target is weak and long, 2. Target is strong and long, 3. Target is weak and short and 4.Target is weak and short

Results And Discussion

Experiment 1

The purpose of experiment 1 was to test the hypothesis that humans integrate temporal information when making judgments of singular causation. For this experiment, as the causal latencies are fixed, the model computes the α value as 0 or 1. Further, as the causal strengths are both deterministic, the model predicts a probability of 1 for the cause with the smaller sum of causal latency and causal onset. Intuitively, since there is no probability involved, the cause which produces its effect the fastest, will always be the actual singular

cause. The collected data support this hypothesis and is summarized in the tables below alongside relevant statistical tests: A visualization is included in the appendix.

Table 1: Summary for experiment 1 Condition 1

	Short Cause	Long Cause
Mean	0.75	0.22
Median	0.8	0.1
Stdv	0.24	0.23
95%CI	$[0.68, 0.81]$	$[0.15, 0.29]$

A t-test between the means reveals a significant result: $t(94) = 11.01, p < 0.01, d = 2.25$

Table 2: Summary for experiment 1 Condition 2

	Short Cause	Long Cause
Mean	0.83	0.25
Median	0.9	0.1
Stdv	0.21	0.25
95%CI	$[0.76, 0.88]$	$[0.18, 0.32]$

A t-test between the means reveals a significant result: $t(94) = 12.15, p < 0.01, d = 2.48$

Table 3: Summary for experiment 1 Condition 3

	Short Cause	Long Cause
Mean	0.66	0.47
Median	0.8	0.5
Stdv	0.30	0.32
95%CI	$[0.57, 0.74]$	$[0.38, 0.56]$

A t-test between the means reveals a significant result: $t(94) = 3.02, p < 0.01, d = 0.61$

Table 4: Summary for experiment 1 Condition 4

	Short Cause	Long Cause
Mean	0.69	0.42
Median	0.75	0.5
Stdv	0.28	0.25
95%CI	$[0.60, 0.76]$	$[0.35, 0.49]$

A t-test between the means reveals a significant result: $t(94) = 4.80, p < 0.01, d = 0.98$

The results show that the shorter cause was always rated as having higher probability as the actual singular cause than was the longer cause. This means that the subjects were sensitive to temporal variables when making singular causation judgments. This is also inline with what the model predicted. However, it is interesting to note that even on a deterministic scenario, the subjects were still uncertain and did not consistently rate the shorter cause as being deterministic. This is contrasted to the model, which does predict a deterministic outcome. This might seem as a potential flaw of the model, however this model is not designed for deterministic scenarios, and neither it should. As we operate in the natural world, where causal strengths and causal latencies might be probabilistic, it makes sense to build a model to take this into account. Also of interest in this first experiment is the fact that the differences between the means in the first two conditions are less than the differences between the means in the last two conditions. This can be explained by the fact that in the last two conditions, a more complex computation has to be made to determine which cause is the fastest acting. Whereas in the first two conditions, since only one variable is changed, the computation is easier.

Experiment 2

The purpose of this experiment was to determine if different gamma distributions for causal latency produce a different rating of probability of singular causation. The different gamma distributions correspond to different α values. The model predicts that as α increases, the probability of the target cause being the singular cause will decrease (given deterministic causal strengths). A summary of the results is included below:

Table 5: Model predictions for experiment 2

$\alpha = 0.01$	$\alpha = 0.25$	$\alpha = 0.50$	$\alpha = 0.75$	$\alpha = 0.99$
≈ 1	0.75	0.5	0.25	≈ 0

Table 6: Behavioural results for experiment 2

	$\alpha = 0.01$	$\alpha = 0.25$	$\alpha = 0.50$
M	0.90	0.63	0.53
MD	0.90	0.7	0.5
σ	0.12	0.23	0.24
CI	$[0.85, 0.93]$	$[0.56, 0.70]$	$[0.46, 0.61]$

	$\alpha = 0.75$	$\alpha = 0.99$
M	0.32	0.09
MD	0.3	0.1
σ	0.17	0.1
CI	$[0.27, 0.37]$	$[0.06, 0.12]$

A correlation test between the model predictions and the behavioural data's mean resulted in $r = 0.99$ and $p < 0.01$ A visual is included in the appendix. As expected, the probability of the target cause being the actual cause decreases as alpha increases. This is because α is the the probability of the target cause being pre-empted by the alternative cause. These results show that subjects are able to make judgments about singular causation when causal latencies follow a probability distribution. Further, the ratings of the subjects are dependent on the gamma distributions and thus on the α value. This suggests that humans are able to form an estimate for α, or the probability that the target cause is pre-empted, and then make a judgement on the probability that the target cause is the actual cause using this information.

Experiment 3

The purpose of this experiment was to determine the interaction between causal strength and causal latencies. The model predicts that when the target cause has a longer causal latency than the alternative cause, then subjects will be less confident of the target cause when both causal strengths are strong than when both causal strengths are weak. Intuitively, we are comparing the cases when the alternative cause is acting faster but unreliably (weak causal strength) and when it is acting faster and reliably (strong causal strength). We expect the former to produce higher ratings than the latter. Conversely, when the target cause has relatively short causal latency, we expect the rating to be higher on the strong condition, since the target cause is happening quicker and reliably. The model predictions and experimental results are included below, and a visual in the appendix. Here the columns refer to the conditions on the target cause.

Table 7: Model predictions for experiment 3

weak&long	weak&short	strong&long	strong&short
0.33	0.67	0.15	0.85

Table 8: Behavioural results for experiment 3

	weak&short	strong&short
M	0.66	0.72
MD	0.7	0.8
σ	0.19	0.24
CI	$[0.60, 0.72]$	$[0.64, 0.80]$
	weak&long	strong&long
M	0.39	0.27
MD	0.35	0.2
σ	0.23	0.21
CI	$[0.32, 0.46]$	$[0.20, 0.33]$

As can be read from the table, the predicted interaction was supported by the experimental data. The interaction in the long condition was significant: $t(78) = 2.50 p < 0.02 d = 0.56$. However, in the short condition the interaction was not significant [1]. This might mean that humans are more sensitive to the case where they are presented an alternative cause that is happening faster, than when the alternative cause is happening slower.

Conclusion

In this project, we measured how people make singular causation judgments in a scenario where they observe a single instance of an effect and are given information about two potential causes. The model can be applied in fields like psychology, neuroscience and artificial intelligence to determine the signal cause of a target effect. Some possible extensions can be extending the model's ability to consider more than one alternate cause as in real-life scenarios, any outcome can have multiple competing causes. Another extension can be incorporating the model's ability to include the context of the causal judgement. The causal reasoning and required evidence to draw causal inferences would be different in a medical setting than in a legal setting. Lastly, incorporating uncertainty of human judgment into the model. Often time, an individual's personal habits, their experiences shape their reasoning. Based on that humans can often make wrong causal inferences about an outcome. Therefore, the ability to include that uncertainty would create a more nuanced model. However, through this project, we have successfully modelled a crucial component of human judgement where our time and singular causation model largely mimics human behaviour and thus can be further advanced and refined.

References

Cheng, P. W. (1997). From covariation to causation: A causal power theory. *Psychological Review*, *104*, 367–405.

Cheng, P. W., & Novick, L. R. (n.d.). Constraints and nonconstraints in causal learning: Reply to white (2005) and to luhmann and ahn (2005).

Danks, D. (2017). *The oxford handbook of causal reasoning*. Online: Oxford Academic.

J. Phillips, J. K., J. B. Luguri. (2015). Unifying morality's influence on non-moral judgments: The relevance of alternative possibilities. *Cognition*, *145*, 30–42.

N. Marchant, T. Q., & S. Chaigneau, S. (2023). *Uncertainty can explain apparent mistakes in causal reasoning.*

Pearl, J. (1985). *Bayesian networks: A model of self-activated memory for evidential reasoning* (Tech. Rep. No. CSD-850021). Los Angeles, CA: University of California, Los Angeles.

Stephan, S., Mayrhofer, R., & Waldmann, M. R. (2020). Time and singular causation—a computational model. *Cognitive Science*, *44*.

[1] This statistic was not included in the original paper

Stephan, S., & Waldmann, M. R. (2018). Preemption in singular causation judgments: A computational model. *Topics in Cognitive Science*, *10*.