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### **Title**

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### **Permalink**

<https://escholarship.org/uc/item/81f5566x>

### **Journal**

Proceedings of the Annual Meeting of the Cognitive Science Society, 43(43)

### **ISSN**

1069-7977

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### **Publication Date**

2021

Peer reviewed

# Temporal Continuity and the Judgment of Actual Causation

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## Abstract

Psychological theories of actual causation aim to characterize which of multiple causes of an event is singled out as the primary cause. We present one such theory called the continuity account of actual causation. The continuity account treats events as changes of state in continuous time and traces a sequence of stage changes backwards through time from an event to its primary cause. The account is broadly compatible with the physical process view of causation and we test it by asking people to identify the primary cause of events occurring in simple physical systems. An initial experiment confirms that root causes are more likely to be chosen as primary causes than are immediate causes. A second experiment demonstrates that root causes that have temporal continuity with the effect are preferred even when probability raising accounts would predict otherwise. The results of both experiments are consistent with the continuity account, and suggest that inferences about changes of state in continuous time may underpin an important class of actual causation judgments.

**Keywords:** causal inference; causal selection; singular causation; token causation; causal explanation

Around 5:27 pm on November 9, 1965, Martin Saltzman found himself trapped in a dark elevator roughly a quarter of a way up the Empire State building (Gelb & Rosenthal, 1965). The stopping of Saltzman's elevator was one of thousands of unexpected events that occurred that evening and all of the people involved must have wondered about the causes of these events. Subsequent accounts of the Great Northeastern Blackout often trace these events back to the tripping of a relay on line Q29BD in Ontario that triggered a cascade of failures.

Identifying the primary cause of an event (e.g. the stopping of an elevator) requires a judgment of actual causation, also known as singular or token causation (Danks, 2017). Psychologists and philosophers have explored actual causation judgments in detail and have developed formal models of actual causation, including models based on Bayesian networks (Halpern, 2016), force dynamics (Wolff & Thorstad, 2017) and mental simulation (Gerstenberg, Goodman, Lagnado, & Tenenbaum, to appear). Here we present and evaluate an account of actual causation that highlights the role of temporal continuity. On this account, the tripping of relay Q29BD was the primary cause of the elevator stopping because the tripping initiated a continuous sequence of state changes that culminated in the stopping of the elevator. Identifying this primary cause may involve two steps: the first is to assemble a set of causes (e.g. a set that includes all of the state

changes just mentioned), and the second is to select the primary cause from among this set (Stephan, Mayrhofer, & Waldmann, 2020). Our general approach has implications for both steps, but in our setting the second step (often called causal selection) is the more challenging of the two and we focus on it throughout.

Most accounts of actual causation are consistent with one of two broad views of causation: the counterfactual approach and the physical process approach (Hall, 2004). The counterfactual approach suggests that the tripping of relay Q29BD is a cause of the elevator stopping because if the relay had not tripped then the elevator would not have stopped. The physical process view suggests that the relay tripping is a cause of the elevator stopping because the two are linked by a physical process involving the transmission of force or energy. Both accounts need additional machinery in order to specify which among the many causes of an event is singled out as the primary cause. Our continuity account fits most naturally with the physical process approach and can be viewed as an attempt to bring out some implications of this approach for actual causation.

The continuity account relies on two core principles. First, the effect to be explained and its cause are both changes of state. The tripping of a relay qualifies as a candidate cause, but the steady-state setting of a relay does not. Our emphasis on state changes is consistent with the common view that causal relationships are relationships between events rather than facts or states of affairs, but Glymour et al. (2010) point out that most Bayesian accounts of actual causation ignore changes of state. State changes are embedded in continuous time, and a change that begins at a specific moment is typically the direct result of an event that occurred an instant before. Focusing on state changes therefore motivates a principle of temporal continuity that allows continuous causal sequences to be traced back in time from an effect to its primary cause. Some previous work on actual causation highlights the importance of time (Stephan et al., 2020), and our approach is highly compatible with work by Michotte and others suggesting that temporal information is often critical for identifying causes (Young & Sutherland, 2009; Davis, Bramley, & Rehder, 2020). To our knowledge, however, previous work has not explored the implications of temporal continuity in the way that we do here.

The continuity account does not aspire to capture all of

people’s intuitions about actual causation. Like the physical process approach more broadly, it is most applicable to judgments about physical rather than social systems, and does not capture cases of causation by omission (Wolff, Barbey, & Hausknecht, 2010). To us it seems likely that judgments of actual causation rely on multiple principles that resist unification under a single heading (Hall, 2004; Danks, 2017). In focusing on physical systems we aim to characterize one paradigmatic class of judgments but acknowledge that additional approaches are needed to understand actual causation in other settings.

The following sections introduce the continuity account of actual causation and present two experiments designed to test core predictions of the account. Among previous theories of actual causation a natural comparison is Spellman’s probability raising account, which proposes that the actual cause of an effect is the cause that increased its probability to the greatest extent (Spellman, 1997). Our second experiment directly compares the continuity account with the probability raising account and we find that the continuity account provides the better account of our data.

### The continuity account of actual causation

We introduce the continuity account using a scenario similar to the cover story used in the experiments. Figure 1a shows a network of particle detectors (white squares), including a special detector called the Gauge of Critical Moment (GCM). The detectors activate and turn black when they absorb a radioactive particle. Activation is transmitted across links in the network, and a detector activates if all of its input detectors are active.

Although we focus on processes unfolding in continuous time, it will be convenient to divide up the temporal dimension into intervals brief enough that at most one event occurs per interval. The effect to be explained is an event that happens within one of these intervals: for example, the GCM’s change of state from inactive to active. The continuity account suggests that there can be at most one event that triggered a continuous sequence of state changes that culminated in the effect, and proposes that this triggering event (if it exists) is singled out as the primary cause. The triggering event can be identified using a procedure that starts with the effect and steps backwards through time until the primary cause is identified.

The immediate cause of the GCM’s activation must be an event that took place in the interval preceding the activation. In Figure 1a, the event immediately preceding the GCM’s activation was the activation of  $I_C$ . Having identified this immediate cause, we then step backwards and identify the immediate cause of this event, and so on. The procedure terminates once we arrive at an event that has no immediate cause within the system of interest, and this cause is selected as the primary cause of the effect. In Figure 1a the primary cause is the activation of  $R_C$ .

In most cases, the backward-tracing procedure just de-

scribed will identify a single primary cause of the effect. If the effect has no immediate cause within the system, then no primary cause will be identified. Because there is at most one event per interval, the procedure can never identify more than one primary cause.

The assumption that at most one event occurs per interval follows from the idea that there can be no coincidences in continuous time. If the temporal dimension is continuous it is exceedingly improbable that two events would occur at precisely the same time — in technical terms this kind of coincidence can be described as a “measure zero” possibility. If there are no coincidences, then slicing the time dimension sufficiently finely will ensure that there is at most one event per interval.

The boundaries of the causal system under consideration will depend on context. For simplicity, our discussion of Figure 1a has focused only on events internal to the particle detection network, and has attempted to characterize which of these events is best viewed as the primary cause of the GCM’s activation. The particle detection network, however, could also be considered part of a broader causal system that includes both the network and the network’s surroundings. For example, if the network is embedded in an apparatus for carbon dating, then the spontaneous decay of a carbon atom could be identified as the immediate cause of  $R_C$  and the primary cause of the GCM’s activation.

### Experiments

We developed two experiments to test the continuity account of actual causation just presented. Both experiments asked participants to imagine that they worked in a nuclear control room, and that their job was to monitor networks of particle detectors. Participants were told that “for each activation sequence that you see, your job is to decide what caused the activation of the GCM.”

Both experiments included chains in which the GCM received input from one detector, and dual branch networks such as Figure 1a in which the GCM received input from two detectors. For chains, the continuity account predicts that participants will tend to choose the root cause of the effect (i.e. the detector that initiates the activation sequence) rather than the immediate cause (the detector that immediately precedes the GCM). For dual branch networks, the continuity account predicts that participants will tend to choose the root cause on the branch whose activation is temporally continuous with the activation of the GCM. We refer to this branch as the continuous branch, and refer to the root cause and the immediate cause on this branch as  $R_C$  and  $I_C$  respectively, where the subscript denotes “continuous.” We refer to the other branch as the “delayed branch,” and use  $R_D$  and  $I_D$  for the root and immediate causes on this branch. The “delay” in this naming scheme refers to the delay that occurs between  $I_D$  and the activation of the GCM. It is convenient to use the same labels for both detectors and events: for example,  $R_C$  will be used to denote both a detector on the continuous branch and the acti-

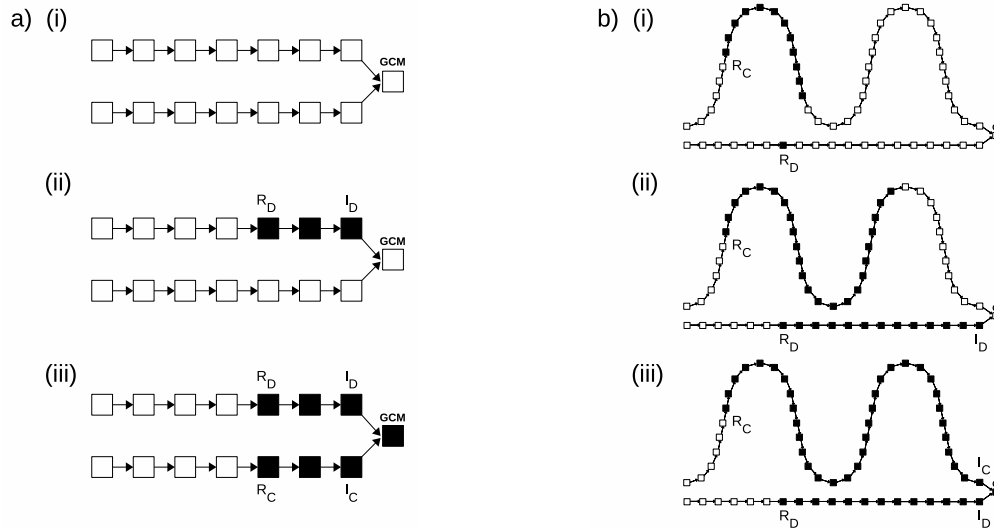


Figure 1: (a)(i) A network of particle detectors. All components are initially inactive. (ii) Detector  $R_D$  activates and activity propagates along the top branch. (iii) Detector  $R_C$  activates and ultimately triggers the activation of the GCM, which activates only when both  $I_D$  and  $I_C$  are active. (b) An activation sequence in which (i) the top branch activates first, (ii) activation starts and finishes along the bottom branch while activation continues along the top branch, and (iii) the top branch completes its activation and ultimately triggers the GCM.

vation of this detector. This flexible use of notation, however, does not imply that actual causation involves a relationship between objects rather than events.

Our presentation of the experiments focuses on two theoretical accounts: the continuity account and the probability raising account. For completeness, though, we first discuss the counterfactual and physical process accounts of causation. Both accounts can be formulated in different ways. Here we present what we take to be the default version of each account and argue that neither makes clear predictions about our task. The General Discussion considers ways in which these default formulations can be adjusted to better account for our data.

The counterfactual account makes no clear prediction about whether root causes or immediate causes should take priority. For a chain network, if the root cause had not occurred, the effect would not have occurred, but if the immediate cause had not occurred, the effect would also not have occurred. If we consider dual branch networks and restrict attention to root causes only, then the account makes no clear prediction about whether  $R_C$  should be preferred to  $R_D$ . If  $R_C$  had not occurred, then the effect would not have occurred, and likewise for  $R_D$ .

The physical process account is similarly inconclusive. Both root and immediate causes are connected by a physical process to the effect, and there seems to be no clear reason for preferring one to the another. If we focus only on root causes, again there is no clear preference between  $R_C$  and  $R_D$ . After the activation of  $I_D$ , one can think of this detector as continuously sending activation towards the GCM which

only “unlocks” the detector once  $I_C$  is also active. It follows that both  $R_C$  and  $R_D$  are connected to the effect by physical processes.

Because both of these general accounts of causation make no clear predictions, it seemed possible that people’s inferences about the networks in our experiments would be highly variable and would reveal no clear trends. Our first experiment therefore explored simple cases analogous to the example in Figure 1a with the goal of establishing whether the basic experimental procedure was viable. The second experiment focused on more elaborate dual-branch cases that aimed to distinguish between the continuity account and Spellman’s probability raising account (Spellman, 1997).

## Experiment 1

Experiment 1 included both causal chains and dual branch networks. For all of the dual branch stimuli,  $I_D$  occurred before  $R_C$  (Figure 2a) which means that the delayed branch completed its activation before the continuous branch began to activate. The probability raising and continuity accounts both predict that root causes should be preferred to immediate causes, and that for dual branch networks  $R_C$  should be preferred to  $R_D$ . The primary purpose of the experiment was to test both predictions.

To apply the probability raising account to the dual branch stimuli, let  $b$  (for base rate) be the probability of activation along along a given branch during a given trial. At the start of a dual branch trial, the probability that the GCM will activate is  $b^2$ , because independent activations are required along both branches. After  $R_D$  the probability increases to  $b$ , and after  $R_C$  the probability increases to 1. The two probability

increments are therefore  $b - b^2$  and  $1 - b$ , and it is straightforward to show that  $b - b^2 \leq 1 - b$  for all  $b$ , with equality attained only when  $b = 1$ . As a result, the probability raising account selects  $R_C$  if  $b < 1$ , and is indifferent between  $R_C$  and  $R_D$  if  $b = 1$ .

For each dual branch network shown, activation eventually occurred along each branch, and some participants may have therefore assumed that  $b = 1$ . Most participants, however, probably implicitly assumed that  $b < 1$ , which allows for the possibility of trials during which a branch never activated. We will therefore assume that  $b < 1$  when considering the predictions of the probability raising account.

**Participants.** 30 participants were recruited via Amazon Mechanical Turk and paid \$3 for an 18 minute experiment.

**Materials.** The experiment used a customized interface built using the jsPsych library (De Leeuw, 2015). For all networks presented, participants clicked a “Run” button to observe an activation sequence. The first event in the sequence (i.e. the first change of state) always took place 5 seconds after the Run button was clicked, and the delay between successive activations along a chain of detectors was set to 100 ms. After the final event in a sequence (i.e. the activation of the GCM), all detectors became clickable after a delay of 1 second. Clicking on a detector turned its border red, and at most one detector could be selected at any time. After a sequence completed, participants could view it again if they wished by clicking a “Run again” button.

**Design.** The experiment included activation sequences over 15 networks (3 chains and 12 dual branch networks). Excluding the GCM, each chain and each branch of each dual network had 7 detectors. Within each dual branch sequence, the root causes on the two branches ( $R_D$  and  $R_C$ ) were equidistant from the GCM, but these distances varied across sequences. We refer to the sequences as *short*, *medium* or *long* based on the distance between the root causes and the GCM. Excluding the GCM, the short, medium and long sequences showed 1, 3 and 6 active detectors respectively per branch at the end of the sequence. The sequence in Figure 1a is a medium dual branch sequence (3 active detectors in each branch excluding the GCM).

Each dual branch sequence (short, medium and long) came in four versions. The *state* version showed the delayed branch (including detectors  $R_D$  and  $I_D$ ) as active from the very beginning of the sequence. The activation of this branch was therefore presented as having occurred at some indefinite time in the past, resulting in a steady state of activation. The three *event* versions all showed  $R_D$  activating 5 seconds into the sequence, and had delays of 2, 4 and 6 seconds between the activation of  $I_D$  and  $R_C$ . The predictions of the continuity account are unaffected by the delay, but we tested different delays just in case this variable affected people’s responses. The continuity account also makes the same prediction about both state and event sequences, but we anticipated that the state sequences might make participants especially likely to choose  $R_C$  over  $R_D$ .

**Procedure.** Participants first read instructions which introduced the task and included examples of a chain and a dual branch network. They then answered three questions about the task and the detectors, and were sent back to read the instructions again if they answered incorrectly. They continued cycling through the instructions and the test questions until they answered all questions correctly.

The 15 activation sequences in the experiment proper were presented in random order. For dual branch sequences, the vertical position of the delayed branch was also randomized (Figure 1 shows the delayed branch on the top rather than the bottom). The orientation of the network (GCM on the left or the right) was randomized within participants. The prompt after each sequence was “In this sequence what caused the activation of the GCM? Respond by clicking on a detector,” and participants were required to choose a single detector in response.

**Results.** Network orientation (left or right) and position of the delay branch (top or bottom) had no apparent effect, and we therefore collapse across these variables. Figure 2 summarizes the results for short, medium and long dual-branch sequences. The delay between  $I_D$  and  $R_C$  had no significant effect, and Figure 2 combines results for all three delays.

Although  $R_D$ ,  $I_D$ ,  $R_C$  and  $I_C$  all qualify as causes of the effect, Figure 2 suggests that  $R_C$  tends to be singled out as the primary cause. This result can be separated into two general conclusions. First, participants were more likely to choose root causes than immediate causes. Root and immediate causes were identical for the short sequences and therefore cannot be distinguished, but the results for medium and long sequences reveal a preference for root causes. The second conclusion is that for dual branch sequences, the root cause on the continuous branch ( $R_C$ ) is preferred to the root cause on the delayed branch ( $R_D$ ). As explained earlier, this result is consistent with both the continuity and probability raising accounts.

To support these two conclusions we ran two Bayesian mixed effects regression models using the *brms* package in R (Bürkner, 2017) with default priors.<sup>1</sup> For the first model, each response was coded as root or immediate, and all responses that could not be classified in this way (including all responses for short sequences) were discarded. We then ran a logistic regression in which *stativity* (i.e. state vs event) and network length (long vs medium) were included as predictors of a binary dependent variable (root vs immediate). We included a random intercept for participant to allow for statistical dependencies between multiple responses from the same participant. The posterior mean of the intercept variable was 11.0 and the 95% credible interval was [2.6, 28.7], which supports the conclusion that root causes were chosen more often than immediate causes. The credible intervals for the other coefficients both included 0 (state: -2.7, [-9.3, 1.1];

<sup>1</sup>By default *brms* uses improper flat priors on the coefficients of fixed effects, and a half student-t prior with 3 degrees of freedom, a location of 0 and a scale of 2.5 on the intercept and on the standard deviations of the random effects.

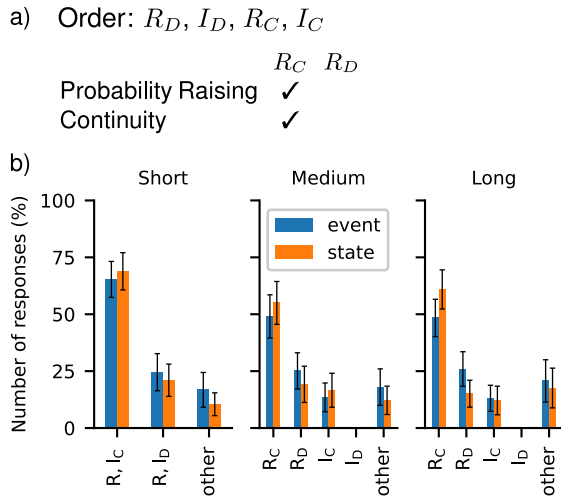


Figure 2: Experiment 1. (a) Order of the four key events for dual branch sequences. The probability raising and continuity accounts both single out  $R_C$  as the primary cause. (b) Responses for dual branch sequences. The delayed branch was either inactive (event sequences) or active (state sequences) at the start of the sequence. Error bars show the standard error of the mean estimated using 100 bootstrap samples.

long: 2.1,  $[-1.7, 9.0]$ ), suggesting that the preference for root causes was not strongly affected by either stativity or network length.

The second analysis was very similar except that the binary dependent variable now indicated whether participants chose a cause on the continuous branch or the delayed branch. The posterior mean of the intercept variable was 1.7 but the 95% credible interval included zero ( $[-0.06, 3.7]$ ), suggesting only weak support for the conclusion that the continuous branch was chosen more often than the delayed branch. The credible intervals for the state variable excluded 0 (1.0,  $[0.05, 1.9]$ ), suggesting that state sequences led to a stronger preference for the continuous branch than did event sequences. Credible intervals for both length variables included 0 (medium: 0.4,  $[-0.5, 1.4]$ ; long: 0.5,  $[-0.5, 1.4]$ , where the reference level is small), suggesting that the preference for the continuous branch was not strongly affected by network length.

It is notable that people’s judgments are largely unaffected by the delay between  $I_D$  and  $R_C$  and the activation length of each sequence (short, medium or long). Both of these variables affect the time that elapses between the root causes and the effect, and our results suggest that people’s judgments are not exquisitely sensitive to this sort of variation.

## Experiment 2

The results of Experiment 1 are broadly consistent with both the continuity and probability raising accounts, and the goal of Experiment 2 was to distinguish between these accounts. The two make different predictions for dual branch sequences in which the delayed branch activates after activation has already begun along the continuous branch. One

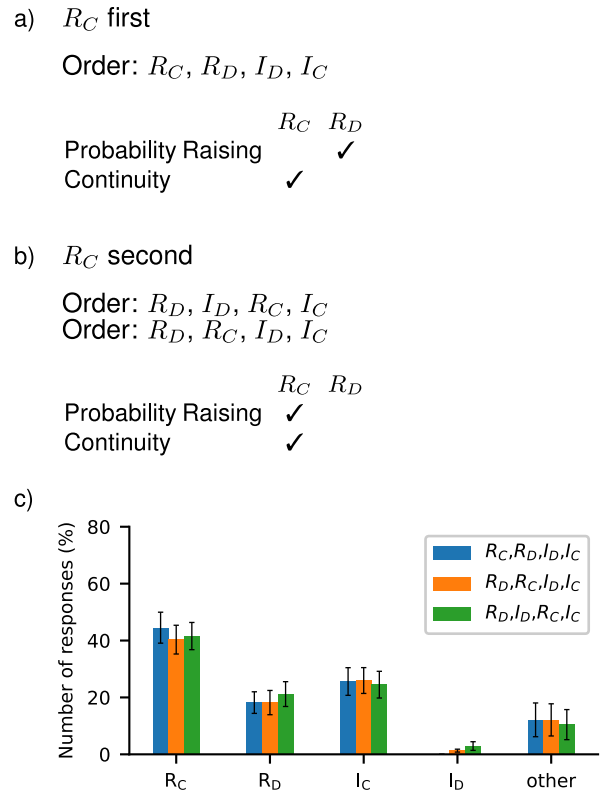


Figure 3: Experiment 2. (a) The probability raising and continuity accounts make different predictions for dual branch sequences in which  $R_C$  occurs before  $R_D$ . (b) The two accounts agree when  $R_C$  occurs after  $R_D$ . (c) Responses for the three possible orders of the key events.

such sequence is shown in Figure 1b. In cases like this, the continuity account still treats  $R_C$  as the primary cause, but the probability raising account now treats  $R_D$  as the primary cause. For dual branch stimuli, the argument presented earlier shows that the probability raising account identifies the second of the two root causes (i.e.  $R_C$  in Figure 1a and  $R_D$  in Figure 1b) as the primary cause if the base rate  $b < 1$ . If  $b = 1$  the account is unable to select any event as the primary cause.

The materials and procedure for Experiment 2 are similar to those for Experiment 1 and we will highlight only the few points of difference.

**Participants.** 100 participants were recruited via Amazon Mechanical Turk and paid \$1 for a 6 minute experiment.

**Design.** The experiment included one chain sequence and 6 dual branch sequences. Each dual branch included one straight branch and a longer curved branch, as shown in Figure 1b. The dual branch sequences included the events  $R_C$ ,  $R_D$ ,  $I_C$ , and  $I_D$  in three different orders shown in Figures 3a and 3b. The sequence in Figure 1b is an instance of the order in Figure 3a, because the activation on the delayed branch starts ( $R_D$ ) and finishes ( $I_D$ ) while activation is propagating along the continuous branch. The 6 dual branch sequences included 2 variants of each of the three orders. In one variant

$R_C$  belonged to the curved branch (as in Figure 1b) and in the other  $R_C$  belonged to the straight branch.

**Procedure.** The position of the curved branch (top or bottom) was randomized. The presentation order of the activation sequences and the orientation (GCM on the left or right) were randomized as for Experiment 1.

**Results.** Network orientation (left or right) and position of the curved branch (top or bottom) had no effect and we collapsed across these variables. Figure 3c summarizes the results for the three orders listed in Figures 3a and 3b. Consistent with Experiment 1,  $R_C$  is preferred over  $R_D$  given the order  $\{R_D, I_D, R_C\}$ , and a similar effect was found for the  $\{R_D, R_C, I_D\}$  sequences. The critical finding is that  $R_C$  is also preferred for the  $\{R_C, R_D, I_D\}$  sequences, even though the probability raising account makes the opposite prediction. When referring to the orders we have dropped the fourth event because this event is always  $I_C$ .

To further analyze the data we used mixed effects models analogous to the two described for Experiment 1. The first analysis used a logistic regression in which order (i.e. the temporal order of the key events) was included as a predictor of a binary dependent variable (root or immediate). The posterior mean of the intercept variable was 5.0 and the 95% credible interval [2.0, 8.9], which supports the conclusion that root causes were chosen more often than immediate causes. The credible intervals for both order variables included 0 ( $\{R_D, R_C, I_D\}$ : -0.2, [-1.5, 1.2];  $\{R_D, I_D, R_C\}$ : -0.4 [-1.7, 0.9], where the reference level was  $\{R_C, R_D, I_D\}$ ), suggesting that the preference for root causes was fairly consistent across the three orders.

The second and more critical analysis used a binary dependent variable that indicated whether participants chose a cause on the continuous branch or the delayed branch. The posterior mean of the intercept variable was 1.9 (95% credible interval [1.3, 2.5]), indicating that the continuous branch was chosen more often than the delayed branch. The credible intervals for both order variables included 0 ( $\{R_D, R_C, I_D\}$ : -0.4, [-0.9, 0.1] ;  $\{R_D, I_D, R_C\}$ : -0.5, [-1, 0.1]) suggesting that the preference for the continuous branch was of similar strength across all three orders.

Relative to Experiment 1, the frequency with which  $I_C$  is chosen has increased in Experiment 2. This difference may reflect the increased difficulty of Experiment 2. When both branches of a dual branch structure are simultaneously active, keeping track of both root causes and the order in which they occurred is relatively challenging, which may lead some participants to fall back on the simple strategy of choosing the immediate cause that directly precedes the effect.

## General discussion

We presented an account of actual causation that highlights the role of temporal continuity and described experiments that support two of its predictions. First, people tend to identify root causes rather than immediate causes as the primary cause of an effect. Second, when an effect is produced by the

convergence of multiple causal pathways, participants tend to identify the pathway that has temporal continuity with the effect as the primary cause.

Because the continuity account considers changes of state in continuous time, it can exploit a “no coincidences” principle to identify a single primary cause of an effect. This principle applies broadly to causation in physical systems, but is less applicable to social scenarios such as voting scenarios (Livengood, 2013). Exceptions to the “no coincidences” principle are possible even for physical systems, and the continuity account can capture the illusory perceptions of actual causation that arise in some such cases (Thorstad & Wolff, 2016). For example, an 11 year old boy whacked a stick against a telephone pole at the instant at which the Great Northeastern Blackout hit his town, and ran home terrified that he had caused the power outage (Gelb & Rosenthal, 1965).

The continuity account highlights ideas such as the “no coincidences” principle that go beyond default formulations of the counterfactual and physical process accounts of causation. Key predictions of the account, however, can be reconstructed within both of these general frameworks. The counterfactual account can exploit fine-grained temporal information if each event is supplemented with a timestamp. If the effect to be explained in Figure 1 is “the activation of the GCM at exactly 10 sec after the start of the trial,” then the counterfactual account suggests that  $R_C$  is a cause of the effect but  $R_D$  is not, because if  $R_D$  had been different (e.g. by occurring slightly earlier or later) then the effect would still have occurred at the same instant.

As suggested earlier, standard formulations of the physical process view treat both  $R_C$  and  $R_D$  as causes of the effect in Figure 1. Our data are consistent with the possibility that people view  $R_C$  and  $R_D$  as equally valid causes, but pick  $R_C$  when forced to break the tie for reasons that may be relatively superficial. For example, perhaps  $R_C$  is preferred because the activation on the continuous branch is continuous both in space and time, and therefore more prototypical of a causal process than the activation along the delay branch. We have planned a future study that asks whether the preference for  $R_C$  over  $R_D$  is fundamental or superficial using a task in which participants rank multiple candidate causes in order of importance, and in which ties are permitted.

The experimental paradigm we have developed can easily be extended to a more diverse set of causal systems. For example, by introducing preventive causal links, we can explore case of double prevention (e.g. cases where an active detector that would have prevented the activation of the GCM is itself inactivated by another detector). Glymour et al. (2010) point out that current work on actual causation is based largely on discussions of an “infinitesimal fraction” of the set of possible cases, but our paradigm can potentially be used to carry out a comprehensive survey of judgments about actual causation.

## Acknowledgments

AF carried out this work at the University of Melbourne as part of a Master's degree at the École Normale Supérieure.

Code and data are available at <https://github.com/A-Fermo/continuity-cogsci21>

## References

- Bürkner, P.-C. (2017). brms: An R package for Bayesian multilevel models using Stan. *Journal of statistical software*, 80(1), 1–28.
- Danks, D. (2017). Singular causation. *The Oxford Handbook of Causal Reasoning*, 201–215.
- Davis, Z. J., Bramley, N., & Rehder, B. (2020). The paradox of time in dynamic causal systems. In *Proceedings of the 42nd annual conference of the Cognitive Science Society*.
- De Leeuw, J. R. (2015). jsPsych: A JavaScript library for creating behavioral experiments in a web browser. *Behavior research methods*, 47(1), 1–12.
- Gelb, A., & Rosenthal, A. M. (1965). *The night the lights went out*. New York: New American Library.
- Gerstenberg, T., Goodman, N., Lagnado, D., & Tenenbaum, J. (to appear). A counterfactual simulation model of causal judgment. *Psychological Review*.
- Glymour, C., Danks, D., Glymour, B., Eberhardt, F., Ramsey, J., Scheines, R., ... Zhang, J. (2010). Actual causation: a stone soup essay. *Synthese*, 175(2), 169–192.
- Hall, N. (2004). Two concepts of causation. In J. Collins, N. Hall, & L. Paul (Eds.), *Causation and counterfactuals* (pp. 225–276). MIT Press.
- Halpern, J. Y. (2016). *Actual causality*. MIT Press.
- Livengood, J. (2013). Actual causation and simple voting scenarios. *Noûs*, 47(2), 316–345.
- Spellman, B. A. (1997). Crediting causality. *Journal of Experimental Psychology: General*, 126(4), 323–348.
- Stephan, S., Mayrhofer, R., & Waldmann, M. R. (2020). Time and singular causation—a computational model. *Cognitive Science*, 44(7), e12871.
- Thorstad, R., & Wolff, P. (2016). What causal illusions might tell us about the identification of causes. In *Proceedings of the 38th annual conference of the Cognitive Science Society*.
- Wolff, P., Barbey, A. K., & Hausknecht, M. (2010). For want of a nail: How absences cause events. *Journal of Experimental Psychology: General*, 139(2), 191–221.
- Wolff, P., & Thorstad, R. (2017). Force dynamics. *The Oxford Handbook of Causal Reasoning*, 147.
- Young, M. E., & Sutherland, S. (2009). The spatiotemporal distinctiveness of direct causation. *Psychonomic Bulletin & Review*, 16(4), 729–735.