Actual Causes as Changes of State in Continuous Time

Anonymous CogSci submission

Abstract

Keywords:

Introduction

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 virtually 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 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 these judgments in detail and developed formal models of actual causation, including models based on Bayesian networks (), force dynamics (?, ?) and physical simulation (?, ?). 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 is the main cause of the elevator stopping because the tripping initiated a continuous sequence of state changes that culminated in the stopping of the elevator.

Most accounts of actual causation are consistent with one of two broad views of causation: the counterfactual approach and the physical process approach. 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 main 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, it assumes that 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 Bayes net 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 main cause. Some previous work on actual causation highlights the importance of time (Young & Sutherland, 2009; Stephan, Mayrhofer, & Waldmann, 2020), but to our knowledge previous work has not explored the implications of temporal continuity in the way 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 the most 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

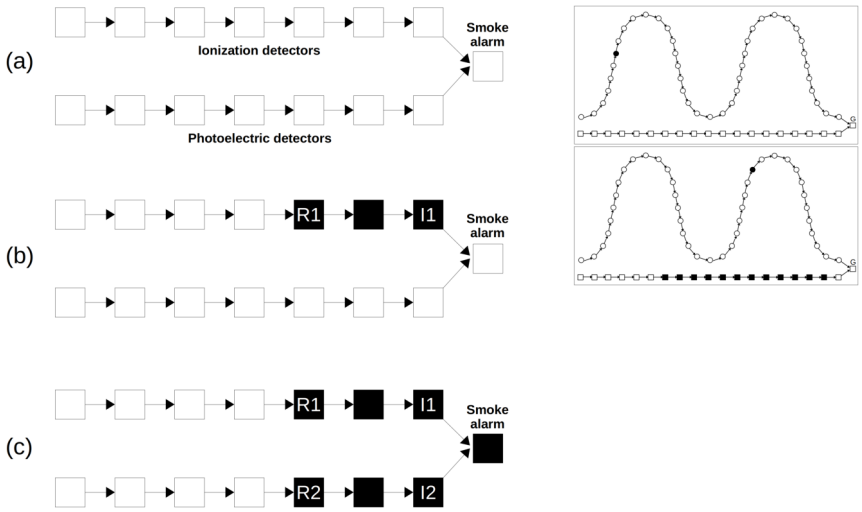

Figure 1: (a)(i) A smoke alarm connected to a chain of ionization detectors (top branch) and a chain of photoelectric detectors (bottom branch). All components are initially inactive (ii) Detector R_D activates and activity propagates along the top branch. (iii) Detector R_C activates and ultimately triggers the activation of the alarm, 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 smoke alarm.

(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 there is at most one event 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. Given this setup, our theory can be formulated using a procedure that starts with the effect and steps backwards through time until the actual cause of the effect 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 declared to be the main cause of the effect. In Figure 1a the main cause is the activation of R_C.

In most cases, the backward-tracing procedure just described will identify a single main cause of the effect. If the effect has no immediate cause within the system, then no main cause will be identified. Because there is at most one event per interval, the procedure can never identify more than one main cause.

The assumption that at most one event occurs per interval follows from the idea that there are no coincidences. In continuous time it is exceedingly improbable that two events would occur at exactly 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 attemped to characterize which of these events is best viewed as the main 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 will probably be identified as the immediate cause of R_C and the main 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 activation 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 smoke detector example in Figure 1 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.

Participants. 30 participants were recruited via Amazon Mechanical Turk and paid XXX for a YYY minute experiment.

Materials. The experiment used a customized interface built using the jsPsych library (?, ?). 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. 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 chain sequences showed 1, 3 and 6 active detectors respectively at the end of the sequence. 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. The sequence in Figure 1a is a medium dual branch sequence (3 active detectors in each branch excluding the GCM), and short and long versions had 1 and 6 active detectors respectively per branch.

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.

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 main 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 bet distinguished, but the results for medium and long sequences reveal a preference for root causes. STATISTICAL ANALYSIS.

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). This result is consistent with the continuity account but also consistent with the probability raising account, because R_C guarantees the occurrence of the effect but R_D does not.

Figure 1 suggests that event and state sequences lead to similar patterns of responses. The continuity account makes the same prediction for both kinds of sequences, but we anticipated that the event sequences might make participants especially likely to choose R_C over R_D. If this effect exists, our data suggest that it is relatively small.

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.

Thus far we have focused on dual branch sequences, and the chain sequences are of interest primarily because they provide additional evidence of a preference for root causes. NUMBERS HERE

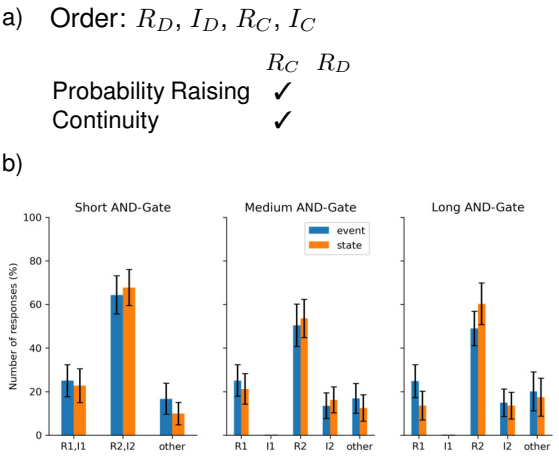

Figure 2: Experiment 1. (a) Order of the four key events for dual branch sequences. The probability raising and continuity accounts both single out R_C as the main cause. (b) Responses for dual branch sequences. The delayed branch was either inactive (event sequences) or active (state sequences) at the start of the sequence.

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 such sequence is shown in Figure 1b. In cases like this, R_D guarantees the occurrence of the effect, and therefore qualifies as the primary cause according to the probability raising account. The continuity account, however, still treats R_C as the primary cause.

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

Participants. 100 participants were recruited via Amazon Mechanical Turk and paid XXX for a YYY minute experiment. Two did not submit a valid completion code, leaving 98 sets of responses for analysis.

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 bot-

a) R_C first

Order: R_C, R_D, I_D, I_C

	R_C	R_D
Probability Raising		✓
Continuity	✓	

b) R_C second

Order: R_D, I_D, R_C, I_C
Order: R_D, R_C, I_D, I_C

	R_C	R_D
Probability Raising	✓	
Continuity	✓	

c)

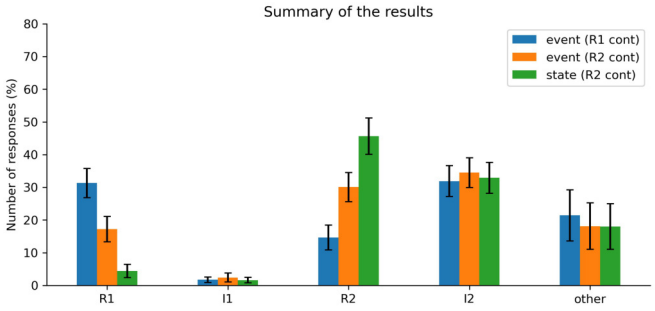

Figure 3: Experiment 2. (a) The probability raising and continuity accounts make different predictions for dual branch sequences in which R_C occurs before R_D. (b) The two accounts agree when R_C occurs after R_D. (c) Responses for the three possible orders of the key events.

tom) 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 $\{R_D,I_D,R_C,I_C\}$, and a similar (but weaker) effect was found for the $\{R_D,R_C,I_D,I_C\}$ sequences. The critical finding is that R_C is also preferred for the $\{R_C,R_D,I_D,I_C\}$ sequences, even though the probability raising account makes the opposite prediction. STATISTICS HERE.

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 fo choosing the immediate cause that directly precedes the effect.

General discussion

Outline:

I. Summary and implications of the results

II. To say why these results cannot really be explained by the current theories of causation and why our theory can better account for them. I don't know if I should talk more about CF and PP accounts than I do in introduction, like saying something like that is probably not useful:

[*According to the CF account, the general idea is that an event A is said to be a cause of a distinct event B if the occurrence of A makes a difference in the occurrence of B. More precisely A is a cause of B if and only if A and B are true, and A hadn't occurred B wouldn't have occurred. This interpretation calls upon counterfactual scenario or possible worlds where the presumed cause of the effect is removed from the system while all other relevant factors are kept as unchanged as possible compared to the actual world. According to us the main problem of this interpretation is that its best models explicitely rejects the idea of grounding causation on temporal information. According to the PP account, an event A causes B when there is a physical connection between them. In one of its latest and probably most convincing formulation, this theory distinguishes two things : causal process and causal interaction (i.e. causation). A causal process is a physical process involving an object which conserves a certain quantity, like mass-energy, accross space and time. A causal interaction or causation is an exchange of that conserved quantity. However the theory goes sometimes against some of our best causal intuitions and to makes implicitely use of the CF analysis to explain our judgements: if I hold the head of my ennemy under water and make him die, I'm not genuinely the cause of his death; rather I'm actually preventing the possibility of a genuine causation which is breathing oxygen in order to live.*]

Or if I do have to explicit both theories I should insist on what the predictions of both theories are for our experiments and why they cannot explain people's intuition (the role of time).

III. To insist, maybe, on the work of Glymour where our project in part originated: explaining his caveat about treating events as pure propositions. To explain how our experiments extend this idea and insist more on the role of changes in continuous time. Maybe we should present some insights about how we could formalize further the model? How we could use formal tools like Graphical models?

IV. To present further experiments that would be interesting to make (with loops, cases of prevention, etc.)

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