Temporal Binding and Internal Clocks: Is Clock Slowing General or Specific?

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Abstract

The perception of time is distorted by many factors, but is it possible that causality would affect our perception of time? We investigate timing changes in the temporal binding effect, which refers to a subjective shortening of the interval between actions and their outcomes. Two experiments investigated whether binding may be due to variations in the rate of an internal clock. Specifically, we asked whether clock processes in binding reflect a general timing system, or a dedicated clock unique to causal sequences. We developed a novel experimental paradigm in which participants made temporal judgments of either causal and non causal intervals, or the duration of an event embedded within that interval. While we replicated the temporal binding effect, we found no evidence for commensurate changes to time perception of the embedded event, suggesting that temporal binding is effected by changes in a specific and dedicated, rather than a general clock system.

Keywords: temporal binding; internal clock models; motor-sensory recalibration; causality; time perception

Temporal binding refers to the perceptual attraction of actions to their effects (Haggard, Clark & Kalogeras, 2002). When an action triggers a causal outcome, the action is perceived to occur later, and its outcome earlier, than two unrelated events. Temporal binding can be interpreted as bi-directional constraint of Bayesian causal inference (Eagleman & Holcombe, 2002): The closer together two events occur in time, the more likely they will be judged as causally related (e.g. Buehner, 2005). Consequently, it follows that if two events are known to be causally related, they are also more likely to be contiguous in time.

Early experiments (Haggard *et al*, 2002) on temporal binding used the Libet clock method (Libet, Gleason, Wright & Pearl, 1983), in which participants watch a fast-moving clock hand (1 rotation every 2560ms) while experiencing different events. The participant's has to report the hand position at the time when she perceives a target event. Judgment errors derived over repeated trials are then used as a proxy measure of event awareness. Using this method, Haggard *et al*. found a systematic shift in judgment errors for causal actions (key presses), which triggered an outcome (tone) after 250ms. More specifically, participants showed *delayed* awareness of their causal action, and *early* awareness of its consequence, relative to single-event judgment errors. In other words, actions and outcomes mutually attracted each other in subjective awareness. This *temporal binding* effect did not occur when participants reported the times of two unrelated events.

Studies using the stimulus anticipation method (SAM) have replicated and expanded upon the temporal binding effect (Buehner & Humphreys, 2009; Buehner, 2012). In the SAM, participants have to press a key in anticipation of a target event. A series of studies using the SAM has repeatedly demonstrated early anticipation of target events that were triggered by a causal relation, compared to targets that were equally predictable, but were merely associated with the predictor, rather than caused by it. Specifically, Buehner and Humphreys (2009) found that it is not sufficient for an intentional action to be followed by the target – the action has to cause it. Furthermore, even a non-intentional mechanical cause that triggers an outcome results in binding (Buehner, 2012). Thus, causality is both necessary and sufficient to bind events together in subjective time.

However, temporal binding occurs not only in the form of shifts in the perception of events delineating an interval, but also manifests itself via direct distortions of time perception. For example, Humphreys and Buehner (2010) found verbal estimates of intervals separating causal actions from their outcomes to be reliably lower than estimates of intervals separating two unrelated events. These changes to time perception are not merely based on post-perceptual judgment biases, but also occur when participants reproduce the experienced interval (by holding down a key for the duration of the experienced interval; Humphreys & Buehner, 2010), or when they compare target intervals to standards in a method of constant stimuli (Nolden, Haering & Kiesel, 2012). Thus, temporal binding manifests itself both via shifts in event perception as well as a direct shortening of experienced time.

Eagleman and Holcombe (2002) suggest that temporal binding arises due to a recalibration of sensory streams: Differences in processing latencies associated with different modalities are overcome by realigning sensory streams, thus ensuring a unitary percept. Because motor acts usually produce causal outcomes immediately, a delay between action and outcome forces a recalibration of the system. A short delay between an action and its outcome, it is argued, can be adapted to, thus realigning the perceptual-motor system to bring action and outcome closer together in time.

In contrast, temporal binding could also arise due to changes in time perception. Our sense of time is distorted by many factors, such as arousal (Droit-Volet & Meck, 2007). These distortions are typically explained by cognitive models (commonly referred to as 'internal clock' models), such as scalar expectancy theory (SET: Gibbon, Church & Meck, 1984). Internal clock models contain a pacemaker-accumulator process that represents perceived durations: A pacemaker emits pulses at rate *r*, which are counted in an accumulator; changes to *r* affect temporal judgments, such that decreases and increases in *r* result in respectively fewer

and more pulses accumulated in a given interval *I*. Consequently, increases and decreases in *r* will lead to changes in temporal perception of *I*. Most work on internal clock models, however, is generally of a hypothetical nature. Few studies have attempted to find empirical support for changes in *r*. However, Wearden, Edwards, Fakhri and Percival (1998), found that auditory stimuli are perceived as longer then visual stimuli, and, more importantly, that regressions of subjective over objective durations yielded a higher slope for auditory compared to visual stimuli. In other words, the difference between judgments in the two modalities grew as a function of duration, as would be expected by differences in *r*. Might it be possible that changes in the causal nature of event sequences likewise lead to modulation of pacemaker speed whereby causal intervals are perceived as shorter due to a slower *r*? To date, there exists only one study that directly investigated this possibility (Wenke & Haggard, 2009).

Wenke and Haggard (2009) combined the temporal binding procedure with a temporal discrimination paradigm: In a causal condition, participants pressed a key to generate a tone after 600, 800 or 1000 ms; in a corresponding non-causal condition, their finger was passively pulled down by a motor, which was followed by a tone – the computer scheduled both events, making it obvious that there was no causal connection between the passive movement and the tone. In addition, participants experienced two electric shocks administered via electrodes on their fingers. On a given trial, participants were prompted to either report whether the shocks were successive or simultaneous, or to estimate the duration of the action/movement – tone interval. In addition to replicating the binding effect, Wenke and Haggard found that simultaneity detection on causal trials was poorer (i.e. higher thresholds) compared to noncausal trials. This is what would be predicted if temporal binding is effected via a slowing of the internal clock: a slowing of *r* lengthens the period between pulses, increasing the likelihood that two sequential shocks fall into the same period, which in turn leads to higher discrimination thresholds.

However, considering the causal structure of Wenke and Haggard's (2009) design reveals that we have to be careful in interpreting their results. Because the shocks always occurred after the action or passive movement and never before it, they would have been subject to temporal binding in the causal conditions, just as much as the tone. This means that the first or both shocks would subjectively appear closer to the action. Thus, higher thresholds in the causal condition may not have resulted from a slower *r*, but could simply reflect temporal binding of shock(s) to key press via sensory realignment. Consequently, it remains unclear whether binding reflects changes to *r*.

We set out to investigate more carefully whether temporal binding might implicate changes to *r*. It is important to note that changes to internal clock processes in temporal binding may manifest in two distinct ways: First, temporal binding might reflect a general slowing of the timing system; secondly binding might only affect clock processes that are specific to the action-outcome interval. According to the first possibility, a slowing of a general all-purpose clock would result in changes to *any* stimulus presented simultaneously with the interval. According to the latter, effects of clock slowing will be limited only to the causal interval. Therefore, a key question we ask is whether clock slowing is a general or specific process.

We developed a new *embedded interval estimation procedure* to address this question. Participants experienced causal and noncausal intervals; in the former they pressed a key to trigger a tone after a random interval, in the latter a visual change on the computer screen was followed by the tone. We embedded an additional event into certain trials at different points; sometimes this event occurred before the interval, in other trials it occurred during the interval, and in others not at all. Participants had to estimate the duration of either the interval, or the embedded event. If clock slowing is general, then embedded event estimates should be shorter for events embedded into causal intervals, compared to events embedded into noncausal intervals. Alternatively, if clock slowing is a binding-specific process then estimates for embedded events should not differ between causal and noncausal conditions. Likewise, if temporal binding does not implicate any changes to clock speed, then there should also be no differences in the estimates for embedded events.

Method

A key concern in developing the *embedded interval estimation procedure* was to ensure that the embedded stimulus was independent of the main interval. More specifically, embedded events had to be perceived as causally unrelated to key presses. To make it clear that the embedded event was independent of the action, we scheduled one-third of trials to contain an embedded event before the key press, one-third after the key press, and one-third to contain no embedded event. To achieve this, we used an algorithm that predicted a participant's key press time for each causal trial, and scheduled delivery of the embedded event either before or after this predicted time. In noncausal trials we employed stimulus delivery times recorded from participants in a pilot experiment with the same algorithm. This procedure ensured noncausal trials mirrored causal trials as closely as possible.

Participants were asked to estimate the duration of either the interval or the embedded event. To make sure that they focus on all aspects of the task, participants were not told which event they had to estimate until the end of each trial.

Participants Thirty-five and 34 Students of Cardiff University participated in exchange for course credits or £5 in Experiments 1 and 2, respectively.

Apparatus and stimuli The experiment was implemented in Psychopy (Peirce, 2007) on PCs connected to 19" monitor with resolution of 1280 x 1024 pixels. In Experiment 1, the embedded event was a tone (523.25 Hz), and in Experiment 2, a yellow polygon (vertices bounded by

a 270 x 210 pixel rectangle) and lasted for either 300 or 500 ms. A 1000 ms presentation of a red square (400 pixels^2) served as the outcome on causal trials and marked the end of noncausal intervals. A black square (400 pixels^2) was presented at the beginning of each trial. All visual stimuli were presented centrally on the screen.

Design and procedure Three factors were employed in the study: Trial type consisted of two levels (causal, noncausal), embedded event location of three levels (before interval, during interval, no event) and embedded event duration of two levels (300, 500 ms). Dependent variables (measured on separate trials) were estimates of overall interval and embedded event duration.

Causal trials began with the black square on the screen. Participants pressed a key at a time of their choice. This led to the immediate disappearance of the black square, and triggered the red square after a random interval (range 700-1300 ms). Noncausal trials also began with the black square, which remained on the screen according to a predetermined time derived from the instrumental trials of previous pilot participants as explained below; following the disappearance of the black square, the red square appeared after a random interval of the same 700-1300ms range. Both types of trials, were presented in blocks of 30 trials, 10 of which were scheduled to contain an embedded stimulus, *during* the relevant interval, 10 to contain an embedded stimulus *before* the interval (i.e. before the participant pressed the key or before the black square disappeared on its own), and 10 trials in which no embedded stimulus was scheduled. At the end of each trial, participants were prompted to estimate *either* the duration of the embedded stimulus, *or* the duration of the key press – outcome interval (on causal trials) or the duration between the disappearance of the black square and the appearance of the red square (on noncausal trials).

To schedule delivery of the embedded stimulus, an algorithm was used to predict participants' key press time, with the embedded event scheduled at a random time (range 50 - 400 ms) before or after the predicted key press. Based on pilot data, we set the algorithm to begin with a prediction of 800 ms on the first trial, and implemented a cumulative average based on key press times up to the first five trials). Thereafter, a rolling average calculated over the last five trials was employed. Values shorter than 400 ms or longer than 3000 ms were not considered for the averages.

At the beginning of each causal trial, the computer thus determined the length of the to-be-experienced key press – outcome interval (from a random range of 700-1300 ms), as well as whether and when it was to contain an embedded event. If an embedded event was scheduled, its duration could be either 300 or 500 ms (see design specifics below). For the participant, the trial began with the display of the black square, which remained on the screen until he or she pressed the Z key, which led to the immediate disappearing of the black square and triggered the appearance of the red square after the scheduled interval. Depending on the schedule, the embedded event occurred between 50-400 ms before or after the *predicted* time of the key press, or not at all. Following the display of the outcome (red square), participants were prompted to estimate either the duration of the action-outcome interval (on a scale from 0 – 2000 ms) or the duration of the embedded event (on a scale from 0 – 1000 ms). The screen then blanked for a random duration (1200 - 2000 ms) before the next trial.

For noncausal trials we adopted an analogous procedure. We replayed values from causal trials of a pilot version of the experiment, where we recorded the time of participants' key presses, the durations of the intervals, as well as the positions and durations of the embedded events (based on the same prediction algorithm as described above). In the current experiment, for each participant, we randomly selected a pilot participant's data file and replayed its values in noncausal trials, using the recorded key press time to schedule the disappearance of the black square. For example, if a pilot participant pressed the key 900 ms into the trial, experienced an interval of 1100 ms, with a 300 ms embedded event presented 800 ms after his or her key press, a corresponding noncausal trial in the current experiment would display the black square for 900 ms at the start of the trial, followed by the red square after 1100 ms; in addition an embedded event of 300 ms duration would be presented 800 ms after the disappearance of the black square. Note that the algorithm cannot perfectly predict a participant's key press, and that consequently the number of trials where the embedded event was experienced before or after the action will fluctuate between participants (see Table 1). To optimize the experience on noncausal trials, we screened previous participants' stimulus patterns and excluded those

Table 1: Mean percentage of trials on which an embedded event was experienced before and during the overall interval, for interval and embedded event judgment trials in Experiment 1 and 2 (standard deviation in parenthesis).

Judgment prompted	Experiment 1				Experiment 2			
	Causal		Noncausal		Causal		Noncausal	
	Before	During	Before	During	Before	During	Before	During
Interval	50.16 (11.09)	44.35 (12.63)	41.94 (6.28)	53.63 (6.35)	47.66 (9.71)	45.55 (11.55)	44.38 (7.04)	51.72 (6.64)
Embedded Event	50.40 (10.02)	45.16 (11.76)	46.05 (5.62)	49.27 (6.23)	51.41 (10.34)	41.95 (12.28)	46.48 (5.53)	48.67 (6.57)

Note: Before, During = Embedded Event occurred before/during the interval. Percentages do not sum to 100 because a mean range of 3.91 - 6.80% of trials contained embedded events that began during or after the outcome, and are thus not included. 2 x 2 (Trial Type [causal, noncausal] x Embedded Event Location [before, during]) ANOVAs found no significant effects (ps>.05), with one exception: Interval judgment trials in Experiment 2 had more events in noncausal than causal trials (means of 48.05 and 46.60 %, respectively), $F(1, 31) = 7.79$, $p < .01$. We attribute this to random fluctuation.

where embedded event timings deviated by more then 30% from the schedule (e.g. where the balance of embedded events occurring before and during the relevant interval deviated from the scheduled 50-50% balance to an extent exceeding 35-65% in either direction). Each causal and noncausal block consisted of 30 trials prompting for an interval and 30 requesting an embedded event duration judgment, presented in random order. For each judgment type, there were 10 trials with the embedded event scheduled before, 10 with it scheduled during the interval, and 10 trials with no event. The duration of embedded events was either 300 or 500 ms, with both durations occurring equally often across trial and judgment types. 10 trials in each block prompted for an embedded event judgment when in fact no embedded event had occurred. These served as catch trials, and participants were instructed beforehand to enter an X on such trials.

Each participant worked through two causal and noncausal blocks in an alternating sequence, with the beginning of the sequence (causal, noncausal) counterbalanced.

Results

Data analysis Data were screened based on catch trials. Participants who failed to correctly recognize more than 30% of catch trials in at least one condition (Four participants in Experiment 1, two in Experiment 2) were not considered for analysis.

Data for interval judgments were classified into three categories: trials where an embedded event occurred before the interval, during interval, or not at all. Data for embedded event judgments were classified into two categories: trials where the embedded event occurred before the interval and trials where it occurred during the interval. Note that this classification is based on *actual* rather than *scheduled* embedded event location. Table 1 shows that the prediction algorithm achieved approximately equal distribution of embedded events before and during intervals. There are no systematic deviations from the intended 50-50 balance, and no deviation is larger than 9%.

All analyses were computed with respect to judgment errors, defined as the difference between the estimated and the actual value, where *under*estimation is conveyed by negative values and *over*estimation by positive values.

Experiment 1

Intervals Figure 1 shows that intervals were generally underestimated, with causal intervals underestimated to a greater extent, replicating the typical binding effect. An exception to this are noncausal intervals with an embedded event before the interval, which were overestimated. Figure 1 also shows that intervals with an event presented during the interval, were underestimated more than intervals with no event and an event before. Analysis of variance (ANOVA) found a significant effect of trial type, $F(1, 30) = 24.97$, $p < .001$, and a significant effect of embedded event location, $F(2, 60) = 17.58$, $p < .001$. The Trial Type x Embedded Event Location interaction was also significant, $F(2, 60) = 6.33$, $p < .01$.

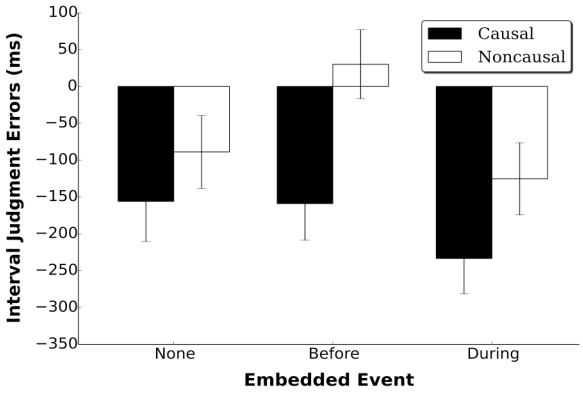

Figure 1: Mean interval judgment errors from causal and noncausal trials broken down by embedded event location. Error bars represent standard error.

Embedded Events Inspection of Figure 2 finds a general overestimation of embedded events, with somewhat greater overestimation for events embedded in causal intervals, contrary to what is predicted from a general clock slowing hypothesis. ANOVA found no effect of trial type, $F(1, 30) = 1.29$, $p = .27$, or embedded event location, $F(1, 30) = .83$, $p = .37$, nor an interaction, $F(1,30) = .00$, $p = .97$.

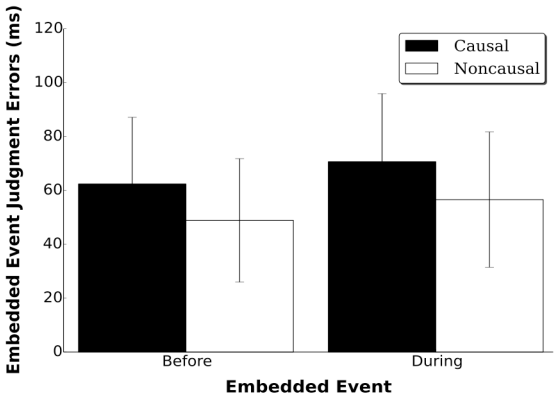

Figure 2: Mean embedded event judgment errors for events occurring before and during the interval, broken down by trial type. Error bars represent standard error.

Experiment 2

Intervals Figure 3 shows that intervals again were generally underestimated, with greater underestimation for causal intervals. An exception, similar to Experiment 1, are interval judgments from noncausal trials where an embedded event occurred before the interval, which seemed to reflect overestimation. Above all, Figure 3 shows a clear binding effect regardless of embedded event location. ANOVA supports these findings, with a significant effect of trial type, $F(1, 31) = 13.03$, $p < .01$. The effect of embedded event location, $F(2, 62) = 7.14$, $p < .01$, as well as the Trial Type x Embedded Event Location interaction, $F(2, 62) = 3.75$, $p < .05$ were also significant.

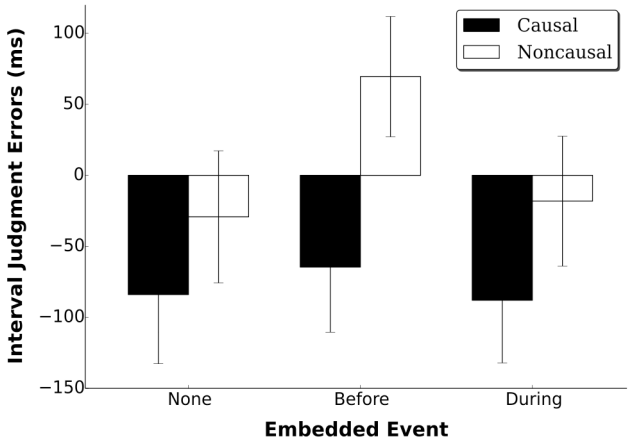

Figure 3: Mean interval judgment errors from causal and noncausal trials broken down by embedded event location. Error bars represent standard error.

Embedded Events Figure 4 shows that embedded visual events were underestimated, in contrast to embedded auditory events in Experiment 1, which were overestimated. This most likely reflects the typical finding that auditory events are judged longer than visual events (Wearden *et al*, 1998). More important for our purposes here, though, is whether embedded event judgments varied as a function of trial type. As in Experiment 1, this was not the case. There were no significant effects of trial type, $F(1, 31) = .03$, $p = .87$, embedded event location, $F(1, 31) = 2.36$, $p = .14$, nor a Trial Type x Embedded Event Location interaction, $F(1, 31) = .00$, $p = .96$.

Discussion

We developed a new procedure - the *embedded interval estimation procedure* - to study the role of internal clocks in temporal binding. Using this method, we replicated the binding effect in two experiments, with causal intervals judged as shorter for all embedded event locations. In contrast, we found no difference between causal and noncausal embedded event judgments, both when the event began *before* and *during* the interval, and regardless of whether the embedded event was visual or auditory. Interestingly, we found embedded events overestimated in Experiment 1 and underestimated in Experiment 2, thus replicating the finding that auditory stimuli are judged as longer than visual (Wearden *et al*, 1998).

An unanticipated result concerns judgments of noncausal intervals from trials with an embedded event *before* the interval, which were longer than from any other interval category. One possible explanation for this implicates the trial structure we employed: Because on a given trial at most one embedded event occurred, the presentation of an event *before* the interval meant participants knew that no further event will occur during the remainder of the trial. Participants would then have been able to attend exclusively to the interval, whereas otherwise they would still divide

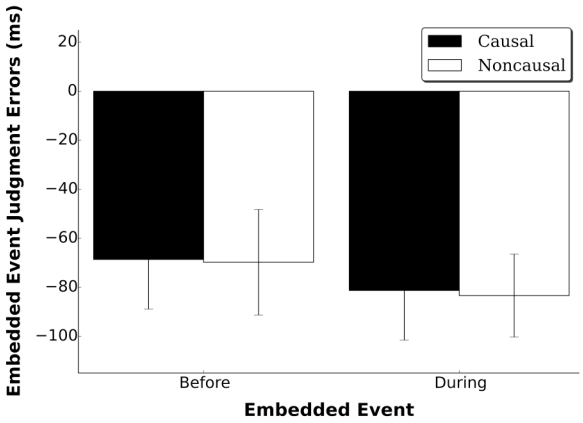

Figure 4: Mean embedded event judgment errors for events occurring before and during the overall interval, broken down by trial type. Error bars represent standard

their attention between tracking overall interval duration and monitoring the potential occurrence of an embedded event. A common assumption of internal clock models is that subjective time is modulated by attention, with greater attention paid to time passing resulting in more clock pulses accrued leading to in the experience of a subjectively longer interval (cf. the common adage "A watched pot never boils" or "Time flies when you are having fun", Avni-Babad & Ritov, 2003). This attentional modulation would have affected causal and noncausal trials equally, as reflected by analogously less negative judgment errors on causal trials. However, the binding effect presumably was so robust as to prevent causal trials to be overestimated.

Importantly, our finding that temporal binding is robust regardless of the presence of events embedded into the causal interval demonstrates the reliability of the procedure, as does replicating the audio-visual illusion from Wearden *et al* (1998). What then, can our results say about time perception within the temporal binding effect? At a minimum, our results suggest that temporal binding does not occur due to general slowing of the pacemaker. Does this rule out a clock-slowing account of temporal binding? Not necessarily. It could be entirely possible that temporal binding selectively affects dedicated time-keeping processes, allocated to keeping track of action-outcome (or cause-effect) intervals. Computationally, this would necessitate multiple clocks, each capable of independent and simultaneous timing. Buhusi and Meck (2009) provide evidence for such a notion: Rats were trained to time 3 different durations, presented simultaneously. Quantitative modelling demonstrated (1) that rats were able to time each duration by independently stopping and resetting separate clocks, and (2) that durations are perceived differently depending on context, (e.g., the relative overestimation of auditory relative to visual stimuli implicates modality differences in clock rates, Wearden *et al*, 1998). Tentative support for clock-slowing in binding is evidenced in Humphreys and Buehner (2009), who found a linear relationship between perceived and actual durations for a range of intervals. Specifically, they reported different

slopes for causal and noncausal intervals, suggesting a stable pacemaker rate *within* but variable *between* conditions.

In both experiments we focused on interval perception. What about the sensory realignment perspective on binding? It is important to note that clock- and event-based perspectives on binding need not be mutually exclusive. A slower clock between action and outcome, for example, can lead to a contraction of time (i.e., binding), which in turn would result in the outcome perceived sooner. Thus, there could be shifts in event perception *in addition* to a slower clock. Consider now, general and specific clock slowing. In the former case, there would be no perceived shift in the outcome because events would now be judged in relation to the *same* slowed clock speed. In the latter, the action-outcome clock rate would differ to other timing streams, meaning that the outcome would be perceived earlier relative to other events timed at a standard clock rate. Thus, a specific clock process could account for the perceived event shifts in temporal binding in addition to distortions of interval perception.

In sum, our results have conclusively ruled out the possibility that temporal binding is effected by a slowing of a general clock. They fit equally well with sensory realignment perspectives on binding that do not implicate subjective time-keeping at all (e.g., Eagleman & Holcombe, 2002) and the notion of multiple dedicated specific clock processes. One way to disentangle these accounts is by systematically investigating clock processes in causal (i.e., action-outcome) and purely observational (noncausal) intervals. We are currently investigating differences in the point of subjective equality (PSE) for a range of causal and noncausal interval durations. Regressing PSEs over actual durations will allow us to conclusively test for differences in *r*: Steeper slopes for noncausal compared to causal PSEs would unambiguously implicate different pacemaker rates. Thus, our ongoing research extends the line of thought explored in the current article that suggests a dedicated clock process in action-outcome (causal) intervals. We therefore suggest that the perceptual shifts associated with temporal binding are possible due to multiple clocks operating concurrently.

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