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

Richard Fereday (FeredayR@cardiff.ac.uk), Marc J. Buehner (BuehnerM@cardiff.ac.uk)
Cardiff University, School of Psychology, Park Place, Cardiff, CF10 3AT, Wales, UK

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 than 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 noncausal 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
Thereafter, we set the algorithm was used to predict noncausal (on stimulus, scheduled. Pressed the key or before the black square disappeared on its during of B disappearance of the black square, the red participants which remained on the screen according to a predetermined triggered two levels (300, 500 ms). During interval, no event) and embedded event duration of 5 trials (means of 48.05 and 46.60 %, respectively), F(1, 31) = 7.79, p < .01. We attribute this to random fluctuation.

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 (p> .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.

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).

<table>
<thead>
<tr>
<th>Judgment prompted</th>
<th>Experiment 1</th>
<th></th>
<th>Experiment 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Causal</td>
<td>Noncausal</td>
<td>Causal</td>
<td>Noncausal</td>
</tr>
<tr>
<td></td>
<td>Before</td>
<td>During</td>
<td>Before</td>
<td>During</td>
</tr>
<tr>
<td>Interval</td>
<td>50.16</td>
<td>44.35</td>
<td>41.94</td>
<td>53.63</td>
</tr>
<tr>
<td></td>
<td>(10.09)</td>
<td>(12.63)</td>
<td>(6.28)</td>
<td>(6.35)</td>
</tr>
<tr>
<td>Embedded Event</td>
<td>50.40</td>
<td>45.16</td>
<td>46.05</td>
<td>49.27</td>
</tr>
<tr>
<td></td>
<td>(10.02)</td>
<td>(11.76)</td>
<td>(5.62)</td>
<td>(6.23)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Experiment 1: 2 x 2 (Trial Type [causal, noncausal] x Embedded Event Location [before, during]) ANOVAs found no significant effects (p> .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.
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 1

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.
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 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

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

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 errors.
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.

**References**


