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Memory, Arousal, and Temporal Binding: A Computational Model

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Abstract

In the interval estimation task, participants are asked to estimate the time interval between two events. Interval estimates tend to be smaller if the first event is a voluntary action. The cognitive mechanisms that generate this temporal binding effect remain unclear. In this study, we propose a computational model of this phenomenon. We hypothesize that participants perform this task by comparing the availability of experimental events in memory. More specifically, we claim that temporal binding effects may be attributed to differences in emotional arousal at the time of encoding due to the type of event. For instance, voluntary actions may be accompanied by slightly higher arousal, leading to stronger traces of the first event relative to the involuntary case.

Keywords: temporal binding; sense of agency, interval estimation

Introduction

Temporal binding refers to a cognitive phenomenon where the time interval between a voluntary action and a consequent event is perceived to be smaller than it is in reality (Haggard et al., 2002). For example, an individual may report a shorter time interval between a button press and the consequent onset of a light as compared to a similar report between two unrelated events separated by the same interval. Also often called "intentional binding" in literature, this effect has been replicated in studies exploring many potential variables such as distribution of attention, cause-effect relationships, temporal predictability, and somatic sensations (Buehner, 2012; Engbert et al., 2007; Moore & Obhi, 2012).

Two experimental paradigms are prevalent in the literature on temporal binding. In the Libet clock method, participants estimate the time at which the first and second events occurred using a clock face (Libet et al., 1983). More recent studies adopt the *interval estimation paradigm*, which is the focus of the present study. In this paradigm, participants are instructed to simply report the perceived interval (in ms) between an action and effect (Moore & Obhi, 2012).

A typical interval estimation study follows the timeline depicted in Figure 1. First, participants perform a voluntary action or an involuntary movement. An example of a voluntary action would be pulling a lever or pushing a button, whereas an involuntary action may be induced through transcranial magnetic stimulation (TMS) or simply involve watching an action happen outside of the body's agency. After this initial action, there is a time interval, whose duration may vary between studies and conditions. Following this time interval, an auditory tone occurs, and participants are asked to estimate the time interval between the two events.

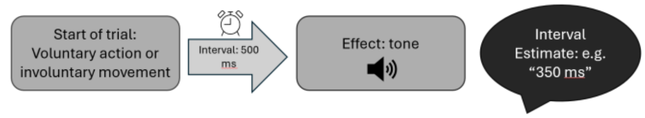

Figure 1: Timeline of the Interval Estimation Paradigm

Accounts of temporal binding are categorized into three main camps: agency, causation, and multisensory integration. The agency view argues that the interval compression is tied to the intentionality of voluntary action, with classic evidence showing temporal binding effects for voluntary actions but not involuntary ones (Haggard et al., 2002; Moore & Obhi, 2012). In contrast, the causation camp claims that rather than being specific to agency, temporal binding arises when one perceives a causal link between the two events (Buehner & Humphreys, 2009). This view is supported by findings that binding occurs if events are believed to be causally connected. Finally, multisensory integration accounts frame temporal binding as a perceptual mechanism through which the brain integrates uncertain or ambiguous sensory information into a coherent timeline (Buehner, 2012). Consistent with this perspective, binding effects have been observed to strengthen when actions or outcomes are less perceptually certain (Klaffehn et al., 2021).

The present study investigates the question: "Can we provide an integrative and mechanistic account of temporal binding effects, while also explaining its connections with sense of agency?" and proposes that there is a causal chain of mechanisms that can successfully synthesize this connection. To this end, we present a memory-based mechanistic cognitive model of temporal binding phenomena capable of motivating a syncretic perspective on theories of temporal binding and of accounting for a wide variety of observations concerning temporal binding phenomena.

More specifically, we propose that having agency over an action may induce a change in emotional state, such that voluntary actions are associated with greater arousal relative to non-voluntary events. This relative difference in emotional state may then influence the parameters of memory retrieval and retention, which may, in turn, influence how we perform interval estimations, ultimately leading to temporal binding effects. Here we investigate the case where a heightened emotional arousal state associated with the first (action) event may lead to temporal binding effects due to a

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slower decay rate for the action event trace in memory.

Model

To formalize our proposed arousal-memory mechanism, we draw on the rational analysis of memory, which rests on the premise that the human memory system is adaptive. In this view, the function of the memory system is to ensure that memory traces more likely to be needed in the present are made more available (Anderson & Schooler, 1991; Anderson et al., 2012; Schooler & Anderson, 2017). An important consequence of this basic notion is that the availability of a trace is determined, in part, by its history of use, specifically how frequently and recently the trace has been used in the past.

This history factor is formalized in the base-level activation equations of the ACT-R cognitive architecture (Anderson, 1990; Anderson et al., 2012). From a Bayesian perspective, the base-level activation of a memory chunk represents the prior odds of needing that chunk for retrieval as a function of its history of use as shown in the following equation.

$$B_i = \alpha \sum_{j=1}^{n} t_j^{-d} \tag{1}$$

In this equation, B_i represents the base level activation of chunk i, j indexes each invocation of chunk i, t_j represents the time elapsed since the jth invocation event. The base-level activation equation also contains the scaling constant alpha and the decay rate d as free parameters.

It has, on occasion, been proposed that the parameters of the base-level equation may vary between invocations (e.g., Pavlik & Anderson, 2005). Recently, work has begun to explore how a modification of this sort may account for the influence of emotion on memory (Gajraj & Mekik, 2024). This work hypothesizes that emotion, or more specifically *core affect* (Russell, 2003), may influence memory by modulating the free parameters of the base-level activation equation. Formally, this notion may be captured by the following generalized form for the base-level equation, where the scale and decay parameters are allowed to vary between invocations.

$$B_i = \sum_{j=1}^{n} \left(\frac{t_j}{\alpha_j}\right)^{-d_j} \tag{2}$$

In this modification of the base-level learning model, the scale and decay rate of each invocation trace is determined, at least in part, by the participant's core affect at invocation (i.e., encoding) time. Regarding the relation between core affect and the model parameters, we hypothesize that the decay parameter is modulated by the arousal component of core affect and that the scale parameter is modulated by the valence component. The first of these hypotheses is central to the present study and it is informed primarily by findings that, while increased arousal appears to impede retrieval in the short term, it facilitates retrieval in the longer term (see, e.g., Kleinsmith & Kaplan, 1963). These qualitative observations support the hypothesis in that they align well with the qualitative effects of the two parameters on the contributions of individual invocations to base-level activations over time.

Here, we argue that this modified form of the base-level activation equation may explain temporal binding phenomena. Notably, given knowledge of the scale, decay, and time parameters associated with two distinct invocations, one may recover the exact amount of time between the two invocation events via the following transformation, where B_1 and B_2 are base-level activations associated with two single invocations.

$$\Delta t = \alpha_1 B_1^{-\frac{1}{d_1}} - \alpha_2 B_2^{-\frac{1}{d_2}} \tag{3}$$

This observation naturally suggests a parsimonious mechanistic theory of interval estimation where subjects monitor and compare base-level activations associated with events of interest through metacognitive processes. Within such a theory, temporal binding effects and other distortions of time perception may be explained by constraints on the accessibility of the relevant parameters to metacognitive monitoring and comparison processes.

We hypothesize that the momentary scale and decay parameters associated with an invocation are only available at encoding time and that the metacognitive system uses the core affect parameters available in the current moment to generate interval estimates. These assumptions yield the following general formula for interval estimates as a function of the model's parameters.

$$\Delta t \approx \alpha_3 \left(B_1^{-\frac{1}{d_3}} - B_2^{-\frac{1}{d_3}}\right) \tag{4}$$

This formulation can account for temporal binding effects via differences in the arousal state induced by the first event. According to our parameter mapping, an arousing event, such as a voluntary event requiring the exertion of effort, will be associated with a smaller decay value as compared to an event associated with a lower level of arousal. If an interval estimate is made at a time that exceeds the scale parameter associated with the first event, the activation associated with the higher-arousal event will be greater than that associated with the lower arousal event. Assuming that the second event is identical between conditions, this pattern will yield a smaller interval estimate.

The interval estimation paradigm is known to be highly sensitive to experimental parameters (Moore & Obhi, 2012). This sensitivity may be explained by two related and salient characteristics of our proposed analysis. First, the non-linearity of the base-level activation equation may produce inconsistent associations between interval estimates and experimental conditions depending on the exact configuration of cognitive and experimental parameters. For instance, an interval estimate for an arousing first event may result in an attenuation or even reversal of the binding effect if the estimate is made at a time that approaches or falls below the scale parameter associated with the first event. Second, by the same token, our analysis suggests that interval estimation tasks are sensitive to the duration of the second event. Thus, otherwise identical designs may nevertheless produce inconsistent results if this second delay is not considered.

To our knowledge, no prior account of temporal binding predicts reversals or sensitivity to the duration of the second event. In that regard, these two predictions are substantive empirical hypotheses that follow from our model. Since no prior theory of temporal binding predicts that the effect may be sensitive to the duration of the second event, there appears to be no data that directly bears on this hypothesis. However, a recent study by Galang et al. (2025) reports the first observation of the reversal of a temporal binding effect in an interval estimation task.

Method

As an initial test of our proposed model, we present a reanalysis of data from Galang et al. (2025). This target study compares, in two experiments, interval estimates for pairs of events where the initial event is a button press performed under either rule-following or rule-breaking conditions. Stronger temporal binding effects are reported in the rule-following condition as compared to the rule-breaking condition, but only for the shortest interstimulus interval of 100ms. Interestingly, in Experiment 2, a reversal of this pattern is observed for the longer intervals of 400ms and 700ms.

Galang et al. (2025) argue that the reversal effect may be attributed to greater cognitive conflict associated with the rule-breaking condition, which is supported by the observation that response times in the rule-breaking condition are significantly slower than response times in the rule-following condition. Although our model does not make specific predictions about response times in interval estimation tasks, it nevertheless invites an analysis consistent with this conflict theory. Notably, we may hypothesize that the rule-breaking condition induces greater arousal than the rule-following condition, generating observed differences in interval estimates as a function of the interstimulus interval. Here we test this hypothesis by fitting our model to the Galang et al. data.

Data

Galang et al. (2025) use an online free-choice interval estimation paradigm to measure temporal binding in rule-following vs. rule-breaking. On each trial, participants first choose whether to follow or break a simple stimulus-response rule (square vs. triangle mapped to the “A” vs. “D” key) and then execute the corresponding keypress. The keypress triggers a visual outcome (the left or right circle turning blue) after an experimentally controlled delay (100 ms, 400 ms, or 700 ms). Participants then provide an interval estimate (in ms) of the perceived delay between their action and the outcome using a slider scale ranging from 1-1000 ms. Interval estimates serve as the primary measure, with smaller estimates interpreted as stronger temporal binding.

Experiment 1 was the initial run of this paradigm. In this run, there was substantial loss of participants, as they tended overwhelmingly to follow the rule, triggering exclusion criteria. This also created an imbalanced design and forced the authors to deviate from their preregistered plan to use repeated-measures ANOVA in favor of linear mixed-effects models. Experiment 2 is therefore a direct replication; the task is unchanged, but participants are additionally prompted to follow or break the rule more evenly.

Across the two experiments, Galang et al. find a consistent temporal binding effect at the shortest delay (100 ms), where participants give smaller interval estimates when following the rule than when breaking it (Experiment 1: $M = 19.01$, $SE = 4.94$, $p < .001$; Experiment 2: $M = 10.9$, $SE = 5.03$, $p < .05$). The pattern at longer delays differs across experiments. In Experiment 1, the rule-following versus rule-breaking difference is not statistically significant at 400 ms ($M = 1.83$, $SE = 4.86$, $p = .7$) or 700 ms ($M = 2.48$, $SE = 4.87$, $p = .6$). In Experiment 2, by contrast, the effect is reversed at these longer delays, such that participants give smaller interval estimates when breaking the rule than when following it (400 ms: $M = -11.7$, $SE = 4.97$, $p < .05$; 700 ms: $M = -22.0$, $SE = 4.94$, $p < .001$).

Analytic Strategy

We make the following simplifying assumptions to derive a form for our model that is amenable to being fitted to the data via simple linear regression. These assumptions also serve to eliminate degrees of freedom in the model that are not relevant to the current experimental setting. Specifically, we assume that $\alpha_1 = \alpha_2 = \alpha_3 = \alpha$, where α is some constant. There are two primary motivations for this assumption: Our analysis of the Galang et al. (2025) data makes no differential prediction concerning this parameter and fixing this parameter leads to a simple identifiable statistical model, as shown below. By the same token, we also assume that $d_2 = d_3 = ½$, with ½ being the default value for the decay parameter (Salvucci, 2014). This leaves $d_1 = d$ as the only other free parameter, which we let vary between participants and conditions, allowing the model to capture individual variability in responses to rule breaking versus rule following.

With these assumptions, our model’s interval estimation equation is mathematically equivalent to the following log-linear relationship, where y represents participants’ interval estimates, x represents the interstimulus interval, and c represents the duration of the second event.

$$\log(c + y) = (1 - 2d)\log(\alpha) + 2d\log(c + x) \quad (5)$$

Table 1: Parameter Fits

	Study 1	Study 2
(Intercept)	2.67 ***	0.59 ***
	(0.15)	(0.15)
$\log(t_1)$	0.65 ***	0.92 ***
	(0.02)	(0.02)
Follow Rule	-0.18 *	-0.42 ***
	(0.09)	(0.11)
$\log(t_1)$ x Follow Rule	0.02 *	0.06 ***
	(0.01)	(0.01)
AIC	-48581.23	-37152.84
BIC	-48461.32	-37035.00
Log Likelihood	24305.61	18591.42
Num. obs.	21890	19073
Num. groups: id	273	236

In this form, our model may be directly fitted to the experimental data via a linear mixed-effects regression model of the following form, letting $Y = \log(c + y)$, $X = \log(c + x)$, and C be a dummy variable indicating whether a trial is in the rule-following condition.

$$Y_{pt} = \beta_{0p} + \beta_{1p}X_{pt} + \beta_{2p}C_{pt} + \beta_{3p}X_{pt}C_{pt} + \varepsilon_{pt} \quad (6)$$

In this statistical model, all coefficients are allowed to vary by participant. This is because we assume that the decay parameter may vary by participant and by condition, and both terms of Equation 5 depend on this parameter. The parameters β_{0p} and β_{1p} represent, respectively, estimated values for the intercept and coefficient terms in Equation 5 for participant p in the rule-breaking condition. Estimated values for these terms in the rule-following condition are given, respectively, by the terms $\beta_{0p} + \beta_{2p}$ and $\beta_{1p} + \beta_{3p}$.

Given fitted values for d and $A = (1 - 2d) \log \alpha$, we may compute a deterministic prediction for participants' interval estimates via the following transformation. Note that given our specifications, this prediction varies only by participant, condition, and inter-stimulus interval.

$$y = e^{A+2dX} - c \quad (7)$$

Furthermore, if we assume that the activation noise associated with the trace for the consequent event is zero, we may implement a simple simulation of the interval estimation process using the residual variance of our statistical model to set the activation noise level for the first event.

Thus, we formulate the following hypotheses and tests to evaluate the fit of our model to the Galang et al. (2025) data. First, we expect a positive interaction between the log-retention-interval ($c + x$) and the experimental condition upon fitting Equation 6. Second, we expect interval estimates inferred from fitted model parameters to correlate with observed interval estimates. Third, we expect interval estimates obtained from the model via bootstrap simulations to reproduce the contrasts observed in the Galang et al. study.

Table 2: Goodness of Fit Analyses

	Study 1	Study 2
(Intercept)	1.90	1.31
	(2.55)	(2.63)
Predicted Est.	1.01 ***	1.02 ***
	(0.01)	(0.01)
R^2	0.96	0.96
Num. obs.	1437	1383

To test our first hypothesis, we fit Equation 6 to both data sets from the Galang et al. (2025) study and analyze inferential statistics for the interaction coefficient. We then use the participant-specific coefficients obtained from these fits to generate deterministic interval estimates. We test our second hypothesis by fitting a bivariate linear regression model with our model's deterministic estimates as the predictor variable and observed mean interval estimates by participant, condition, and interstimulus interval as the dependent variable. As a test of our third hypothesis, we compute participant-specific noise variances based on the residuals of the parameter fit and compute bootstrap estimates of the experimental contrasts with 200 replicates of each data set.

Results

We fit Equation 6 to each cleaned dataset provided in the supplementary materials of Galang et al. (2025) after computing the requisite variables. Although the duration of the second event is not reported in the main text, inspection of experimental logs reveals that this delay is set to about 1500ms. Table 1 summarizes the fit of the model to each dataset. Consistent with our first hypothesis, we observe statistically significant positive interactions between the log-retention-interval and rule following condition in both Experiment 1 ($b = .02$, $SE = .01$, $p = .04$) and in Experiment 2 ($b = .06$, $SE = .01$, $p < .001$), indicating a greater decay rate in the rule following condition as compared to the rule breaking condition.

Predicted interval estimates based on fitted model parameters are strongly correlated with observed mean interval estimates computed by participant, condition, and interstimulus interval ($r^2 = .96$). Furthermore, the regression coefficient on predicted estimates is near unity (Experiment 1: $b = 1.01$, $SE = .01$, $p < .001$; Experiment 2: $b = 1.02$, $SE = .01$, $p < .001$) and intercept estimates are non-significant (Experiment 1: $b = 1.90$, $SE = 2.55$, $p = .46$; Experiment 2: $b = 1.31$, $SE = 2.63$, $p = .62$), suggesting that the model

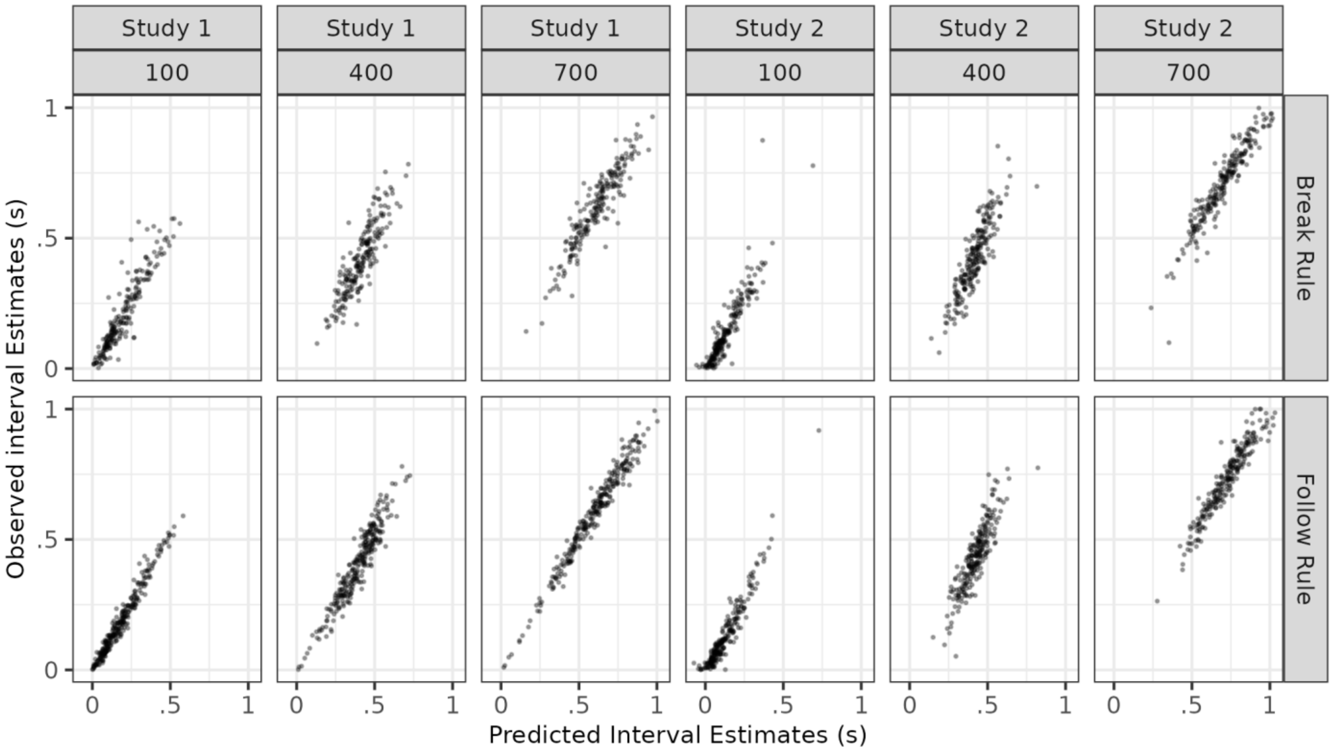

Figure 2: Plot of Predicted Interval Estimates Against Observed Interval Estimates

accurately captures variation in interval estimates due to experimental parameters and individual differences among participants. See Table 2 for detailed regression results. Figure 2 depicts the relationship between predicted and observed interval estimates by condition.

Bootstrap simulations yield between-condition contrasts consistent with those reported in Galang et al. (2025). For Experiment 1, simulated interval estimates are significantly smaller in the Follow Rule condition as compared to the Break Rule condition for the 100 ms interstimulus interval ($M = 14.09$, $SE = 3.88$, $p < .001$), but not for the 400 ms interval ($M = 7.00$, $SE = 4.01$, $p = .16$), nor for the 700 ms interval ($M = -1.65$, $SE = 4.65$, $p = .72$). For Experiment 2, interval estimates are significantly smaller in the Follow Rule condition as compared to the Break Rule condition for the 100 ms interstimulus interval ($M = 9.18$, $SE = 3.85$, $p = .034$). For the 400 ms interval, the pattern trends in the opposite direction but does not reach significance ($M = -6.70$, $SE = 4.33$, $p = .122$), and, for the 700 ms interval, this pattern is reversed and statistically significant ($M = -25.34$, $SE = 5.04$, $p < .001$).

Discussion

This paper develops a mechanistic account of temporal binding in interval estimation tasks. We propose that participants generate interval estimates by comparing the relative accessibility of events in memory which, we hypothesize, is modulated by encoding-time core affect. We formalize this process within the framework of the rational analysis of memory, and we present a test of the resulting predictions in a reanalysis of interval estimation data from Galang et al. (2025).

The Galang et al. study investigates temporal binding phenomena under rule-following versus rule-breaking conditions. Crucially, this study finds a temporal binding effect for rule-following at short interstimulus intervals and a reversal of this effect at longer intervals. Such reversals of the temporal binding effect are, to our knowledge, not predicted by any prior theoretical account. However, our model predicts this kind of behavior if rule-breaking induces higher arousal and the time interval between the first event and the interval estimate is sufficiently long.

Our reanalysis suggests that the Galang et al. data are indeed consistent with this interpretation. When the model is fitted to the experimental data, the rule-breaking condition is associated with a lower memory decay parameter which, within our theory, corresponds to a higher arousal state. Furthermore, when fitted model parameters are used to deterministically predict participants' interval estimates, predicted and observed interval estimates are virtually indistinguishable. Finally, bootstrap interval estimates sampled from the fitted model reproduce the main empirical findings of the Galang et al. study, including the observed reversal of the temporal binding effect with increased interstimulus interval.

Our model contributes to a growing body of memory-based mechanistic models of temporal binding. Notably, Saad et al. (2022) show that temporal binding effects may emerge from regression to the mean phenomena in a Bayesian memory model by reanalyzing data from Weller et al. (2020). Later, Saad et al. (2023) present a cognitive model capturing interval estimates in action trials from the same dataset implemented within the ACT-R cognitive architecture. This model generates interval estimates via memory retrieval processes using ACT-R's pacemaker module to encode interstimulus intervals and its blending mechanism to generate interval estimates. By contrast, our model operates by co-opting fundamental activation processes associated with the adaptive function of memory via metacognitive monitoring processes and aligns, in this respect, with the work of Staddon (2005).

Our model also differs from prior mechanistic models of temporal binding in that it invites a more syncretic theoretical perspective. This syncretism is a direct consequence of the fact that our model explains temporal binding phenomena via fluctuations in core affect (Russell, 2003). In this light, temporal binding may well be viewed as an implicit indicator of sense of agency provided (i) sense of agency correlates with the arousal component of core affect and (ii) other factors that may influence core affect between experimental conditions are ruled out. At the same time, temporal binding effects produced by other kinds of stimuli, like observations of causally related events, may also be understood via their influence on core affect.

Despite these initially promising results, more research is needed to directly and more comprehensively test our theory. To this end, we plan to extend this analysis to other publicly available interval-estimation datasets. Furthermore, we are currently working on testing our model with human participants using an online interval-estimation paradigm that experimentally manipulates affect at the time of the first event. However, perhaps the strongest test of our model may be to investigate whether binding effects are influenced by the duration of the second event in the manner predicted by the model. This is another study which we plan to undertake in the near future.

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