Motor Agency and Causal Agency: The Underlying Mechanism of the Intentional Binding Effect

Authors: Wu Di, Gu Jingjin, Li Ming, Ling Yibai, Zhang Ming, Zhao Ke, Fu Xiaolan, Zhao Ke

Date: 2018-12-17T00:00:00+00:00

Abstract

The temporal compression effect of voluntary action refers to the subjective compression of time between a voluntary action and its outcome. The action-based sense of agency and the causality-based sense of agency constitute two theoretical explanations for this effect. This paper first introduces the action-based sense of agency and the causality-based sense of agency separately, and provides corresponding evidence from behavioral and neural mechanisms to explain the mechanism underlying the temporal compression effect. Additionally, this paper proposes a potential relationship of serial processing and parallel processing between the action-based sense of agency and the causality-based sense of agency in the temporal compression effect of voluntary action.

Full Text

Sense of Agency Based on Action and Causation: The Mechanism of Intentional Binding Effect for Voluntary Action

WU Di[1,2], **GU Jingjin**[1,2], **LI Ming**[3], **LING Yibai**[1,2], **ZHANG Ming** , , **ZHAO Ke**[1], , **FU Xiaolan**[1,2]

[1]State Key Laboratory of Brain and Cognitive Science, Institute of Psychology, Chinese Academy of Sciences, Beijing 100101, China
[2]Department of Psychology, University of Chinese Academy of Sciences, Beijing 100049, China
[3]Jishou University, Jishou 416000, China
Key Laboratory of Mental Health, Institute of Psychology, Chinese Academy of Sciences, Beijing 100101, China
Department of Psychology, Dalian Medical University, Dalian 116044, China

Abstract

The intentional binding effect refers to the subjective temporal compression between a voluntary action and its outcome. Two theoretical explanations—sense of agency based on action and sense of agency based on causation—have been proposed to account for this phenomenon. This paper first introduces these two constructs separately and provides corresponding evidence from behavioral and neural perspectives to explain the mechanisms underlying the temporal binding effect. Additionally, we propose potential relationships between action-based and causation-based sense of agency in intentional binding, specifically sequential processing and parallel processing frameworks.

Keywords: voluntary action; sense of agency; causation; intentional binding effect

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Voluntary action refers to movements performed with anticipatory intent that produce specific outcomes (James, 1890). Key-press actions, in particular, represent a widely used response method across psychology, cognitive neuroscience, and computer science. The intentional binding effect is a phenomenon associated with voluntary action, characterized by subjective temporal compression between the action and its sensory consequence (visual, auditory, etc.) (Capozzi, Becchio, Garbarini, Savazzi, & Pia, 2016; Cavazzana et al., 2014; Haggard, 2017; Haggard, Clark, & Kalogeras, 2002; Moore & Obhi, 2012; Ruess, Thomaschke, & Kiesel, 2018; Sidarus & Haggard, 2016; Yabe & Goodale, 2015). Professor Patrick Haggard at University College London pioneered research on this effect using the Libet clock, in which a rotating pointer completed one revolution every 2560 ms. In these experiments, participants voluntarily pressed a key, which triggered a tone after a 250 ms delay. Participants reported the clock position at the moment of key press and at tone onset. Results demonstrated subjective compression of the interval between the voluntary action and its auditory consequence (Haggard et al., 2002). An alternative paradigm, the interval estimation method, requires participants to directly estimate the duration between action and outcome (Humphreys & Buehner, 2009; Zhao, Chen, Yan, & Fu, 2013), yielding convergent evidence for temporal compression in voluntary action.

Researchers widely agree that sense of agency—the subjective experience of controlling one' s own actions and their external consequences (Haggard, 2017)—drives this temporal compression. Two distinct forms of agency have been distinguished: a low-level action-based sense of agency and a high-level causation-based sense of agency (Synofzik et al., 2008). Correspondingly, two theoretical accounts have emerged to explain intentional binding. The first posits that agency arises from matching predicted and actual sensory feedback, thereby compressing subjective time. This explanation builds on the comparator model, suggesting that action-based agency produces temporal compression. The second account argues that participants infer causal relationships between actions and outcomes based on temporal contiguity and succession, and that this causal

inference itself generates temporal compression.

[Figure 1: see original paper] Schematic representation of Haggard et al.'s (2002) findings. When participants performed voluntary key presses, they reported the action occurrence as later (15 ms) and the tone onset as earlier (46 ms) than their actual timing.

Intentional Binding Effect and Action-Based Sense of Agency

Action-based sense of agency refers to the subjective experience of controlling one' s actions and their environmental consequences during voluntary movement. This construct is grounded in the comparator model (Blakemore, Frith, & Wolpert, 1999; Wolpert, 1997), which proposes that agency emerges when predicted outcomes match actual sensory feedback. Under this framework, temporal binding occurs when predicted and actual sensory feedback align, making this match crucial for the intentional binding effect.

Two factors critically influence action-based sense of agency. First, **subjective intention** plays a pivotal role. In Haggard' s original studies, TMS stimulation of the motor cortex induced involuntary key presses without subjective intention, eliminating the binding effect between action and outcome (Haggard et al., 2002). Similarly, research using voluntary versus involuntary action paradigms found that passive key presses abolished temporal binding (Zhao et al., 2016). In a more recent study, participants were coerced by an experimenter to administer electric shocks. Results showed reduced binding under coercion compared to voluntary conditions, which the authors attributed to diminished subjective intention (Caspar, Christensen, Cleeremans, & Haggard, 2016).

Second, **timely sensory feedback** is essential. According to the comparator model, agency arises from matching predicted and actual feedback, making temporal contiguity critical. Comparisons between active key pressing and key lifting reveal that the match between pressing action and tactile feedback is a key factor in generating binding. Active key pressing, which provides immediate tactile feedback, produces stronger binding than key lifting without such feedback. When post-action tactile feedback is removed, pressing and lifting actions produce equivalent binding effects (Zhao et al., 2016). Furthermore, when the interval between action and visual outcome is within approximately 200 ms, lifting and pressing actions produce similar binding, suggesting that timely visual feedback can substitute for tactile feedback (Zhao et al., 2013). These findings underscore the importance of matching predicted actions with timely sensory feedback.

Neural mechanisms underlying action-based agency have also been investigated. EEG studies show that voluntary key pressing elicits a larger P1 component than key lifting, with a frontocentral scalp distribution corresponding to delta-theta frequency components. These components are inversely related to the magnitude of temporal binding (Zhao et al., 2014). Regarding action-outcome

mismatches, Band et al. (2009) designed a probabilistic learning task requiring participants to process task-relevant external feedback. Negative outcomes produced a negative ERP component (NFB). Crucially, unexpected, task-irrelevant outcomes evoked a similar component termed the action-effect negativity (NAE), indicating that mismatches between predicted and actual outcomes generate NAE after action-outcome associations are formed (Band, van Steenbergen, Ridderinkhof, Falkenstein, & Hommel, 2009).

Neuroimaging findings implicate the cerebellum in sensorimotor coordination (Blakemore et al., 1998; Blakemore, Wolpert, & Frith, 1999; Blakemore, Frith, & Wolpert, 2001). Blakemore et al. (1998) found reduced cerebellar activation during self-produced versus passive tickling, suggesting cerebellar activity reflects motor coordination—specifically, matching predicted and actual motor commands—rather than representing the causal consequences of actions (Waszak, Cardoso-Leite, & Hughes, 2012). Bastian (2006) proposed that the cerebellum generates predictive models of internal sensory states that learn sensorimotor associations, thereby facilitating feedforward motor control. Using PPI analysis, Blakemore et al. (1999) demonstrated that cerebellar modulation of motor cortex and brainstem activity was associated with reduced activation in sensorimotor cortex during action prediction. Moreover, cerebellar activation correlated with action-outcome delays, further suggesting its role in detecting mismatches between predicted and actual outcomes (Blakemore et al., 2001).

Intentional Binding Effect and Causation-Based Sense of Agency

Philosophical conceptions of causation trace back to Hume (1777/1888), who argued that we infer causality from three factors: resemblance, contiguity (spatial and temporal), and constant conjunction (probability). Psychological research has extensively investigated causal influences (Buehner & Humphreys, 2009; Eagleman & Holcombe, 2002; Wen, Yamashita, & Asama, 2015; Woods et al., 2014). Evidence suggests that intentional binding is related to causal inference. Temporal binding persists even when action-outcome intervals extend to 4 seconds—a finding that action-based agency cannot explain, as predicted and delayed sensory feedback would be mismatched. Causation-based agency better accounts for this phenomenon (Buehner & Humphreys, 2009). Causation-based sense of agency, similar to its action-based counterpart, refers to the detection and inference of causal relationships between actions and outcomes (Scholl & Tremoulet, 2000; Kawabe et al., 2013).

Although causation-based agency cannot be directly perceived, it can be inferred from action-outcome relationships. Its influence on intentional binding manifests in several ways. First, **temporal proximity** increases the likelihood of perceiving causality, thereby producing temporal compression. Haggard et al. (2002) manipulated action-outcome intervals at 250, 450, and 650 ms, finding reduced binding at longer intervals. As temporal distance increases, perceived

causality weakens, and participants are less likely to view the outcome as self-generated.

Second, **event probability** influences binding magnitude. Engbert and Wohlschlager (2007) demonstrated stronger binding in high-probability (80%) versus low-probability (20%) conditions. To further examine causality' s role, researchers compared active key pressing with a robotic hand performing the same action. Both conditions produced binding effects, suggesting that even non-self-generated actions can produce binding when causal relationships exist between action and outcome (Buehner, 2012).

Belief in causality also modulates binding. Desantis et al. (2011) studied this using a setup where real and confederate participants were separated by a partition but viewed identical screens [Figure 2: see original paper]. When participants believed they caused a tone (though they did not), binding increased; conversely, when they believed a confederate caused the tone (though they actually did), binding decreased. Additionally, verbally reported causal relationships produce stronger binding than non-causal reports (Buehner & Humphreys, 2009; Humphreys & Buehner, 2009). Kawabe et al. (2013) found that when tactile and visual stimuli were paired with more auditory stimuli, reports of delay decreased, indicating enhanced causation-based agency. Explicit ratings also show that stronger action-outcome causality produces greater temporal compression. Schizophrenia patients exhibit exaggerated binding effects, possibly due to aberrant causal inference (Voss, Chambon, Wenke, Kuhn, & Haggard, 2017; Voss et al., 2010). Ketamine, a psychotomimetic drug, similarly enhances binding in healthy participants (Moore et al., 2013).

Neural evidence implicates the posterior medial frontal cortex (pMFC) in action-outcome matching, while the supplementary motor area (SMA) and pre-supplementary motor area (pre-SMA) are involved in higher-level decision-making and outcome prediction. PET and fMRI studies suggest that pre-SMA may represent newly formed action-outcome associations (Elsner et al., 2002; Melcher, Weidema, Eenshuistra, Hommel, & Gruber, 2008; Ticini, Schutz-Bosbach, Weiss, Casile, & Waszak, 2012). Elsner et al. (2002) established key-tone associations through learning and found that passively hearing the learned outcome tone activated SMA, indicating its role in representing action-outcome links. EEG evidence also supports causation-based agency: larger readiness potential (RP) amplitudes in pre-SMA correlate with stronger binding (Jo, Wittmann, Hinterberger, & Schmidt, 2014), and increased outcome probability enhances P2 component amplitude (Eppinger, Kray, Mock, & Mecklinger, 2008). Our research confirms that both key pressing and lifting produce similar P2 components, likely reflecting expectation of delayed outcomes (Zhao et al., 2014).

The Relationship Between Action-Based and Causation-Based Sense of Agency in Intentional Binding

Both action-based and causation-based sense of agency play important roles in intentional binding, and these factors are not mutually exclusive—each can independently produce binding effects (Moore & Obhi, 2012; Pacherie, 2008). Accumulating evidence indicates that neither factor alone fully explains the phenomenon. In a Libet clock study comparing 75% and 50% probability conditions, researchers examined the role of expectation by comparing action time estimates when no tone occurred across these conditions. To assess causality' s importance, they compared binding magnitude between trials with and without tones in the 50% condition. Results demonstrated that both expectation and causality are necessary for binding (Moore & Haggard, 2008).

Moreover, neither action-based nor causation-based agency alone can explain differential binding effects between key pressing and lifting actions (Zhao et al., 2013; Zhao et al., 2014; Zhao et al., 2016). Key pressing provides immediate tactile feedback that generates action-based agency; removing this feedback eliminates differences between pressing and lifting, highlighting action-based agency' s contribution. However, as action-outcome intervals increase, pressing and lifting produce divergent binding effects, yet pressing still shows binding at 4-second intervals (Humphreys & Buehner, 2009), indicating that both forms of agency contribute.

The precise relationship between these two constructs remains unclear. Action-based agency emphasizes temporal compression arising from the action itself, while causation-based agency focuses on compression related to the outcome. Two hypotheses have been proposed. First, based on temporal precedence and empirical evidence, a **two-stage sequential relationship** may exist [Figure 3: see original paper]. Temporal succession suggests that action-based agency operates first, generating agency through matching predicted and actual feedback up to a certain time point, after which causation-based agency takes over through prediction and detection of action-outcome relationships. Second, a **parallel relationship** posits that action-based and causation-based agency operate simultaneously at different processing levels—action-based agency as a non-conceptual, low-level process and causation-based agency as a conceptual, high-level process based on causal inference.

The sequential model predicts a temporal boundary between the two processes. Research suggests this boundary occurs around 200 ms: when immediate sensory feedback follows a voluntary action, predicted and actual feedback match, generating action-based agency. Beyond 200 ms, participants may detect a mismatch between action and outcome, yet can still perceive causality, producing binding in both cases (Zhao et al., 2013). Within 200 ms, action-outcome matching produces action-based agency; beyond this window, causation-based agency maintains binding despite perceived mismatch. Supporting this model, Zhao et al. (2014) compared pressing and lifting conditions using interval estimation

while recording EEG. Key pressing produced stronger binding than lifting and elicited a larger P1 component, while both actions generated similar P2 components. The earlier P1 likely reflects matching between action and actual sensory feedback (action-based agency), whereas the later P2 reflects expectation of action-outcome associations (causation-based agency).

Alternatively, the parallel processing view holds that action-based and causation-based agency operate concurrently without temporal precedence. Action-based agency represents a non-conceptual, low-level process, while causation-based agency involves conceptual, high-level processing of causal relationships. From a neural perspective, action-based agency primarily involves lower-level regions like the cerebellum, which coordinates and matches actions (Bastian, 2006; Blakemore et al., 1999; Blakemore et al., 2001). Causal inference, conversely, depends on higher-level cortical regions such as pre-SMA and SMA, which predict action outcomes (Elsner et al., 2002; Melcher et al., 2008; Ticini et al., 2012).

Future Directions

This review systematically summarizes two cognitive mechanisms of intentional binding: comparator-based action agency and causation-based agency, proposing both sequential and parallel processing frameworks. Future research should further investigate their operational modes and neural substrates.

First, action-based and causation-based agency may involve distinct neural mechanisms. Action-based agency appears to specifically involve the cerebellum, while causation-based agency engages cortical regions, particularly SMA and pre-SMA, in outcome prediction. Future studies should differentiate the neural networks underlying these two mechanisms using neuroimaging techniques to identify common and distinct pathways.

Second, the operational relationship between these two forms of agency remains unresolved. Whether their influence on binding reflects parallel or sequential processing is unknown, and empirical work should examine potential interactions and overlaps between them.

Third, investigations of special populations will provide additional insights into binding mechanisms. Research on psychiatric disorders such as depression and schizophrenia will deepen our understanding. Schizophrenia patients may show exaggerated binding due to disturbed causal inference, while depression patients' reduced sense of agency may diminish binding effects. Future studies should examine intentional binding in psychiatric conditions including schizophrenia, depression, and anxiety disorders to clarify the mechanisms underlying different forms of agency.

Note: Figure translations are in progress. See original paper for figures.