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Neural mechanisms of feature binding

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An object is usually composed of different features (e.g., color, orientation, and motion), which are processed by segregated visual pathways and represented by functionally specialized brain areas. However, we perceive an object as a coherent whole, rather than its isolated features. How we integrate those isolated features and achieve a precise perception of objects is a fundamental challenge for the visual system, which is referred to as the binding problem. In particular, where and how the features are integrated together in the brain are essential unsolved questions for our understanding of the binding mechanisms.

The existence of the binding mechanisms. In the past few decades, numerous studies investigated the binding problem and the mechanisms behind it. The binding problem once was argued as an ill-posed problem (Di Lollo, 2012), given that there are many dual-selective and multiple-selective neurons throughout the visual hierarchy. Moreover, in previous studies, features were always presented simultaneously and/or superimposed (e.g., Seymour et al., 2009). Therefore, it is difficult to distinguish between a conjunctive representation of visual features and active feature binding. It is naturally to argue that the binding mechanisms might not exist in the brain (Di Lollo, 2012).

To address this issue, it is necessary to investigate how feature misbinding (or illusory conjunctions) is realized in the brain (Treisman, 1996). When multiple features are presented in the same visual space, observers sometimes integrate features in different locations erroneously and form illusory perception of non-existent objects. This is referred to as feature misbinding. Misbinding of features relies on the active binding processing, rather than the conjunctive representation of visual features, which can provide evidence that feature binding is not a fully automatic process and indicate that the binding mechanisms are actually recruited in the brain. However, feature misbinding (or illusory conjunctions) paradigms were originally based on brief stimulus presentations, which could be confounded by other factors, such as working memory, attention, and expectation. Wu and colleagues (2004) reported a compelling color-motion misbinding illusion, which could overcome these difficulties. The color-motion misbinding provides striking evidence supporting the active existence of the binding mechanisms, and it is extensively referenced in investigating where and how features are integrated in the visual system.

Early and late binding theories. There are two major theories (i.e., early binding theories and late binding theories) that have been proposed to explain the binding mechanisms. These two theories have opposite claims regarding where and how our brain solves the binding problem. Studies supporting early binding theories claim that feature binding takes place at an early stage of visual processing, even in the

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absence of attention (e.g., Seymour et al., 2009, Holcombe and Cavanagh, 2001). For example, a psychophysical study showed that spatially superimposed feature pairs could be bound even at an extremely high flickering rate (Holcombe and Cavanagh, 2001). Adapting to a particular conjunction of features could generate visual contingent aftereffects and these aftereffects occurred specifically for the adapted eye, suggesting that feature binding might occur at the early stage with retinotopic organization. Besides psychophysical studies, some neuroimaging studies also suggest that feature binding takes place in early visual cortex (e.g., Seymour et al., 2009).

Late binding theories argue that features are bound at a late processing stage and that the parietal cortex is critical for feature binding. For example, patients with lesions in the bilateral posterior parietal cortex (PPC) could perceive illusory feature conjunctions which were not presented (Braet and Humphreys, 2009). With 1 Hz rapid TMS applied to the right intraparietal sulcus (IPS), participants could make fewer binding errors (Esterman and Verstynen, 2007). An fMRI study showed that the right temporoparietal junction (TPJ) was also important for the binding of color and spatial frequency (Pollmann et al., 2014). These findings suggest that the partial cortex plays a critical role in the binding process.

Feature integration theory and reentrant processes. Feature integration theory (FIT) is a prominent theory providing a general framework of feature binding (Treisman, 1996). It is worth noting that FIT proposes that attention-dependent reentrant processes are essential for feature binding, which is different with early/late binding theories. According to FIT, visual inputs first activate low-level feature detectors. Feature detectors then communicate to object processing modules in higher cortical areas for generating feature conjunctions. To validate those feature conjunctions, the visual processing stream also needs to trace back to early visual cortex to ensure that visual features are encoded accurately. The critical role of reentrant processing in feature binding is supported by behavioral and neuroimaging studies. Bouvier and Treisman (2010) used the backward masking paradigm to inhibit feedback connections during color-orientation binding and found that subjects made more binding errors. Koivisto and Silvanto (2012) used the same stimuli and applied TMS at different time points after stimulus presentation to interfere with the binding process. Two critical time windows for feature binding were detected: 90–120 ms for feature detection, and 150–240 ms for feature binding. When TMS was applied over the angular gyrus to inhibit feedback connections during the latter time window, subjects could not finish the discrimination task, which required correct feature binding.

As we mentioned above, feature misbinding is a powerful tool to verify FIT. Zhang et al. (2014) used psychophysical and fMRI approaches to explore the cortical mechanisms of feature binding with a color-motion misbinding stimulus. The color-motion misbinding stimulus usually contained two sheets of moving dots: one with upward movement and one with downward movement. On each moving sheet, dots in the central and peripheral areas combine color and motion in opposite fashions. When fixating on the center of the display, observers erroneously perceive the dots in the periphery – the color and motion were bound in the opposite combination for most of the viewing time. They found that perception of the color-motion misbinding could induce a color-contingent motion aftereffect (CCMAE) and a color-contingent adaptation effect was detected in multiple visual areas. In particular, V2 exhibited the strongest adaptation effect and was associated with the behavioral aftereffect. Moreover, results from dynamic causal modelling (DCM) demonstrated that the enhanced feedback connections from V4 to V2 and from V5 to V2 were necessary for feature binding. Supporting evidence also exists in an event-related potential (ERP) study. Zhang et al. (2016) probed the time course of feature binding and found that the amplitude of the C1 component induced by the test stimuli decreased in its descending phase after adapting to the color-motion misbinding, which suggested that feature binding could be manifested in early visual cortex, but later than feature co-occurrence processing. Altogether, these findings support FIT and reveal a critical role of reentrant connections from specialized higher cortical areas to early visual cortex.

Neural oscillations. Regardless of the debates concentrating on the topic of where feature binding occurs, neural oscillations were proposed as a potential mechanism to solve the binding problem, specifically, to solve how features are bound together. Neural oscillation theories, which are originally referred to as neural synchronization theories, propose that features of the same object are coded through synchronized firing of neurons (von der Malsburg, 1981). Previous studies found that feature binding might be implemented through gamma band synchronization (30–100 Hz). Electrophysiological studies recorded synchronized firing at 40–60 Hz in early visual areas of cats and monkeys. With EEG or MEG, similar results were also found in the human brain, especially oscillations at 40 Hz (Tallon-Baudry and Bertrand, 1999). However, gamma oscillations usually reflect monosynaptic connections, whereas feature binding involves interregional connectivity. Therefore, the role of gamma oscillations in feature binding is still unclear.

Accumulating studies suggest that alpha activity (8–12 Hz) is associated with long-range connections and top-down modulations, which could serve as the neural basis of feature binding. Combining EEG and transcranial alternating current stimulation (tACS), Zhang et al. (2019) used the color-motion misbinding stimulus to investigate the roles of alpha oscillations in feature binding. In their study, partici-

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pants were required to report their perception (either active binding (misbinding) or physical binding) when the stimulus was presenting. They found that decreased alpha activity was correlated with a higher time proportion of the active binding, and applying tACS at individual alpha-frequency could causally determine the way of color-motion binding, suggesting that more active representations were required for the active binding than for the physical binding. This is in agreement with previous neuroimaging studies, which found that feedback connections were recruited for the active binding (e.g., Zhang et al., 2014). In addition, individual alpha-frequency was positively correlated with the perceptual switch rate between the two binding states, and applying tACS at different frequencies within the alpha band could causally change participants' perceptual switch rates. These findings suggest that alpha activity is an important neural substrate for feature binding and provide direct evidence in support of neural oscillation theories.

Based on the findings on cognitive functions of alpha and gamma oscillations, Jensen et al. (2014) proposed a more general hypothesis of neural oscillations in feature binding. They argued that the neuronal representations of disassembling visual features were due to the GABAergic inhibition and reflected in a gamma pattern, while alpha oscillations could control information flow dynamically by serving as a global inhibitory rhythm. However, more experiments are still needed to investigate the alpha-gamma coupling and testify this hypothesis.

In summary, during the past few decades, considerable progress has been made in understanding the binding problem. However, more comprehensive studies are still needed to further uncover the binding mechanisms. For example, it is necessary to further explore the binding mechanisms, such as how are the dynamics between early visual cortex and higher function specialized modules involved in feature binding? Does the alpha-gamma coupling actually serve as a potential mechanism for feature encoding and binding in the brain, based on Jensen's hypothesis (Jensen et al., 2014)? Additionally, given that all findings were mainly based on a limited number of features, such as color-motion, color-form, and color-orientation, it is necessary to test a broader range of feature-binding situations and to explore whether all features are integrated by sharing the same mechanism. Moreover, the binding problem could also be used as a bridge to connect to other cognitive functions, such as attention and awareness. It would also be of great interest to associate feature binding to neurological or psychiatric disorders.

Compliance and ethics *The author(s) declare that they have no conflict of interest.*

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