Current Biology 24, 1354–1360, June 16, 2014 ©2014 Elsevier Ltd All rights reserved http://dx.doi.org/10.1016/j.cub.2014.04.045

Report

Misbinding of Color and Motion in Human Visual Cortex

Xilin Zhang,[1,2,3] Jiang Qiu,[4] Yanyu Zhang,[1,2,3] Shihui Han,[1,2] and Fang Fang[1,2,3,*]
[1]Department of Psychology and Key Laboratory of Machine Perception (Ministry of Education), Peking University, Beijing 100871, China
[2]PKU-IDG/McGovern Institute for Brain Research, Peking University, Beijing 100871, China
[3]Peking-Tsinghua Center for Life Sciences, Peking University, Beijing 100871, China
[4]Key Laboratory of Cognition and Personality (Ministry of Education) and Faculty of Psychology, Southwest University, Chongqing 400715, China

Summary

A fundamental challenge for the visual system is to integrate visual features into a coherent scene, known as the binding problem. The neural mechanisms of feature binding are hard to identify because of difficulties in separating active feature binding from feature co-occurrence. In previous studies on feature binding [1–5], visual features were superimposed and presented simultaneously. Neurons throughout the visual cortex are known to code multiple features [6]. Therefore, the observed binding effects could be due to the physical co-occurrence of features and the sensory representation of feature pairings. It is uncertain whether the mechanisms responsible for perceptual binding were actually recruited [7, 8]. To address this issue, we performed psychophysical and fMRI experiments to investigate the neural mechanisms of a steady-state misbinding of color and motion [9], because feature misbinding is probably the most striking evidence for the active existence of the binding mechanisms [10]. We found that adapting to the color-motion misbinding generated the color-contingent motion aftereffect, as well as the color-contingent motion adaptation effect in visual cortex. Notably, V2 exhibited the strongest adaptation effect, which significantly correlated with the aftereffect across subjects. Furthermore, effective connectivity analysis using dynamic causal modeling showed that the misbinding was closely associated with enhanced feedback from V4 and V5 to V2. These findings provide strong evidence for active feature binding in early visual cortex and suggest a critical role of reentrant connections from specialized intermediate areas to early visual cortex in this process.

Results

We used a modified version of the steady-state misbinding illusion reported by Wu and colleagues [9]. Our stimuli (Figure 1A) contained two sheets of isoluminant dots, one sheet moving up and the other moving down. On both sheets, dots in the right peripheral area (right of the white dashed line, effect part) and those in the rest area (induction part) were rendered in different colors (red or green). Intriguingly, when observers fixated at the center of the stimulus, during most of the viewing time, the color and motion of the dots in the effect part were perceived to be bound in the same fashion as those in the induction part. For example, for the left stimulus in Figure 1A, on the upward-moving sheet, dots in the induction and effect parts were red and green, respectively. On the downward-moving sheet, dots in the induction and effect parts were green and red, respectively. The misbinding of color and motion made observers perceive upward-moving red dots and downward-moving green dots in the effect part.

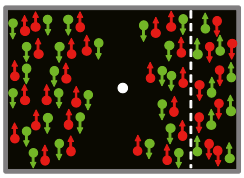
Psychophysical Experiments

In the psychophysical adaptation experiment, we used an aftereffect to investigate whether the human visual system could represent the color-motion misbinding. Adaptation is a general property of almost all neural systems. Due to its power to isolate and temporarily reduce the contribution of specific neural populations, measuring the adaptation aftereffects has been a powerful tool of psychophysics to study the representation of various visual patterns [11]. By using a method of

°/sec upward, 0.3°/sec upward, 0°/sec, 0.3°/sec downward, 0.6°/sec downward). After 30 s preadaptation and 5 s topping-up adaptation, a test stimulus was presented for 0.2 s, and subjects made a two-alternative forced-choice (2-AFC) judgment on the motion direction of the test stimulus, either upward or downward.

Because data from the red and green test stimuli showed as a similar pattern, they were pooled together for analysis. Unless otherwise stated, we present average data across 12 subjects hereafter. Figure 2A shows the psychometric functions for the two adaptation conditions. We plotted the percentage of trials in which subjects indicated directions for the test stimuli that were opposite to the perceived direction of adapting dots (which possessed the same color as the test stimuli) as a function of the real speed of the test stimulus. After subjects adapted to the induction part only (the second condition), they gave nearly perfect performances for all the test stimuli (about 50% level for the 0°/sec stimulus, good judgment for the 0.3°/sec and 0.6°/sec stimuli). However, after adaptation to the induction and effect parts (the first condition), the psychometric function showed a horizontal left shift. In other words, subjects' perception of the moving direction of the test stimuli was biased opposite to the perceived (rather than the physical) moving direction of the adapting dots (with the same color as the test stimulus), suggesting that subjects' visual cortex adapted to the misbinding. To quantitatively measure the CCMAE magnitude, we fit the psychometric

*Correspondence: ffang@pku.edu.cn



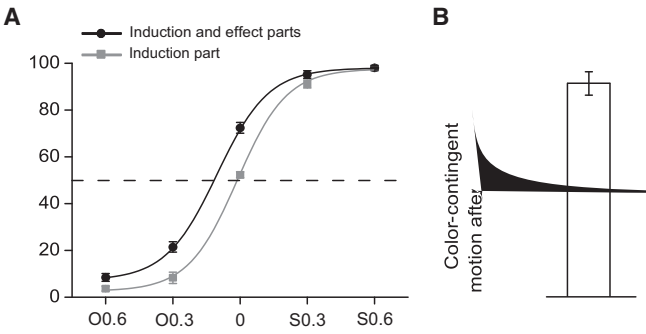
values at the five test speeds with a cumulative normal function for each adaptation condition. We interpolated the data to find the speed expected to be perceived stationary. The speed difference between the two conditions (mean ± SEM: 0.1229 ± 0.0123) was the CCMAE from adapting to the effect part, which was significantly above zero ($t_{11} = 9.996$, $p < 0.001$, Figure 2B).

In a separate experiment, we measured the percentage of time that subjects perceived the color-motion misbinding when they viewed the stimuli in the first adaptation condition. During preadaptation and topping-up adaptation, subjects pressed one of two buttons to indicate their perceptual state—correct binding or misbinding. They perceived the misbinding on average 80% of the time. Remarkably, the percentage of time was significantly correlated with the CCMAE magnitude across subjects ($r = 0.685$, $p = 0.014$, Figure 2C). Taken together, these results showed that the misbinding determined the CCMAE direction and magnitude, suggesting that neurons in visual cortex encode the color-motion misbinding for the dots in the effect part.

fMRI Experiments

To directly investigate where the misbinding is represented in the brain, an event-related fMRI adaptation experiment was designed to measure the color-contingent motion adaptation effect in cortex. Similar to the psychophysical adaptation experiment, subjects adapted to either the induction and effect parts (first condition) or the induction part only (second condition). After 30 s preadaptation and 5 s topping-up adaptation, a test stimulus was presented for 0.5 s (Figure 1C). There were two test stimuli, each containing both red and green moving dots. In one test stimulus, the dots were identical to those in the effect part of the adaptor (i.e., same trials), whereas dots in the other test stimulus moved in opposite directions to those in the effect part of the adaptor (i.e., opposite trials). For attentional control, subjects needed to press one of two buttons to indicate a 0.2 s luminance change (increase or decrease) of the test stimuli. The luminance changes were determined by adaptive staircases before scanning to ensure that subjects performed equally well for the same and opposite trials.

Regions of interest (ROIs) in V1, V2, V3, V3A/B, V4, V5, and PPC (posterior parietal cortex) were defined as the cortical regions responding significantly to the effect part. Blood-oxygenation-level-dependent (BOLD) signals were extracted from the ROIs and then selectively averaged according to the trial type. The peak BOLD signal to the test stimulus was used as a measure of the response amplitude. For each adaptation condition and each ROI, we computed an adaptation index I_A to quantify how much the measured response changed after adaptation relative to the overall response to the stimuli in the ROI. The index was calculated as



C

follows: $I_A = (A_{same} - A_{opposite})/(A_{same} + A_{opposite})$, where A_{same} and $A_{opposite}$ are the mean response amplitudes in the same and opposite trials, respectively. We hypothesized that, if a cortical area encodes the misbinding of color and motion, according to the fMRI adaptation logic, the area should show a higher response in the same trials than that in the opposite trials. The adaptation index of this area then should be higher than zero. However, if the area encodes the correct binding, it should respond in an opposite fashion, and the index should be lower than zero. Figures 3A and 3B show the BOLD signals evoked by test stimuli in the same and opposite trials and the corresponding adaptation indices. For the first adaptation condition, V1–V5 showed a higher signal for the same trials than for the opposite trials, and their adaptation indices were significantly above zero (all $t_{11} > 4.286$, $p < 0.001$). However, PPC did not show the same pattern, and its adaptation index was not significantly different from zero ($t_{11} = 0.726$, $p = 0.483$). For the second adaptation condition, none of these areas showed an index significantly different from zero (all $t_{11} < 0.919$, $p > 0.378$). The index differences between these two conditions are shown in Figure 3C, which reflects the fMRI adaptation effect to the effect part. The differences were significantly above zero in V1–V5 (all $t_{11} > 4.617$, $p < 0.002$), but not in the PPC ($t_{11} = 0.592$, $p = 0.566$). These results demonstrated that visual cortex (but not the PPC) encoded the perceived binding of color and motion rather than their physical binding.

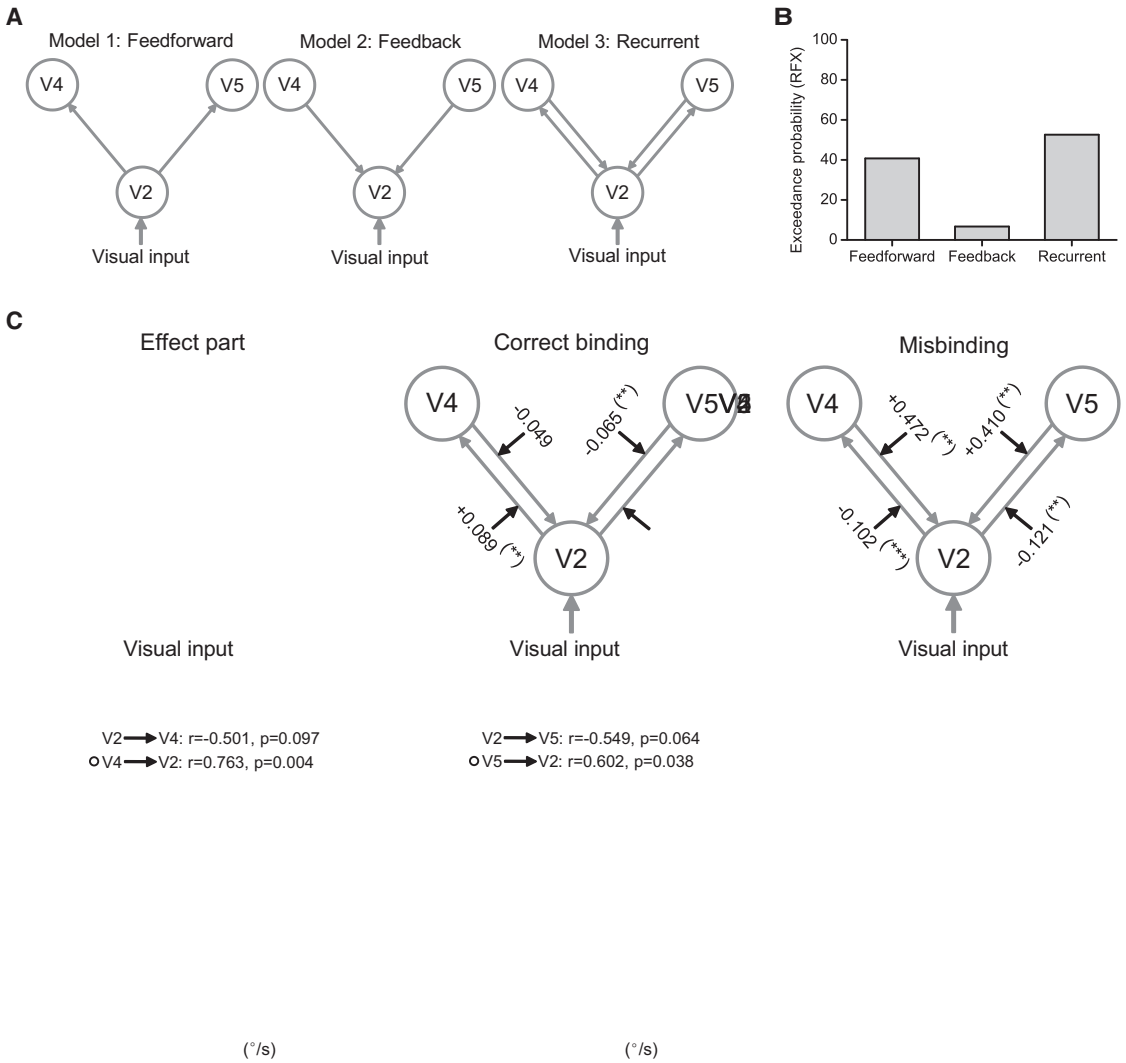
To identify the area showing the largest adaptation effect to the color-motion misbinding, we submitted the adaptation index differences to a repeated-measure ANOVA with cortical area as within-subject factor. The main effect of cortical area was significant ($F_{6, 66} = 4.443$, $p = 0.023$). Post hoc paired t tests revealed that the index difference in V2 was significantly larger than those in V1, V3, V4, V5, V3A/B, and PPC (all $t_{11} > 2.648$, $p < 0.023$). We further evaluated the role of V2 in the misbinding and calculated the correlation coefficients between the psychophysical and fMRI measures across individual subjects. The adaptation index difference in V2 correlated significantly with the CCMAE from adapting to the effect part ($r = 0.781$, $p = 0.003$, Figure 3D) and with the percentage of time subjects perceived the misbinding ($r = 0.786$, $p = 0.002$, Figure 3F), which survived Bonferroni correction for multiple tests in the seven ROIs. No significant correlation was found in any other areas (all $r < 0.576$, $p > 0.05$, Figure 3E and 3G). These results demonstrate a close relationship between the misbinding and V2 activity.

In supplemental psychophysical experiments 1 and 2 and fMRI experiments 1 and 2, we recruited eight new subjects and measured the psychophysical and fMRI adaptation effects to the correct binding and misbinding of color and motion. We found that, compared with adapting to the misbinding, adapting to the correct binding generated stronger adaptation effects, but in the opposite direction (see Figure S1 available online).

Effective Connectivity Analyses

The feature integration theory [12] proposed that attention-dependent reentrant processes are critical for feature binding. According to this theory, the first response to a visual stimulus activates feature detectors in early visual areas that connect automatically to object nodes in higher cortical areas with which they are compatible. To verify whether the feature conjunctions represented by the object nodes are real, the features must be retraced to the early areas where localization is more precise [13]. The role of reentrant processing in feature binding has gained support from behavioral experiments with the paradigm of object substitution masking [13, 14]. But no brain imaging or neurophysiological study has directly examined this issue yet.

It has been hypothesized that neurons in the superficial and deep layers of V2 that receive feedback connections from higher areas might be crucial to feature binding [15]. To test this hypothesis, we performed a second fMRI experiment and used DCM to examine functional changes in interregional connectivity related to the color-motion misbinding. The experiment used a block design, in which stimulus blocks were interleaved with blank intervals. There were three kinds of stimulus conditions or blocks: the effect part condition, the correct binding condition, and the misbinding condition. In the effect part condition, only the effect part was presented, whereas in the other two conditions, both the induction and effect parts were presented. In the correct binding condition, the colors and motions in the two parts were combined in the same way. In the misbinding condition, they were



combined in opposite ways, which was the same as that in the psychophysical adaptation experiment.

The DCM analysis focused on directional connectivities between V2 and V4 and between V2 and V5 based on BOLD signals from these areas. V4 and V5 were selected because they are functionally specialized areas for processing color and motion, respectively. DCMs have three sets of parameters: (1) extrinsic input into one or more regions, (2) intrinsic connectivities among the modeled regions, and (3) bilinear parameters encoding the modulations of the specified intrinsic connections by experimental manipulations [16]. Given the extrinsic input into V2, we defined feedforward, feedback, and recurrent models between V2 and V4 and between V2 and V5 (Figure 4A). The intrinsic connectivity patterns in the DCMs were modeled and then compared by computing the exceedance probability of each model. The result showed that the feedforward, feedback, and recurrent models had exceedance probabilities of 40.71%, 6.70%, and 52.59%, respectively, suggesting that the recurrent model best explains the overall data (Figure 4B).

We next examined modulatory connections in the recurrent model. In DCM, modulatory connections reflect increases or decreases in connectivity between two regions given some experimental manipulation, compared with the intrinsic connections between the same regions that capture connectivity in the absence of experimental manipulation [16]. Figure 4C shows the modulatory connectivities in the three conditions. The effect part condition and the correct binding condition evidenced a similar pattern. The correct binding condition increased the forward connectivities from V2 to V4 (t_{11} = 3.408, p = 0.006) and from V2 to V5 (t_{11} = 8.653, p < 0.001) and decreased the backward connectivity from V5 to V2 (t_{11} = 4.569, p = 0.001). The effect part condition also increased the forward connectivities from V2 to V4 (t_{11} = 5.937, p < 0.001) and from V2 to V5 (t_{11} = 8.653, p < 0.001). However, the misbinding condition showed an opposite pattern: it decreased

the forward connectivities from V2 to V4 (t_{11} = 7.358, $p < 0.001$) and from V2 to V5 (t_{11} = 4.512, p = 0.001), but increased the backward connectivities from V4 to V2 (t_{11} = 4.323, p = 0.001) and from V5 to V2 (t_{11} = 4.190, p = 0.002).

To further evaluate the role of these forward and backward connectivities in the color-motion misbinding, we calculated the correlation coefficients between the CCMAE and the effective connection strengths (the sum of the modulatory and intrinsic connectivities) across subjects (Figures 4D and 4E). The CCMAE was positively correlated with the backward connectivities from V4 to V2 (r = 0.763, p = 0.004) and from V5 to V2 (r = 0.602, p = 0.038), but its correlations with the forward connectivities were negative and only marginally significant (from V2 to V4: r = −0.501, p = 0.097; from V2 to V5: r = −0.549, p = 0.064). Taken together, the DCM results suggest that the effective connectivities between V2 and both V4 and V5 (especially the enhanced feedback) might significantly contribute to the misbinding.

Discussion

Our study provides the following psychophysical and neuroimaging findings. First, adapting to the color-motion misbinding generated a CCMAE, the magnitude of which was determined by the strength of the misbinding experienced by individual subjects. Second, the visual cortex, but not the PPC, exhibited the color-contingent motion adaptation effect to the misbinding. The adaptation effect in V2 significantly correlated with the CCMAE. Third, the misbinding was accompanied by decreased forward connectivities from V2 to both V4 and V5 and by increased backward connectivities from both V4 and V5 to V2. The backward connectivities were also closely associated with the CCMAE. The most parsimonious account of these results is that V2 is more important than other cortical areas for representing the color-motion misbinding and that this representation is likely caused by feedback from brain regions upstream along the visual pathway (i.e., V4 and V5).

We believe that our fMRI results cannot be explained by attention. In the first experiment, subjects were asked to detect the luminance change of the test stimuli. The luminance changes in the same and opposite trials were almost identical (Figure S2A). Behavioral data showed that there was no significant performance difference between the two types of trials (Figures S2B and S2C), suggesting that there is no difference in task difficulty and, presumably, attention. Supplemental psychophysical experiment 3 and fMRI experiment 3 provide further evidence against the attention explanation (see Figure S3). For the second experiment, it could be argued that, in the misbinding condition, perceptual switches between the misbinding and the correct binding in the effect part draw more attention and thus lead to the observed connectivity changes. We performed additional DCM and ROI analyses to rule out this explanation. First, because PPC is a critical part of the frontoparietal attention system, we estimated modulatory connectivities from PPC to V2, V4, and V5 for the misbinding, correct binding, and effect part conditions. All these connectivities were significant, but no significant difference was found among these three conditions (Figures S4A and S4B). Moreover, there was no significant correlation between the connectivities and the CCMAE magnitude (Figure S4C). Second, we calculated BOLD signal amplitudes for these three conditions from the ROIs in V1–V5 and PPC representing the effect part. No significant amplitude differences among the three conditions were found in any of these ROIs (Figure S4D). If perceptual switches in the effect part had drawn attention, then we should have observed stronger BOLD signals and modulatory connectivities in the misbinding condition than the correct binding condition. Furthermore, we carried out supplemental fMRI experiment 4, which was identical to the second fMRI experiment except that we scanned the subjects' entire brain (only the posterior part of the brain was scanned in the second fMRI experiment). We performed a group analysis and did a whole-brain search for cortical area(s) that showed differential responses in the misbinding and correct binding conditions. No such area was found.

Recently, Seymour and colleagues [17, 18] applied multivariate classifiers to voxel activation patterns obtained when subjects viewed feature conjunctures, including color-motion and color-form conjunctions. They found that the conjunctions could be decoded from spatial activation patterns in early visual cortical regions, as early as V1. Their results demonstrated an explicit coding of conjunctions at early visual processing stages and implied an early mechanism of visual feature binding. However, these findings cannot disentangle whether the conjunction information in early visual cortex corresponds to the sensory coding of specific feature pairings or to the perceptual readout of a binding operation. It is still uncertain whether the mechanism of feature binding is indeed recruited for these unambiguous visual stimuli (e.g., red dots rotating clockwise, green dots rotating counterclockwise). Therefore, in order to unravel the binding mechanism, we have to rely on stimuli that can induce feature misbinding.

Feature misbinding (i.e., illusory conjunction) usually occurs when stimulus exposures are brief and attention is overloaded [10]. The poor spatial resolution of peripheral vision can also give rise to binding failures when stimuli are presented peripherally, even with long exposures and no competing attention task [19]. The stable misbinding illusion described by Wu et al. [9] significantly extends the finding by Prinzmetal et al. [19]. It also avoids the common confounds of memory, expectation, and task strategy found in many previous feature misbinding studies [2]. Why does this illusion occur? It is likely to be the result of an ambiguity-resolving mechanism, reflecting the probabilistic integration of prior object knowledge with image features [20]. The contiguity of the induction and effect parts and the equal values of color and motion presented across the stimulus strongly imply that the induction and effect parts belong to the same surface, especially when the effect part is presented in the periphery and its percept becomes ambiguous. Because vision is clearest at a fixation point, it is advantageous to use to the information in the induction part to make inferences about the properties of the effect part and to resolve the ambiguity. Thus, feature binding in the effect part follows the induction part, although the binding is erroneous [9].

What can the fMRI findings in the current study tell us about the neural implementation of the color-motion misbinding? Neurophysiological studies have shown that color and motion are processed in largely distinct, yet mutually connected processing streams [21]. On one hand, color is primarily processed in the blobs of V1, in the thin stripes of V2, and in the human V4 complex, while motion is primarily processed in layer 4B of V1, in the thick stripes of V2 and in the V5/MT$^+$ complex [22, 23]. On the other hand, dually selective neurons for color and motion direction were found in V1 [6] and more frequently in V2 [24]. Furthermore, Shipp et al. [15] reported that dual-selective V2 neurons for color and motion are much

more common in the superficial and deep layers (1, 2, 5, and 6), which receive feedback connections from V4 and V5, compared with the middle layers (3 and 4), which relay ascending signals. This finding suggests that, relative to feedback pathways, color and motion processing in V2 are more independent in feedforward pathways. Color and motion bound initially in the feedforward pathways could be reintegrated in the feedback layers. Taking into account the neurophysiological findings and our fMRI findings, we might speculate the neural implementation of the misbinding as follows. When subjects view the stimulus in the feedforward pathways, visual information in the induction and effect parts is processed independently. Colors and motion directions are initially bound according to the physical property of the stimulus. When these ascending color and motion signals reach V4 and V5, color-defined surfaces and motion-defined surfaces across the induction and effect parts form in these two areas, respectively [25–27]. Note that the unitary surfaces are not consistent with the physical binding of colors and motion directions in the effect part area. To solve this problem, the surface information guides feedbacks to the superficial and deep layers of V2 and activates neurons that are responsive to the effect part area and are also selective for the color-motion conjunction in the induction part. The V2 reactivation leads to the misbinding illusion. This feedback process is implied by the increased backward connectivities from V4 and V5 to V2, as revealed by the DCM analysis. The decreased forward connectivities in the opposite directions are sensible because the veridical perception reflected by the forward connections has been replaced by the misbinding illusion. It should be noted that our speculation only provides a possible mechanism for the color-motion misbinding, which should be tested with neurophysiological techniques in the future.

In sum, our study provides strong evidence for active feature binding in early visual cortex and implies a critical role of reentrant processing in this process [12]. In the future, various misbinding conditions should be investigated to fully understand the solution of the binding problem, which might also be the solution to the mystery of consciousness.

Supplemental Information

Supplemental Information includes four figures and Supplemental Experimental Procedures and can be found with this article online at http://dx.doi.org/10.1016/j.cub.2014.04.045.

Acknowledgments

We thank Yang Sun for helpful discussion. This work was supported by the Ministry of Science and Technology of China (2011CBA00400 and 2010CB833903), the National Natural Science Foundation of China (Project 30925014 and 31230029), and the Key Discipline Fund of National 211 Project (Southwest University, NSKD08019).

Received: November 12, 2013
Revised: April 9, 2014
Accepted: April 22, 2014
Published: May 22, 2014

References

1. Holcombe, A.O., and Cavanagh, P. (2001). Early binding of feature pairs for visual perception. Nat. Neurosci. *4*, 127–128.
2. Wolfe, J.M., and Cave, K.R. (1999). The psychophysical evidence for a binding problem in human vision. Neuron *24*, 11–17, 111–125.
3. Stromeyer, C.F., 3rd. (1972). Contour-contingent color aftereffects: retinal area specificity. Am. J. Psychol. *85*, 227–235.
4. Mayhew, J.E.W., and Anstis, S.M. (1972). Movement after-effects contingent on color, intensity and pattern. Percept. Psychophys. *12*, 77–85.
5. Blaser, E., Papathomas, T., and Vidnyánszky, Z. (2005). Binding of motion and colour is early and automatic. Eur. J. Neurosci. *21*, 2040–2044.
6. Gegenfurtner, K.R., Kiper, D.C., and Fenstemaker, S.B. (1996). Processing of color, form, and motion in macaque area V2. Vis. Neurosci. *13*, 161–172.
7. Di Lollo, V. (2012). The feature-binding problem is an ill-posed problem. Trends Cogn. Sci. *16*, 317–321.
8. Whitney, D. (2009). Neuroscience: toward unbinding the binding problem. Curr. Biol. *19*, R251–R253.
9. Wu, D.A., Kanai, R., and Shimojo, S. (2004). Vision: steady-state misbinding of colour and motion. Nature *429*, 262.
10. Treisman, A., and Schmidt, H. (1982). Illusory conjunctions in the perception of objects. Cognit. Psychol. *14*, 107–141.
11. He, D., Kersten, D., and Fang, F. (2012). Opposite modulation of high- and low-level visual aftereffects by perceptual grouping. Curr. Biol. *22*, 1040–1045.
12. Treisman, A. (1996). The binding problem. Curr. Opin. Neurobiol. *6*, 171–178.
13. Bouvier, S., and Treisman, A. (2010). Visual feature binding requires reentry. Psychol. Sci. *21*, 200–204.
14. Koivisto, M., and Silvanto, J. (2011). Relationship between visual binding, reentry and awareness. Conscious. Cogn. *20*, 1293–1303.
15. Shipp, S., Adams, D.L., Moutoussis, K., and Zeki, S. (2009). Feature binding in the feedback layers of area V2. Cereb. Cortex *19*, 2230–2239.
16. Friston, K.J. (2006). Dynamic causal models for fMRI. In Statistical parametric mapping: the analysis of functional brain images, K.J. Friston, ed. (Amsterdam: Elsevier), pp. 541–560.
17. Seymour, K., Clifford, C.W.G., Logothetis, N.K., and Bartels, A. (2009). The coding of color, motion, and their conjunction in the human visual cortex. Curr. Biol. *19*, 177–183.
18. Seymour, K., Clifford, C.W.G., Logothetis, N.K., and Bartels, A. (2010). Coding and binding of color and form in visual cortex. Cereb. Cortex *20*, 1946–1954.
19. Prinzmetal, W., Henderson, D., and Ivry, R. (1995). Loosening the constraints on illusory conjunctions: assessing the roles of exposure duration and attention. J. Exp. Psychol. Hum. Percept. Perform. *21*, 1362–1375.
20. Kersten, D., Mamassian, P., and Yuille, A. (2004). Object perception as Bayesian inference. Annu. Rev. Psychol. *55*, 271–304.
21. Felleman, D.J., and Van Essen, D.C. (1991). Distributed hierarchical processing in the primate cerebral cortex. Cereb. Cortex *1*, 1–47.
22. Bartels, A., and Zeki, S. (2000). The archifiD[(CodTJ-2rs)-206.3 0(Cliff-10.3rpp,)-433.2(S.,)