Does face detection in the superior colliculus rely on input from the primary visual cortex?

David Y. Mekhaiel,[1,2,3,6,*] Brian D. Corneil,[2,3,4,5] and Melvyn A. Goodale[2,3,4,*]

[1]Graduate Program in Neuroscience, Western University, London, Ontario, Canada.
[2] Western Institute for Neuroscience, Western University, London, Ontario, Canada.
[3] Department of Psychology, Western University, London, Ontario, Canada.
[4] Department of Physiology and Pharmacology, Western University, London, Ontario, Canada.
[5] Robarts Research Institute, London, Ontario, Canada.
[6] Lead Contact.
*Correspondence: dmekhaie(at)uwo.ca (D.Y.M.), mgoodale(at)uwo.ca (M.A.G.)

Keywords: Superior Colliculus, Faces, Sprague Effect, Blindsight, Electrophysiology

Abstract

In a recent study, Yu and Katz et al. (2024) argue that face-selective visual activity in the superior colliculus (SC) relies on input from the primary visual cortex (V1), based on the loss of visual responses following a temporary lesion to the lateral geniculate nucleus. We argue that alternative explanations deserve consideration. Research on visual recovery after V1 damage (Blindsight) suggests that subcortical pathways, including the SC, can contribute to face detection without V1 involvement. Additionally, the well-documented Sprague Effect, which highlights interhemispheric inhibition of SC activity following ipsilateral V1 damage, may account for the observed suppression of SC visual responses. Further investigation into these mechanisms could help clarify whether SC face detection can operate independently of V1 input.

Main text

In an elegant and carefully designed set of experiments, Yu and Katz et al. (Yu et al. 2024) (hereafter referred to as Y&K) examined in detail the category specificity of visually-driven neurons in the superficial and intermediate layers of the superior colliculus (SC) of the monkey, finding that some neurons exhibit a remarkably short-latency preference for faces. The presence of these neurons aligns with extensive research showing that this midbrain structure is a crucial "*first responder*" for the detection of evolutionarily significant stimuli in a wide range of animals, from amphibians to mammals (for review, see Hafed et al. 2023). One key question that Y&K addressed is whether the surprisingly short-latency specificity for faces relies on input from primary visual cortex (V1) or is driven instead by direct input from the retina via the ancient retinocollicular tract. After all, the SC in higher primates, including humans, receives far more visual input from V1 than it does directly from the retina.

Y&K began by recording SC activity while the monkey viewed images of various categories, including faces. They found evidence of object preferences in two phases of visual activity: an early phase peaking around 50ms, in which category-specific neurons overwhelmingly preferred faces (92%), and a later phase peaking at 100ms or more, in which object preference was roughly split amongst faces, fruit, and even human-made objects. Although the late-phase activity could easily reflect input following cortical processing, the early-phase neurons had a median latency of 40ms, much shorter than the latency of face- and object-selective activity in inferotemporal cortex, which typically ranges from 80 to 100ms after stimulus onset. This raises the possibility that the early face-selective activity, in contrast to the late-phase categorical responses, could arise from direct retinal input rather than input from V1 or other cortical visual regions.

To test this idea, they created a large but temporary unilateral muscimol lesion in the lateral geniculate nucleus (LGN) thereby blocking visual signals conveyed by this thalamic structure to V1. This should, in principle, eliminate V1 inputs to the SC, but not interfere with the direct retinal input, which, they predicted, would continue to drive the early face-selective neurons in SC. Surprisingly, they found that visual responses in the SC neurons were almost entirely abolished by the muscimol injection, including the responses of the early face-selective neurons. This result held even for neurons in the superficial layers of the SC receiving direct retinotectal inputs. Based on this result, combined with computational modelling of putative visual processing in V1, they concluded that face detection in the SC, even the earliest face-selective activity, requires that visual information be first processed by LGN and V1. They went on to suggest that the direct retinal inputs “appear insufficient to evoke visual responses in SC on their own.” As compelling as this conclusion might appear, we believe that the evidence presented by Y&K is insufficient to make this strong claim.

A large body of literature, for example, has documented recovery of visual function following V1 lesions in patients (for review, see Danckert, Striemer, and Rossetti 2021). Although much of the recovery in young animals and children is undoubtedly due to the re-organization of visual pathways, residual visual abilities can also emerge in adults with V1 lesions after a relatively short period of recovery. Moreover, a sizable body of literature has also documented sensitivity to faces and facial affect in some individuals with damage to V1. Patients who subsequently develop what has been termed “affective blindsight” after V1 lesions are able to detect faces and emotional expressions at above chance levels. Importantly, these patients also exhibit increased functional activity in the SC as well as functional connectivity between the ipsilesional SC and amygdala when viewing faces (Ajina, Pollard, and Bridge 2020). This evidence strongly suggests that there exists a subcortical circuit for face detection involving the SC that is independent of V1.

Why then did Y&K observe a cessation of collicular visual activity following LGN inhibition? We suggest that this observation may be attributable to the “Sprague Effect.” First described by James M. Sprague in the cat, the eponymous Sprague Effect is a phenomenon whereby a failure to exhibit visuomotor responses towards contralesional visual stimuli after unilateral damage to V1 is rescued by a contralesional SC lesion (Sprague 1966). Since its initial discovery, similar effects have been observed in other species, including not only rodents and cats (Valero-Cabré et al. 2020), but possibly humans as well (Weddell 2004). The Sprague Effect is thought to be due to interhemispheric rivalry in the form of mutual inhibition between the two colliculi, which if not adequately balanced by intrahemispheric excitation from V1, results in too much inhibition of activity in the ipsilesional SC, thus impeding the generation of orienting movements. Simply put, by removing the facilitatory input from V1 to the ipsilesional SC, inhibition from the other SC can overwhelm the responsiveness of visually driven collicular neurons, thereby eliminating orienting behaviour to visual stimuli in contralesional space, and presumably visually evoked activity in the SC. It is worth noting that the Sprague Effect is not limited to vision. Unilateral lesions of the auditory cortex in the cat result in a lack of auditory orienting responses to the contralesional side, without affecting visual orienting responses (Lomber, Malhotra, and Sprague 2007). This deficit, likewise, can be rescued by lesioning the contralesional SC.

Thus, the failure of Y&K to observe face-specific activity in the SC and indeed all visually driven activity (as well as any orienting eye movements to stimuli presented in the scotoma created by the muscimol injection in LGN) might reflect a general inhibition of visual activation in the ipsilesional SC – including visual activation driven by direct retinal input. Until this possibility is ruled out, it is premature to conclude the face-specificity of visual neurons in SC is dependent on V1 input. Although Y&K reported that LGN inactivation did not change the baseline activity of visually responsive SC neurons, as might have been expected by a general inhibition of the SC, inhibition due to the Sprague effect may only be evident when visual information arrives.

Finally, it is worth noting that not all studies of the contribution of V1 to visual activity in the SC can be explained by a possible Sprague effect. Early work by Schiller and colleagues (Schiller et al. 1974) found that temporary cooling of V1 in anaesthetised monkeys completely eliminated visual activity in the deeper layers of SC, but visual activity in the superficial layers of SC, the main recipient zone of direct retinal input, persisted. Visual activity in the superficial layers was even present after a permanent ablation of V1, following a recovery period of 5 days or more. In a subsequent study, Schiller's lab replicated these findings by precisely injecting lidocaine into the magnocellular layers of LGN (Schiller, Malpeli, and Schein 1979). Again, visual activity in the deeper SC layers was essentially absent whereas visual activity in the superficial layers remained. This pattern of results stands in stark contrast to the findings of Y&K who found that all visual activity in the SC of awake monkeys was eliminated after a large muscimol injection into the LGN.

The careful work by Y&K, in which they controlled for many low-level visual features and oculomotor confounds that could have contaminated any conclusions about the category-specific nature of visual activity in SC, offers needed clarity on the role of the SC in mediating rapid orienting movements to biologically relevant stimuli. Their demonstration that faces preferentially evoke low-latency (40ms) visual responses in the SC challenges the conventional cortico-centric models of face detection. At the same time, the complete muting of any and all SC visual activity after muscimol lesions in LGN is not conclusive evidence for their claim that inputs from V1 are necessary for this rapid face detection by the SC. A more convincing approach would be to assess the presence or absence of face-selectivity, particularly in the superficial layers, once visual responses in the SC are restored.

Future studies could examine this question by replicating the Sprague Effect, combining temporary or chronic lesions of LGN or V1 with lesions of the contralesional SC or V1, thereby reducing collicular inhibition. If the visual responsiveness to faces and non-faces is indistinguishable, it would then follow that SC face-selectivity truly depends on input from V1. Alternatively, if SC face- selectivity persists, it would point to the existence of subcortical feature detectors for these evolutionarily relevant stimuli that function independently of V1. Whatever the outcome might be, it is clear that the SC plays a pivotal role in the rapid detection of evolutionarily relevant stimuli, such as faces.

Acknowledgments

This work is supported by Discovery Grants to BDC (NSERC; RGPIN 311680 and 04394-2021) and MAG (NSERC; RGPIN-2017-04088) from the Natural Sciences and Engineering Research Council of Canada and an Operating Grant to BDC from the Canadian Institutes of Health Research (CIHR; MOP-93796).

Declaration of interests

The authors declare no competing interests.

References

Ajina, Sara, Miriam Pollard, and Holly Bridge. 2020. “The Superior Colliculus and Amygdala Support Evaluation of Face Trait in Blindsight.” *Frontiers in Neurology* 11 (July):769.

Danckert, J., Striemer, C. and Rossetti, Y., 2021. Blindsight. In *Handbook of clinical neurology* (Vol. 178, pp. 297-310). Elsevier.

Hafed, Ziad M., Klaus-Peter Hoffmann, Chih-Yang Chen, and Amarender R. Bogadhi. 2023. “Visual Functions of the Primate Superior Colliculus.” *Annual Review of Vision Science* 9 (September):361–83.

Lomber, Stephen G., Shveta Malhotra, and James M. Sprague. 2007. “Restoration of Acoustic Orienting into a Cortically Deaf Hemifield by Reversible Deactivation of the Contralesional Superior Colliculus: The Acoustic ‘Sprague Effect.’” *Journal of Neurophysiology* 97 (2): 979–93.

Schiller, P. H., J. G. Malpeli, and S. J. Schein. 1979. “Composition of Geniculostriate Input to Superior Colliculus of the Rhesus Monkey.” *Journal of Neurophysiology* 42 (4): 1124–33.

Schiller, P. H., M. Stryker, M. Cynader, and N. Berman. 1974. “Response Characteristics of Single Cells in the Monkey Superior Colliculus Following Ablation or Cooling of Visual Cortex.” *Journal of Neurophysiology* 37 (1): 181–94.

Sprague, J. M. 1966. “Interaction of Cortex and Superior Colliculus in Mediation of Visually Guided Behavior in the Cat.” *Science* 153 (3743): 1544–47.

Valero-Cabré, Antoni, Monica N. Toba, Claus C. Hilgetag, and R. Jarrett Rushmore. 2020. “Perturbation-Driven Paradoxical Facilitation of Visuo-Spatial Function: Revisiting the ‘Sprague Effect.’” *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior* 122 (January):10–39.

Weddell, Rodger A. 2004. “Subcortical Modulation of Spatial Attention Including Evidence That the Sprague Effect Extends to Man.” *Brain and Cognition* 55 (3): 497–506.

Yu, Gongchen, Leor N. Katz, Christian Quaia, Adam Messinger, and Richard J. Krauzlis. 2024. “Short-Latency Preference for Faces in Primate Superior Colliculus Depends on Visual Cortex.” *Neuron* 112 (June):1–9.