learns surprisingly rapidly[14], whereas chimpanzees and capuchin monkeys seem to take many trials to learn a similar task[12,15].

It remains to be seen whether chimpanzees and corvids show the sophistication of Blaisdell's rats when reasoning using the general principles of causation. As Blaisdell and colleagues point out, it may be that an animal's apparent failure to display causal understanding in some tasks of physical cognition is a reflection of the demands that the task places on an animal's knowledge of the physical world, rather than on its capacity to reason about causes. It may seem surprising that the humble rat is capable of such causal understanding. Perhaps even more surprising is that this cognitive sophistication was manifest within the artificiality of the Skinner box, which has been claimed to functionally decorticate the rat. Clearly, this is not the case.

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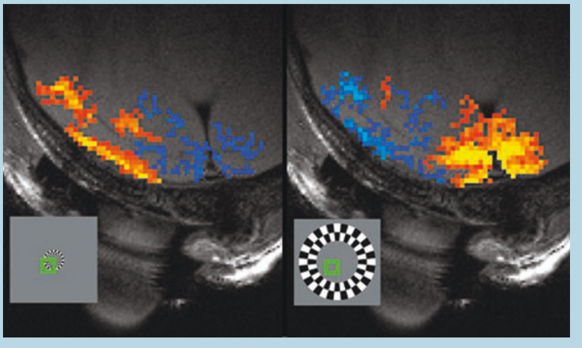
Going down BOLDly



Attractive pictures from functional brain imaging studies showing localized increases in hemodynamic responses have been the belle of the ball, but task-induced decreases in hemodynamic responses have been treated as something of the ugly stepsister. Part of the reason for this neglect is that much less is understood about the basis of such decreases. A new study on page 569 of this issue puts this negative hemodynamic response under the spotlight.

Functional MRI studies commonly measure the blood oxygenation level–dependent (BOLD) signal, and increases in this signal are thought to reflect increases in the underlying neural activity. However, fMRI studies often find a negative BOLD (the BOLD signal dips below the resting baseline) in response to certain tasks, and it is unclear if this negative BOLD response also reflects neural activity or a non-neural process. Although there is experimental support for the view that the negative BOLD response reflects suppression of neural activity, the competing 'vascular blood steal' hypothesis claims that such reductions are due to decreased blood flow with a vascular origin, and the negative BOLD response has little direct relation to underlying neural activity.

Amir Shmuel and colleagues used simultaneous functional MRI and electrophysiological recordings in monkeys to show that the negative BOLD signal is closely coupled to decreases in neural activity. These researchers directly recorded neural activity in primary visual cortex V1 using electrophysiological recordings, while also recording BOLD responses through MRI. During these recordings, the animals saw stimuli that either did or did not overlap with the receptive fields of neurons near the recording site. (The picture shows the circular checkerboard stimuli used, and the green square represents the aggregate receptive fields of these neurons.) As expected, the stimulus overlapping with the neuron receptive fields elicited a positive BOLD response (orange in left panel). However, the nonoverlapping stimulus resulted in a negative BOLD response (blue in right panel). In accord with previous work, the positive BOLD response correlated with increases in neural response as indexed by the electrophysiological recordings. However, the nonoverlapping stimulus resulted in decreases in neuronal activity in the same region of V1. Crucially, the negative BOLD correlated with these decreases in spiking activity and local field potentials.

Although this result suggests a neural origin for the negative BOLD response, it does not completely rule out the vascular blood steal hypothesis: decreased blood flow could result in hypoxia, preventing neurons from maintaining their baseline activity. Decreased blood flow would then also result in a negative BOLD response—the relationship between the negative BOLD and the decreases in neural activity would then be due to an unrelated third factor. However, the onset of the decreases in neural activity closely followed the stimulus presentation, similar to the dynamics of the increased neural activity. The negative BOLD response also lagged behind the decrease in neural activity (also similar to the lag between increased neural activity and the positive BOLD response), making it unlikely that reduced cerebral blood flow is the cause of these results. These findings therefore strengthen the view that the negative BOLD response is connected to underlying decreases in neural activity. Further work is needed to confirm that this explanation applies to areas outside V1 as well. Nonetheless, these results help strengthen the idea that negative BOLD responses also yield useful information about neuronal activity.

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