This last claim, a crucial one for their argument, is erroneous. Only the delay-and-correlate subcomponent of the Reichardt motion unit is velocity-tuned [6]. Full Reichardt detectors, which compute the difference between subcomponents preferring opposite directions of motion, are temporal-frequency tuned [7]. Furthermore, although the subcomponents are indeed tuned to the velocity of a pattern moving in the correct direction, they do not show velocity tuning when responding to a pattern moving in the wrong direction. To see why, imagine that the delay-and-correlate subcomponent is presented with a moving periodic pattern of dots (as in Figure 1A of [2]). First, a dot stimulates the delayed input line of the correlator. Next, although the pattern moves in the ‘non-preferred’ direction, a second, trailing dot stimulates the undelayed input line at exactly the time necessary to activate the correlator. If the spatial frequency of this hypothetical dot pattern were lowered, the stimulus velocity would have to be increased in order to continue stimulating the detector. This demonstrates that the correlator’s activity is not velocity-tuned for motion in the ‘non-preferred’ direction.

A separate discrete sampling process is therefore not necessary to explain the IMR. The 10–15 Hz tuning of the illusion [4] coincides with the overall frequency tuning of normal human motion sensitivity [8]. This is compatible with the Kline et al. theory of rivalry between oppositely-tuned motion detectors [2]. Prolonged stimulation would lead to extreme adaptation of motion units, especially when that stimulation is presented at the temporal frequency for which the system is most sensitive. In turn, this could occasionally allow relatively unadapted detectors selective for the reverse direction to drive the percept.

The wheels keep turning

Reply to Holcombe et al.

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In their original paper, Kline et al. [1] explained the wagon-wheel illusion in continuous light (WWIc) [2] in terms of Reichardt motion detectors [3]. We initially questioned this conclusion because such detectors are primarily tuned to velocity rather than temporal frequency [4], whereas the preferred temporal behavior of the WWIc remains constant over a range of spatial frequencies [5,6]. The authors now counter that their case rested on a subcomponent of Reichardt detectors that is velocity tuned, but only in the forward direction, and that the full detector is in fact sensitive to temporal frequency [7]. Thus, they argue, aliasing of such detectors remains a viable explanation.

It is indeed possible that a subset of detectors with appropriate spatio-temporal parameters could induce competition within a population of motion detectors, and that such rivalry might generate epochs of veridical motion and reversed motion [1]. There are, however, important weaknesses in this line of argument. First, there is no evidence that Reichardt detectors exist in the mammalian visual system. Second, this sort of mechanism would have to explain why the illusion occurs at a similar temporal frequency for both first- and second-order motion [5], which is difficult to explain given that the optimal temporal sensitivity to first- and second-order motion is markedly different [8]. Finally, it is not clear how Reichardt detectors could account for the dependence of the WWIc on attention [5]. What is clear from this exchange is that physiological evidence rather than further speculation will be needed to establish why a stimulus moving in one direction is periodically perceived.
to be moving in the opposite direction, and what this phenomenon indicates about the way the visual system parses time.

References

1 Kline, K. et al. (2004) Illusory motion reversal is caused by rivalry, not by perceptual snapshots of the visual field. *Vis. Res.*, 44, 2653–2658

The origin(s) of confabulation

An amnesic patient with Korsakoff's syndrome is asked to chronicle events of the previous weekend; she describes to her doctor, with full conviction and in vivid detail, a plane ride she took while visiting long-lost friends, when in fact she had never left her hospital room. A stroke patient, suffering from paralysis of the left side of his body, is asked to move his left arm and replies 'I don't feel like it' or 'I have never been ambidextrous'. He further denies having any problem with his arm, despite the clear evidence that he is unable to perform this simple task. A neurologically intact individual is asked to recite a list of recently presented words that are semantically associated (e.g. bed, rest, awake, etc.) and happens to recall a related word that was never actually presented (e.g. sleep), again with absolute conviction. What do these three seemingly unrelated anecdotes have in common? In each case the individual is not lying. They all claim that what they are reporting is true. But what exactly is happening? How do these individuals come to overlook the reality of their respective situations? This is the question that philosopher William Hirstein has attempted to answer in his new book *Brain Fiction: Self-Deception and the Riddle of Confabulation*.

Separate literatures have grown up around these and various other manifestations of confabulation (e.g. misidentification syndromes, split-brain patients, sociopathy), in many cases through the use of case studies. Here, the author takes on the lofty goal of bringing them all together. In so doing, he wants to focus on the commonalities among these various forms of confabulation, weaving together a story around the data coming from neuropsychology, neuroscience, and behavioral studies of memory.

Understanding confabulation is in one respect similar to the attempt to properly classify a recently discovered animal species – it must be placed in a proper family' (p. 71).

The argument for this ‘single entity’ point of view is largely rooted in the argument that confabulation represents a breakdown in reality monitoring [1]. That is, these individuals are all unable to recognize their reports as ill-grounded. Just as reality monitoring breakdowns tell us something about how memory works, Hirstein argues that confabulation tells us something fundamental about people. ‘The phenomenon contains important clues about how humans assess their thoughts and attach either doubt or certainty to them’ (p. 4).

In focusing upon the commonalities among all these cases, the author further identifies the orbitofrontal cortex as the key player that likely underlies the deficits shared by such patients. Indeed, it is well known that damage to regions within orbitofrontal cortex can produce a form of disinhibition in a formerly healthy individual (as is the case with acquired sociopathy), and orbitofrontal cortex has long been thought to play an inhibitory role in the production of thoughts into actions [2].

However, just as it is unclear if all breakdowns in reality monitoring can be traced back to a single cause, it is unclear whether confabulation can be boiled down essentially into one phenomenon. Although many would agree that monitoring processes are involved and that the orbitofrontal cortex underlies some of these processes, many would also argue that there exist many monitoring processes and that there are many brain regions underlying these various processes. For instance, the orbitofrontal cortex itself is not a monolithic entity [3]; differing loci of damage likely lead to differing deficits.

Further difficulties arise in consideration of the disparate neuroanatomical etiologies of confabulatory