

# The virtue of simplicity

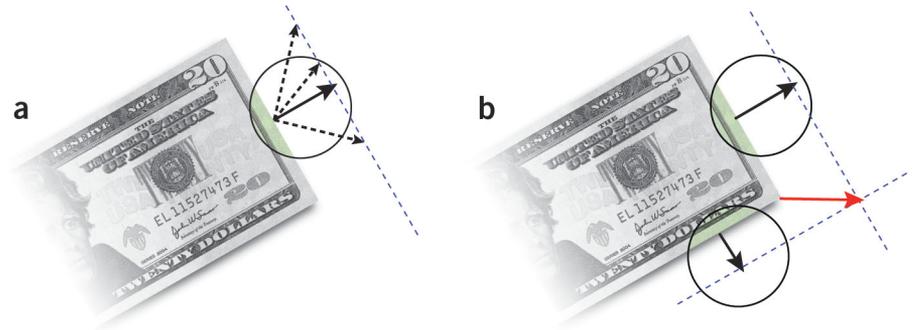
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**Multiple local motions must be combined to determine the direction of object motion, which is harder than it seems. A new paper proposes an elegant and simple solution to this problem, eminently realizable in feed-forward circuits.**

Physicists have long regarded simpler models as more valuable, no matter how complex the problem. Neuroscientists have not always embraced this notion, perhaps because of the confusing plethora of detail that the biology of the brain offers. The visual neuroscience of pattern motion processing is typical of tractable vision problems—there is a welter of experimental detail, along with a variety of models. However, models that work well to explain perceptual phenomena are often difficult to instantiate in ‘wetware.’ For these reasons, a simple model that explains such a complex perceptual problem in neuronally realistic terms provides considerable cause for rejoicing.

The model of Rust *et al.* in this issue<sup>1</sup> combines two simple mechanisms to produce a very good account of how neurons in the middle temporal extrastriate visual cortex (MT, or equivalently V5) might acquire their selectivity for pattern direction. This well-studied, mid-level area is highly specialized for the analysis of motion<sup>2,3</sup>. It receives direct inputs from primary visual cortex (V1) and in turn projects to premotor structures. One of the most provocative findings from MT is that neurons integrate multiple directions of motion to unambiguously signal the motion of patterns—they can solve the so-called ‘aperture problem’<sup>4</sup>.

The aperture problem refers to the observation that an object’s direction of motion can be hard to track when information is available from a limited area of space. Neurons in V1 have very small receptive fields, and thus can only sample a small patch of the moving object (Fig. 1a). This means that each V1 neuron can only capture the component of motion that is perpendicular to the moving edge (black arrow), which may not be the same as the actual moving direction of the object. A whole family of possible object motions will cause the same response in this V1 neuron. In other words, the motion of the moving object appears to be ambiguous to any single V1 neuron. To solve this problem, the visual system



**Figure 1** The aperture problem in an election year. (a) The circle represents the receptive field of a single V1 neuron, and the solid black arrow represents the observed motion component orthogonal to the moving contour. Dashed black arrows indicate different possible object motions, all of which contain the same orthogonal motion component. (b) Once two or more V1 cells are positioned along differently oriented contours of the object, one can reconstruct the actual object motion. In this case, each edge is consistent with a different family of possible motions, but there is only one motion (red arrow) consistent with both observations.

needs to integrate these multiple V1 responses representing the motion of non-parallel moving contours. We can demonstrate this by using two V1 neurons (Fig. 1b). There is only one possible motion (red arrow), which indicates the actual object motion and contains the component motions observed by the two V1 cells.

MT in primate visual cortex has abundant opportunity to integrate inputs from directionally selective cells in V1 (ref. 5). MT cells’ receptive fields are about 10 times bigger than those in V1, often encompassing multiple object contours<sup>6</sup>. The original observation, which has long begged for mechanistic explanation, is that some neurons in MT clearly combine multiple motions to represent the unique pattern direction. Other neurons (‘component selective’) in MT behave much like expanded V1 cells, and responded to single contours but not the pattern as a whole. Many others responded in intermediate ways<sup>4</sup>. The paper by Rust *et al.* not only explains the emergence of pattern responses, but also compactly captures circuit features that contribute to this diversity of response types in MT.

The new model of Rust and colleagues<sup>1</sup> (Fig. 2) contains two stages, a V1 stage and an MT stage. The V1 stage is designed to resemble actual V1 data and contains a feature that turns out to be critical for the model’s success: two kinds of divisive inhibition. Such inhibition has been well documented in V1, though

the details are still being worked out<sup>7,8</sup>. The two types can be thought of as those within single columns in V1 and those between columns. The V1 population then projects to an MT neuron, which weights and adds the inputs, then passes the resulting signal through a nonlinearity that represents the spike generation. This kind of linear-nonlinear (‘LN’) model has been very successful at capturing integration at many stages of the visual system<sup>9,10</sup>. The model was individually adjusted for each cell—necessary for explaining neuronal variability—using a clever reverse-correlation method. Although it seems complex, the actual number of free parameters adjusted was relatively modest. Once developed, the model was tested against actual patterned stimuli of the sort commonly used in these sorts of experiments. The model passed this test convincingly, capturing the responses of the neurons with high fidelity.

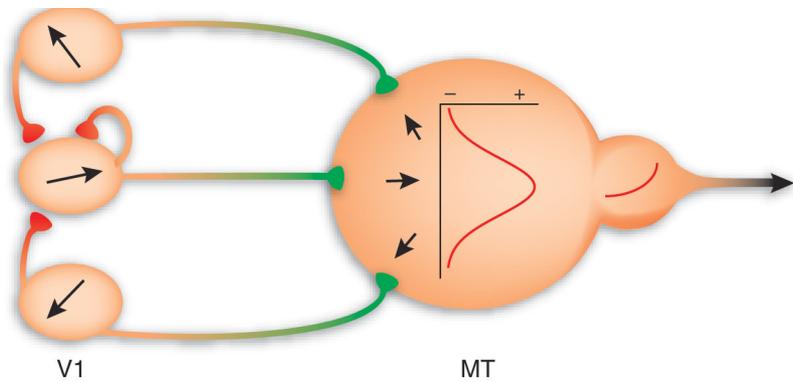
One might reasonably ask at this point where the added value of another clever model lies, especially given that this particular phenomenon has already been successfully explained by other models, some also simple and elegant<sup>11–13</sup>. Models have two chief goals: the consolidation of our ideas (descriptive value) and the generation of useful new experiments (predictive value). Simple, realistic models such as this one win by both metrics. The LN architecture at the heart of the Rust

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*et al.* model<sup>1</sup> is an idea of considerable power. Using it successfully against a long-standing computational problem is further evidence of its generality; this is a significant consolidation. The success of this model in this instance will no doubt drive others to test this architecture against their favorite problems, and many more complex problems await.

The model also makes a number of specific predictions about the circuitry between cortical areas. In particular, it proposes particular afferent architecture to bestow either simple (component) or more complex (pattern) responses on MT cells. These predictions are, unfortunately, rather difficult to test at present, as the tests will require paired recordings between MT neurons and their afferents. Such experiments, though possible with current technology, are arduous, difficult and slow. Some of the simplifying predictions will no doubt prove wrong, as the authors freely admit. MT receives afferents from many sources other than V1, and indeed these are likely to convey different kinds of information. Another simple prediction of the model is almost certainly wrong on the basis of current evidence; we are pretty sure that divisive inhibition operates not only in V1, but also in MT itself<sup>4</sup>. So, bringing a circuit reality to the nonlinearities at the MT level is a target for the next generation of experiments.

The lesson from physics is that models turning out to be wrong is all part of the game and should be viewed with approval. We get to better understanding by climbing a ladder built of the bones of dead models. If there is a core truth, some useful generality achieved by any



**Figure 2** Structure of the Rust *et al.* model. Each black arrow indicates the preferred direction of a V1 neuron in a (hypothetical) population of afferent neurons. Each cell's response is reduced by both tuned (recurrent) and untuned (lateral) inhibition from V1 neurons. Additionally, the tuning bandwidth of each V1 neuron is a free parameter. The MT cell integrates responses from V1 population inputs by calculating a weighted sum; inputs can be either excitatory or inhibitory. The weighted sum then passes through a nonlinear function to be transformed to firing rate.

generation of model, this is major progress. There has long been a desire to find general mechanisms of information processing that will apply across cortical areas<sup>15</sup>, and this paper marks a notable step in that direction. It is starting to look as if the LN family of models might be such a unifying framework.

1. Rust, N.C., Mante, V., Simoncelli, E.P. & Movshon, J.A. *Nat. Neurosci.* **9**, 1421–1431 (2006).
2. Born, R.T. & Bradley, D.C. *Annu. Rev. Neurosci.* **28**, 157–189 (2005).
3. Britten, K.H. in *The Visual Neurosciences* (eds Chalupa, L.M. & Werner, J.S.) 1203–1216 (MIT Press, Cambridge, Massachusetts 2004).
4. Movshon, J.A., Adelson, E.H., Gizzi, M.S. & Newsome, W.T. in *Study Group on Pattern Recognition Mechanisms* (eds Chagas, C., Gattass, R. & Gross, C.) 117–151 (Pontificia Academia Scientiarum, Vatican

- City, 1985).
5. Movshon, J.A. & Newsome, W.T. *J. Neurosci.* **16**, 7733–7741 (1996).
6. Maunsell, J.H.R. & Van Essen, D.C. *J. Neurophysiol.* **49**, 1127–1147 (1983).
7. Heeger, D.J. *Vis. Neurosci.* **9**, 181–197 (1992).
8. Ferster, D. & Miller, K.D. *Annu. Rev. Neurosci.* **23**, 441–471 (2000).
9. Chichilnisky, E.J. *Network* **12**, 199–213 (2001).
10. David, S.V., Hayden, B.Y. & Gallant, J. *J. Neurophysiol.* advance online publication, 20 September 2006 (doi: 10.1152/jn.00575.2006).
11. Simoncelli, E.P. & Heeger, D.J. *Vision Res.* **38**, 743–761 (1998).
12. Wilson, H.R. & Ferrera, V.P. *Vis. Neurosci.* **9**, 79–97 (1992).
13. Grossberg, S. & Mingolla, E. *Percept. Psychophys.* **53**, 243–278 (1993).
14. Britten, K.H. & Heuer, H.W. *J. Neurosci.* **19**, 5074–5084 (1999).
15. Douglas, R.J. & Martin, K.A. *J. Physiol. (Lond.)* **440**, 735–769 (1991).

## New clues for axonal repair in ALS

In amyotrophic lateral sclerosis (ALS, also called Lou Gehrig's disease), corticospinal neurons progressively degenerate, causing the loss of motor function and eventual paralysis seen in these individuals. Damage to these neurons also contributes to the loss of motor function in spinal cord injury. However, little is known about the mechanisms that regulate the survival and differentiation of corticospinal neurons. A paper on page 1371 by Hande Özdinler and Jeffrey Macklis describes new techniques to purify and culture these motor neurons, allowing the authors to dissect the mechanisms by which the morphology of these neurons is regulated.

The authors retrogradely labeled corticospinal neurons with fluorescent microspheres and used fluorescence-activated cell sorting to obtain homogeneous populations. These cultured neurons maintained the morphological and molecular phenotypes of developing corticospinal neurons *in vivo*. Insulin-like growth factor (IGF-1) specifically enhanced axonal outgrowth in these neurons, an effect mediated by the IGF-1 receptor and the PI3K and MAPK signaling pathways. Brain-derived neurotrophic factor, in contrast, induced dendritic branching and outgrowth, but did not affect axonal outgrowth. *In vivo* blockade of the IGF-1 receptor caused axonal outgrowth defects in the corticospinal tract. Critically, this effect of IGF-1 was independent of its effect on survival of these neurons, as corticospinal neurons isolated from mice lacking the apoptosis protein *Bax* behaved similarly to wild-type neurons in response to locally applied IGF-1. By demonstrating that IGF-1 is a potent enhancer of axonal outgrowth in corticospinal neurons, these results may help guide future efforts to use IGF-1 to enhance the outgrowth and functional connectivity of damaged neurons in diseases such as ALS and primary lateral sclerosis.

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