multiple senses in parallel not only to detect the thing's presence but also to provide an appropriate reference frame to establish its location. For example, the location of an object that touches the skin of a hand can only be worked out if the positions of the corresponding wrist, elbow, shoulder and rest of the body are also known.

One other key concept explored in this book concerns the different ways space may be represented in assemblies of neurons and how this representation can vary according to the sensory system involved. For instance, we are shown how the location of a thing that is both noisy and visible is coded in radically different ways by the auditory and visual systems, so requiring very different types of brain processing to extract the spatial information. This leads Groh to ask — how are these different representations of the same spatial attribute combined by the brain? How is this language barrier bridged? The book does not really answer this question, which is fair enough given that it is a mystery that has yet to be solved. However, Groh does reveal that the brain sometimes makes mistakes when combining spatial information from different senses. These errors give rise to illusions, which in the case of sight and sound are routinely exploited by ventriloquists. But, as this book helps us realise, errors and illusions are also keenly exploited by neuroscientists, as they provide important and unique clues about how the brain works.

Be aware that this book does not offer a comprehensive deconstruction of the brain and its senses, but that is not its intention. Rather, it is a succinct attempt to convey a flavour of some of the issues surrounding the brain's analysis of a three-dimensional world. To do this, Jennifer Groh has knitted together very selective strands of knowledge acquired from many sources, including the 2014 Nobel prize-winning work on spatial cognition. In sum, it is a book written with the authority of an expert that can be understood and enjoyed by almost any curious person.

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Quick guide

Elementary motion detectors

Mark Frye

What is an elementary motion detector? An elementary motion detector (EMD) is a theoretical model devised to explain the minimal computations required to perceive movement from the activity of photoreceptors. An eye maps an image of the world onto a sheet of photoreceptors. Any single receptor has a narrow field of view, and responds to fluctuations in illumination over time within its narrow field, but cannot provide unambiguous information about the direction of movement of an image. For example, a receptor response might result from a bright spot in the visual scene moving into its field of view from above, below, left, or right. The direction of motion can only be detected by comparing the activity of at least two receptors. The EMD is one of several models that predict the minimal interactions between two photoreceptors required to detect directional movement of the visual scene from the pattern of activation at each.

What are the components of an EMD? The components of the EMD are roughly similar to any model for motion detection, grounded in the physical principles of movement. In its most basic form, the EMD model is composed of two spatially separated input channels such as photoreceptors, a time delay, and a nonlinear interaction such as multiplication. The *spatial separation* is important because a bright spot within the moving scene would stimulate the first input, followed by the second input, a comparison that provides a correlation in space so that any point in the scene activates the two receptors only if it is in motion. A *time delay* ensures that the signal arriving at the first input is correlated in time with the one arriving at the second input when the scene is moving. Delaying one input provides the added advantage that any correlation between the inputs occurs for image motion in only one direction — from the delayed toward the un-delayed side. For this reason, the EMD is often referred to as a 'delay and correlate' model. Finally, the two signals are *multiplied* to boost tightly correlated activity (Figure 1).

How was the model derived and explored? Bernhard Hassenstein and Werner Reichardt developed the model in the 1950s. They referred to it as a correlation model, and it has become commonly referred to as the Reichardt detector, the Hassenstein-Reichardt EMD, or simply the EMD. The model is simple and elegant, and its key operations are intuitive (Figure 1). However, the internal components, such as the spatial separation of the inputs, the temporal delay and multiplication, each constrain the performance of the EMD in ways that allow direct comparison of the model to the performance of neurons, neural circuits, or whole animal behavior. To explore these predictions, Hassenstein and Reichardt took a psychophysical approach in which they reasoned that visual reflexes are robust in any animal with sophisticated visual behavior. Motivated by pioneering work at the time on the visual behavior of other insects, they glued a beetle by its back to a stiff wire and suspended it within a large rotating visually textured drum. The tethered beetle clasped a lightweight ball that it could 'roll' with its legs, apparently fooled into thinking it was walking on the ground. By rotating the drum around the tethered beetle, and observing its ball rolling reactions, they were able to directly compare the behavioral responses to the predictions made by the model.

What evidence suggests that animal vision is based on the EMD? The model makes specific functional predictions that distinguish it from other theoretical models of motion vision. Consider a simplified visual scene made up of evenly spaced stripes. The EMD encodes the rate of stripes passing over the stationary input arms rather than the true velocity of the moving pattern — in other words, the model cannot distinguish a pattern of thin stripes moving slowly from a pattern of thick stripes moving quickly. A visual neuron or behaving animal that shows similar

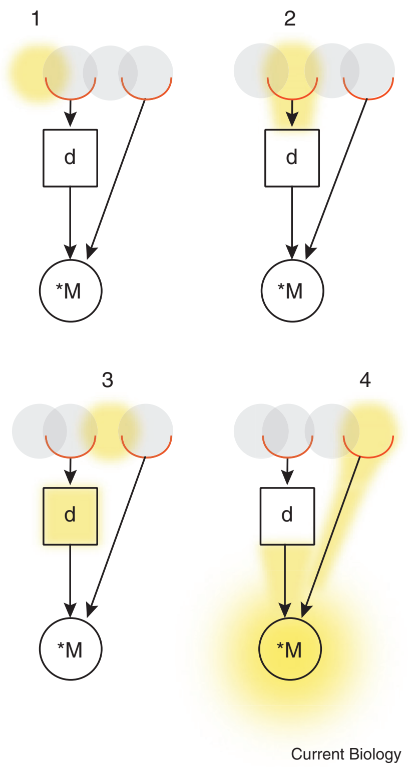

Figure 1. Schematic of the elementary motion detector.
The key components of an elementary motion detector include two inputs (red), a time delay on one input (d), and multiplication on correlated signals (M). To imagine how it operates, consider four snapshots in time. 1: A spot of light from a visual scene moves from left-to-right across the retina. 2: The spot activates the first receptor. 3: The signal from the first receptor is delayed (d) as the spot moves between the two receptors. 4: The spot activates the second receptor, and both signals converge simultaneously onto a multiplication stage, producing a strong directionally selective motion signal. Light passing in the other direction would produce consecutive signals at the multiplication stage and null response from the detector.

dependence on the rate or frequency of stripes rather than the velocity of the striped pattern may be using an EMD-based motion vision system. This is a profound implication — that a biological motion detection system cannot detect true velocity, the corollary of which is that the animal carrying an EMD cannot tell how fast it is moving through a stationary scene! Nevertheless, this peculiarity has been revealed numerous times within single neurons and behaving animals alike. Now, although the 'stripe rate versus stripe speed' dichotomy is useful for experimental verification, it is worth mentioning that natural scenes do not abide by the mathematical and experimental convenience of periodically striped patterns; hence, recent evidence suggests that the behavior of an EMD is more robust under naturalistic stimulus conditions.

Are there additional complexities to the EMD? The EMD I have depicted here is selective for motion from left-to-right, and any response to motion from right-to-left is absent or 'null' (Figure 1). Some motion sensing neurons, however, show opponency, whereby the response to motion in one direction is positive or excitatory, and the response to the motion in the opposite direction is negative or inhibitory, rather than null. Consider a second EMD that is mirror-symmetrical to the one depicted here, thus selective for right-to-left motion. By adding a step that subtracts the outputs from both, the combined detector would produce a positive or 'preferred' output for motion to the right and negative or 'anti-preferred' output for motion to the left.

An additional elaboration of the model is based on the notion that moving images produce both increments and decrements in luminance generated by the movement of bright and dark regions of the image, respectively. Modeling and experimental work has supported the existence of two classes of EMD. One detector is excited by a light ON response at each input, and the other is excited by light OFF at each input. Finally, the contrast of visual scenes varies widely and quickly for an animal moving between, for example, the bright sun of an open field and deep shade of a forest canopy. Various filtering schemes have been proposed to operate on the input arms of the EMD in order to make the model robust to diverse static environmental visual conditions. However, some of the elaborations to the EMD that enable the model to operate under more demanding visual conditions render it less effective at the basic processes for which it is best known. There is much yet to learn and indeed the experimental tools and techniques have now come of age and are flipping the paradigm to inform motion vision theory rather than the other way around.

Is the EMD implemented with real neurons? The ultimate motivation for any model of motion vision — and there are several — is that it be matched to the connectivity and activity of underlying neural circuitry. This is where the EMD model has become distinctively satisfying. Behavioral studies pioneered by Karl Götz, coupled with neuroanatomical examinations by Karl-Friedrich Fischbach, established the fruit fly *Drosophila* as a powerful model for studying the general algorithms and putative anatomical circuitry of EMD-based motion vision. The unparalleled intellectual investment in *Drosophila* genetics and molecular biology over the past few decades has yielded uniquely powerful methods for studying the components of the EMD with single neuron resolution. Thanks in large part to tools for genetically encoded markers, activating and silencing reagents, as well as opto- and electro-physiology recordings from pre-specified classes of neurons, optic lobe interneurons have been identified that implement key components of the EMD, including: the separate pathways for carrying ON and OFF signals from each retinal photoreceptor into the optic lobe; the temporal delay filter; the layered cells that provide motion selectivity in four cardinal directions. In response to motion stimuli, each of these identified cellular pathways obeys the EMD model. Other putative cellular participants have been inferred by electron microscopic anatomical analysis and await experimental interrogation.

What does the future hold? There are still many open questions about the EMD itself and how it interacts with visual perception generally. What is the cellular implementation of the non-linear multiplication stage? Where is the spatial comparison made between neighboring inputs? Is the EMD implemented by similar cells in different insects or with analogous circuitry in other taxa? A modified correlator model has predicted a different set of computations for an especially challenging form of visual perception, small object detection: which neurons make these special computations, and how do they interact with the EMD? More broadly, how is it that flies, which apparently rely on the EMD for motion vision, nevertheless perceive special visual objects that fail to arouse output from the classical form of the model? Is EMD output modulated by behavioral state or by corollary discharge, such as might occur during voluntary changes in gaze? In flies, the itemized network of neurons and synaptic connections for EMDs and those regions devoted to decoding and integrating EMD output comprise only a fraction of the visual circuitry of the optic lobes identified to date. What functions do the vast majority of visual processes provide? And how do these processes interact with the signals for self-motion generated by the EMD?

Finally, are there other elementary detector schemes for different sensory modalities? The powerful combination of *Drosophila* neurogenetics and molecular biology, coupled with rapidly evolving technologies for tracking and manipulating complex visual behavior, is providing an exceptionally clear view on the cellular, cell circuit, and behavioral levels of organization for the elementary motion detector and beyond.

Where can I find out more?

Behnia, R., Clark D.A., Carter, A.G., Clandinin, T.R., and Desplan, C. (2014). Processing properties of ON and OFF pathways for Drosophila motion detection. Nature *512,* 427–430.
Borst, A., and Euler, T. (2011). Seeing things in motion: Models, circuits, and mechanisms. Neuron *71,* 974–994.
Buchner, E. (1976). Elementary movement detectors in an insect visual system. Biol. Cybern. *24,* 85–101.
Clark, D.A., Burstzyn, L., Horowitz, M.A., Schnitzer, M.J., and Clandinin, T.L. (2011). Defining the computational structure of the motion detector in *Drosophila*. Neuron *70,* 1165–1177.
Hassenstein, V., and Reichardt, W. (1956). System theoretical analysis of time, sequence and sign analysis of the motion perception of the snout-beetle Chlorophanus. Z Naturforsch. B, *11*, 513–524.
Maisak, M.S., Haag, J., Ammer, G., Serbe, E., Meier, M., Leonhardt, A., Schilling, T., Bahl, A., Rubin, G.M., Nern, A., *et al*. (2013). A directional tuning map of Drosophila elementary motion detectors. Nature *500,* 212–216.
Takamura, S., Bharioke, A., Lu, Z., Nern, A., Vitaladevuni, S., Rivlin, P.K., Katz, W.T., Olbris, D.J., Plaza, S.M., Winston P., *et al*. (2013). A vision motion detection circuit suggested by Drosophila connectomics. Nature *500,* 175–181.
Wiederman, S.D., Shoemaker, P.A., and O'Carroll, D.C. (2013). Correlation between OFF and ON channels underlies dark target selectivity in an insect visual system. J. Neurosci. *33,* 13225–13232.

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White-Nose Syndrome fungus introduced from Europe to North America

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The investigation of factors underlying the emergence of fungal diseases in wildlife has gained significance as a consequence of drastic declines in amphibians, where the fungus *Batrachochytrium dendrobatidis* has caused the greatest disease-driven loss of biodiversity ever documented [1]. Identification of the causative agent and its origin (native *versus* introduced) is a crucial step in understanding and controlling a disease [2]. Whereas genetic studies on the origin of *B. dendrobatidis* have illuminated the mechanisms behind the global emergence of amphibian chytridiomycosis [3], the origin of another recently-emerged fungal disease, White-Nose Syndrome (WNS) and its causative agent, *Pseudogymnoascus destructans*, remains unresolved [2,4]. WNS is decimating multiple North American bat species with an estimated death toll reaching 5–6 million. Here, we present the first informative molecular comparison between isolates from North America and Europe and provide strong evidence for the long-term presence of the fungus in Europe and a recent introduction into North America. Our results further demonstrate great genetic similarity between the North American and some European fungal populations, indicating the likely source population for this introduction from Europe.

Diversity among genetic markers is a powerful tool to reconstruct colonisation events and exchanges between populations [5]. Populations in recently colonised areas harbour genetic signatures distinct from long established ones (for example, [5]). We therefore used genetic data to test if *P. destructans* is long established in Europe (that is, native) and assess whether it is a likely source population for the recent introduction to North America. Twenty-eight *P. destructans* isolates, collected from *Myotis* bats over a five-year timeframe and covering regions in Europe with the highest number of reported cases of *P. destructans* infection [6] (Figure 1A), were sequenced at eight genomic loci and combined with published data from seventy-one North American isolates covering a similar range and timeframe [4,7] (see Supplemental Experimental Procedures).

Seven of the eight genes sequenced were polymorphic among the European isolates (Tables S1 and S2), sharply contrasting with the absence of variation observed across the North American isolates [4,7]. These data demonstrate the older origin of the European population of *P. destructans* compared with that of the North American population. The number of isolates sequenced was larger in North America (n = 71) than Europe (n = 28), likely leading to an under-estimate of the number of haplotypes present in Europe. Photographic evidence has suggested the presence of *P. destructans* in Europe for decades without any associated mass mortality, consistent with an endemic European distribution and host–pathogen co-evolution [2,6,8], although such data did not inform on the presence of the fungus in Europe over longer timeframes.

Combining the gene fragments for each isolate allowed the detection of eight haplotypes across Europe, and the most common (Hap_1) was shared with all North American isolates (Figure 1). Hap_1 was found in Western but not Eastern Europe (Figure 1A). Phylogenetic reconstruction identified samples from France, Germany and Belgium as the most basal (Figure 1B). The absence of genetic variability at these eight loci in North American isolates suggested either novel appearance in the area [4,7] or recent emergence of a virulent strain of a previously benign fungus not necessarily present on bats [2]. The fact that the most common European haplotype is 100% identical at the sampled loci to the clonal haplotype from North America corroborates a recent inter-continental fungal transfer from Europe to North America [6], rather than the emergence of a virulent strain