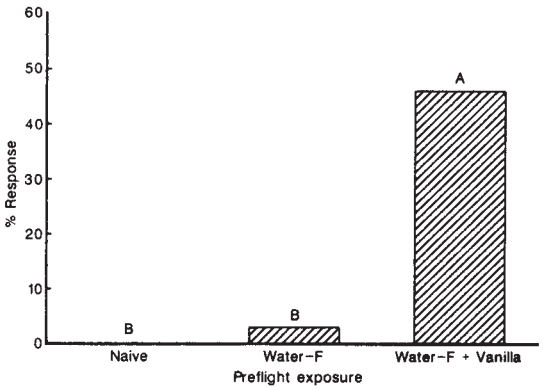

Fig. 4 Flight response of *M. croceipes* females to a source of vanilla extract (Nielsen Massey) in a wind tunnel after no experience (naive), or contact with water-F, or water-F plus vanilla extract. Bars capped by different letters are significantly different, $P < 0.01$, Waller-Duncan K-Ratio t-test, minimum significant difference = 12.4%, $n = 90$ wasps (three replications, three treatments, ten wasps per replication per treatment).
Methods. This experiment was conducted in the same way as previous experiments (Figs 2 and 3). Pure vanilla extract (80 μl) was pipetted onto a piece of Whatman number 1 filter paper and used as the attractant source as described before. Materials used for preflight conditioning of the females were prepared as before. When water-F and vanilla were combined, 28 μl of water-F was first placed on the filter paper and then 1 μl of vanilla extract was added to the centre of the moist spot.

extracts of either *H. zea* or *T. ni* faeces.

Our findings show that the host-seeking behaviour of *M. croceipes* females is mediated by both a nonvolatile, contact chemical and by volatile chemical cues. The nonvolatile substance is water extractable and distinct from the hexane soluble volatile cues that attract females. The nonvolatile compound is also host specific. The volatile attractants may be plant-derived. The role of the relatively nonvolatile, hexane soluble kairomone, 13-methylhentriacontane, previously identified for this parasitoid[8], is not known. It may facilitate the association process described here by enhancing the antennal rubbing behaviour.

M. croceipes parasitizes *Heliothis* species, which are highly polyphagous. Thus the ability to locate its hosts among other plant feeders in diverse systems of vegetation is crucial. Learned responses to varying trail odours through association with a host recognition kairomone in the host faeces may give *M. croceipes* females an adaptive advantage. Associative learning in connection with host-detection by parasitoid and plant feeding insects has been shown by a number of authors[2-5,9-12]. Contact with the target organism (or a model) and subsequent oviposition, however, generally was necessary. We believe this to be the first demonstration of associative learning in insects where encounter with the target organism is not involved. This phenomenon provides a valuable way of finding hosts or prey species and may be widespread in parasitic and predatory systems as the target organisms (but not their by-products) often elude the searching parasitoid or predator.

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Perception of three-dimensional structure from motion in monkey and man

R. M. Siegel* & R. A. Andersen†

The Salk Institute for Biological Studies, La Jolla, California 92139, USA

Information on motion is important for the determination of the three-dimensional (3-D) structure of the environment for both human and non-human primates[1-8]. For example, if a person were to close one eye and look at an evenly illuminated, irregularly shaped object, he would be unlikely to guess its shape correctly. But if the object is moved about, the correct shape immediately becomes apparent[9]. Little is known about how the primate visual system actually does this, although various theories have been proposed[7,8,10-15]. We have developed novel, highly controlled motion stimuli to use with psychophysical and physiological techniques to study how 3-D structure is obtained from motion. We show that the Rhesus monkey can detect 3-D structure from motion in the same way as human subjects. Furthermore, the dependence of both species on certain parameters of the display shows that information is integrated both spatially and temporally for this higher visual function.

We have defined the ability to extract structure from motion in terms of a reaction-time task. The subject is told, or conditioned, to pull on a lever at the onset of the stimulus and to try to release it within 1 s after the stimulus changes at a randomly selected time[16]. In these experiments, the stimulus changes from a control with no structure to a test with 3-D structure.

The control unstructured and the test 3-D structured stimuli are computed from the parallel projection of points covering the surface of a cylinder (Fig. 1). For the structured stimulus the cylinder revolves at a fixed angular velocity of 35 degrees s^{-1}. The horizontal velocity of the projected points varies as a sinusoidal function of their horizontal location on the screen. Points at the centre move fastest whereas those at the edges move slowest. The unstructured stimulus is generated by computing the velocity trajectories of the points as described above and displacing each trajectory using a randomly selected distance chosen from a uniform distribution equal to the width of the display. This allows conservation of the velocity distribution while destroying the spatial relationship of the points with respect to each other.

Each point is displayed for a pre-selected duration defined as its lifetime[7,8,17]. At the end of the point's lifetime it disappears and randomly reappears at a new location on the screen to begin a new motion trajectory. The beginning of the lifetimes are staggered so that the points asynchronously flicker on and off. This effect can be imagined by visualizing the reflections of sunlight on a waterfall. Any possible form cues are constantly changing. The point density across the screen is also kept constant leaving only the change in the structure of the display as a cue for the release of the key. The change from unstructured motion to structured motion for each point occurs during the relocation of that point. The total time to switch from the unstructured to the structured display is equal to the point lifetime.

The three human and three monkey subjects tested were able to detect the change from completely unstructured motion to a completely structured motion at the 90-100% correct level (the number of points was 128; point lifetime was 532 ms). To quantify this, psychometric functions were obtained by plotting the

* Present address: Laboratory of Neurobiology, The Rockefeller University, 1230 York Avenue, New York, New York 10021, USA.
† To whom correspondence should be addressed at Department of Brain and Cognitive Sciences, E25-236 Massachusetts Institute of Technology, 77 Massachusetts Avenue, Boston, Massachusetts 02139, USA.

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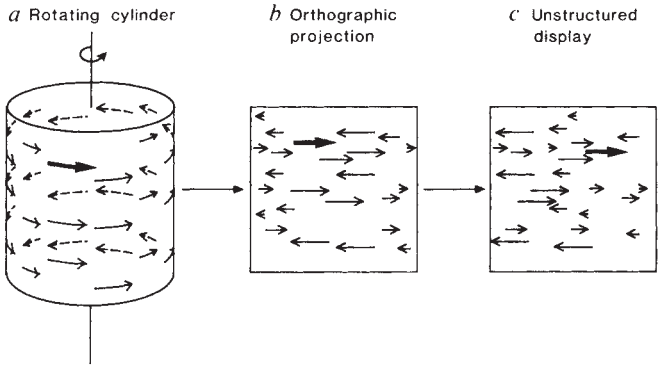

Fig. 1 Generation of the motion displays. The velocity of each point is indicated by the length of the arrows. *a*, The stimuli are generated by first computing the location of points on the surface of the rotating cylinder for each instant in time. *b*, The points are then parallel (orthographically) projected onto a plane perpendicular to the observer's line of sight. This is the 'structured display'. *c*, The 'unstructured display' is computed by taking each point's motion trajectories from the test display and displacing them randomly in a window equal to the width of the display. The fate of an individual motion trajectory for a point crossing the front of the display is shown by the bold arrow. The point density on the surface of the display is kept constant; each point is displayed for a finite amount of time. At the end of this time, the point vanishes and reappears randomly on the screen to follow a new motion trajectory.

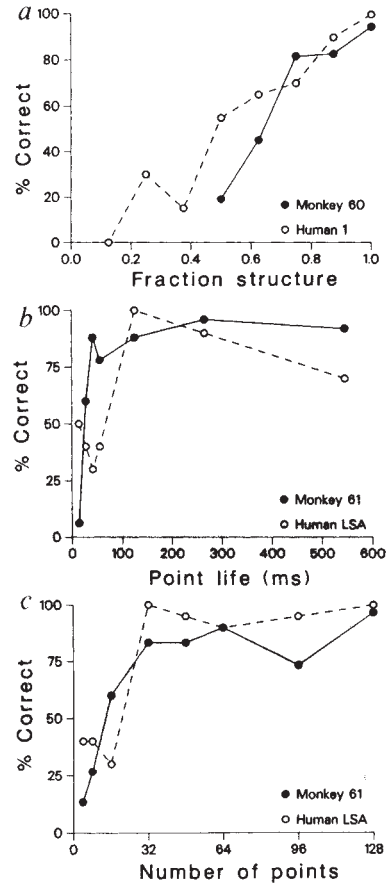

Fig. 2 A human subject's and Rhesus monkey's performance as a function of the parameters of the display. *a*, The percentage of trials where the subject released the key within the requisite time window is plotted as a function of the fraction of structure. The two primates have similar psychometric functions that have a decrease in detection when the fraction of structure is less than ~0.65. There was no different between the human and monkey subjects (χ^2 for independent samples, $P < 0.05$). In this experiment, 128 points were viewed with a point lifetime of 532 ms; the cylinder revolved at 35 degrees s^{-1}; the display was refreshed at 35 Hz. The ability of the monkey and human subjects to perform the task depended on both the point lifetime (*b*) and number of points (*c*) of the display. The significant difference in minimal point lifetime required to successfully perform the task between the human and primate subject is probably due to the additional training given the monkey subjects. It can be seen that decreasing the number of points makes it more difficult for the subjects to perform the task. In *b* and *c*, the fraction of structure was 0.875; the refresh rate was 70 hz; the cylinder revolved at 35 degrees s^{-1}.

percentage of trials where the change was detected within the reaction-time window as a function of the structure of the test display (Fig. 2*a*). The structure of the test display was reduced by increasing the range over which the points were randomly displaced. (The fraction structure of the display is defined as one minus the ratio of the range over which the trajectory displacement can occur divided by the width of the display.) Both species have similar psychometric functions for the dependence on the fraction of structure in the display (Fig. 2*a*). These experiments therefore provide evidence that non-human primates can detect the change from unstructured to structured motion and that this ability is similar to that of humans.

A control experiment was performed to ensure that the subjects were not using local analysis of the motion-flow field to perform this task. When the display was masked so that only the central 1/25th of its area was visible, the three human subjects and two monkey subjects tested were unable to detect the change from no structure to complete three-dimensional structure ($P < 0.01$, Kolomogrov-Smirnov test for each of the five subjects). This result shows that local artifactual cues such as a change in the distributions of speeds were insufficient to perform the task. The subjects were probably not able to detect structure from motion in the smaller areas of the surface because too few points were visible to perform the computation.

Computational and psychophysical studies have suggested that the point lifetime[7,8] and number of points[5-8,14,15] in the display are important constraints for abstracting structure from motion. It was found that the subjects' performance of the task declined for shorter point lifetimes (Fig. 2*b*). When 128 points were viewed and the lifetime was shorter than 75 ms and 100 ms for monkeys and humans respectively, they were unable to do the 3-D task. It was found that both species' performance was reduced by ~40–70% when less than 32 points were viewed.

Our results show in two primate species that more than four times the theoretical minimum number of points (based on geometric considerations of the structure-from-motion theorem[14]) were necessary to abstract structure from motion in the primate visual system with the flickering dot display. The observation that the minimum point life could be as short as 100 ms has important implications for selecting algorithms to model the structure-from-motion process. The average reaction time for detecting the shift from no structure to structure was 600–1000 msec depending on the individual. Thus algorithms[15] that require the positions of points continuously during the entire computation period would fail under these conditions. Rather, in our experiments, representations of entire surfaces appear to be computed from the flow field; points can exist momentarily anywhere on the surface and still be used for the computation.

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Brain-derived neurotrophic factor prevents neuronal death *in vivo*

M. M. Hofer & Y.-A. Barde

Department of Neurochemistry, Max-Planck-Institute for Psychiatry, 8033 Martinsried, FRG

Developing vertebrate neurons are thought to depend for their survival on specific neurotrophic proteins present in their target fields[1]. The limited availability of these proteins does not allow the survival of all neurons initially innervating a target, resulting in the widely observed phenomenon of naturally occurring neuronal death[1]. Although a variety of proteins have been reported to promote the survival of neurons in tissue culture[2–4], the demonstration that these proteins increase neuronal numbers and/or decrease neuronal death *in vivo* has only been possible with nerve growth factor (NGF)[5–7]. The generalization of the concept that neurotrophic proteins regulate neuronal survival during normal development critically depends on the demonstration that the survival of neurons *in vivo* can be increased by the administration of a neurotrophic protein different from NGF. We report here that this is the case with brain-derived neurotrophic factor, a protein of extremely low abundance purified from the central nervous system.

Brain-derived neurotrophic factor (BDNF) is a small, basic protein[8] that supports the survival of various neurons *in vitro*, in particular of primary sensory neurons originating from the neural crest and ectodermal placodes[9]. Given the limited availability of BDNF, we decided to inject it into quail embryos: not only is the size of these embryos small when compared, for example, with those of the chick, but also their development is more rapid (16 versus 21 days[10]). We first counted the number of neurons at various embryonic stages in both the nodose ganglion of placodal origin[11] and a dorsal root ganglion of neural crest origin[11]. It was found that in both ganglia, many neurons are lost during development (Fig. 1): about 30% and 38% for the nodose and dorsal root ganglia respectively. Either NGF (10 μg) or BDNF (1 μg) were injected daily from embryonic day 3 until day 7. Embryos were killed at day 8 and the neurons counted (Table 1). Upon dissection, the only macroscopically visible effect was an increase in the size of the sympathetic chain in the NGF-treated embryos. No such effects were seen with BDNF. As expected from previous studies with the chick[6], it was found that repeated injections of NGF rescued many neurons in the dorsal root ganglion: the number of neurons was significantly higher at day 8 than in control, and corresponded roughly to the peak value found before the period of neuronal death. Similar results were obtained with BDNF, supporting an earlier suggestion, based on findings[12] *in vitro*, that some primary sensory neurons, corresponding in this case to the population normally eliminated, could survive either with an excess of (peripherally derived) NGF or (centrally derived) BDNF. To establish directly that exogenous BDNF prevents normally occurring cell death *in vivo* (a fact firmly established *in vitro*[8,9,12]), we also counted degenerating neurons (Table 2). We found that the total number of degenerating neurons could be significantly reduced by administering BDNF. In the nodose ganglion, as in the dorsal root ganglion, injections of BDNF resulted in an increased number of neurons at day 8 when compared with controls. Again, this number corresponds to the highest number of neurons found before cell death. No effects of NGF were seen on the nodose ganglion.

From the results that increased neuronal numbers are found in dorsal root and nodose ganglia after application of exogenous BDNF, we conclude that this protein, like NGF in peripheral target fields[13], is normally present in limiting amounts in the central target fields of the primary sensory neurons. It is known from studies in the chick that the presence of the central target, the neural tube, is essential for the development of the dorsal root ganglion: removal of the neural tube[14] or interposition of a membrane between the neural tube and the dorsal-root-ganglion anlage[15] both result in the death of neural-crest-derived

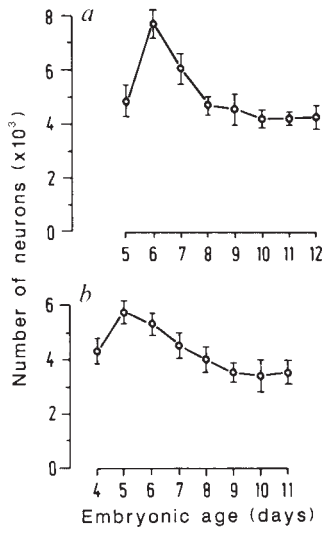

Fig. 1 Time course of naturally occurring cell death in *a*, quail dorsal root ganglion (DRG, 25th according to ref. 18) and *b*, nodose ganglion (NG). The values are means ± s.d. of 4-8 ganglia. No left and right differences were found for either ganglia.
Methods. Quail embryos were staged according to Zacchei[10], the ganglia fixed in 4% formaldehyde, embedded in paraffin, sectioned at 6 μm and stained with thionine. All neurons (clearly identified as such by their large size and intensive staining) containing at least one nucleolus were counted in alternative sections at a magnification of ×250. The number of neurons obtained was multiplied by 2 and no corrections were made: only 4% of the nuclei were found to contain more than one nucleolus and the size of one nucleolus (1–2 μm) is substantially smaller than the section thickness[7]. We directly checked that split nucleoli did not affect neuronal counts by comparing ganglia sectioned at either 6 or 10 μm: no significant differences in neuronal numbers were found[20].

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