

1. M. M. Haith and J. J. Campos. *Ann. Rev. Psychol.* 28, 251 (1977).
2. G. Fouts and P. Atlas, paper presented at the meeting of the American Psychological Association, New Orleans (August 1974); R. A. Spitz and K. W. Wolf, *Genet. Psychol. Monogr.* 34, 57 (1946); L. A. Sroufe, E. Waters, L. Matas, in *The Origins of Fear*, M. Lewis and L. Rosenblum, Eds. (Wiley, New York, 1974), p. 49.
3. K. S. Robson, *J. Child Psychol. Psychiatry Allied Discip.* 8, 13 (1967); ———, F. A. Pederson, H. A. Moss, *Child Dev.* 40, 619 (1969); P. Wolff, in *Determinants of Infant Behavior*, B. M. Foss, Ed. (Methuen, London, 1961), vol. 2, p. 113.
4. G. C. Carpenter, *Merrill Palmer Q.* 20, 181 (1974); D. Maurer and P. Salapatek, *Child Dev.* 47, 523 (1976); D. Stern, in *The Effect of the Infant on Its Caregiver*, M. Lewis and L. Rosenblum, Eds. (Wiley, New York, 1974), p. 187.
5. Maurer and Salapatek [see (4)] made judgments of infant fixations on real adult faces. Since they did not measure fixations or the regions of the adult faces, their determination of regional fixations must be regarded as estimates. They reported as we do here an increase in face and eye looking to a still face between 4 and 8 weeks of age. Donnee [see (13)] measured fixations of infants as they scanned a face photograph.
6. C. E. Lord and M. M. Haith, *Percept. Psychophys.* 16, 413 (1974).
7. The cooing and smiling of the older infants to the faces indicated that they found the situation to be relatively natural and realistic.
8. Mirror Y (Libby-Owens-Ford No. 956-1) transmitted most of the infrared light and reflected most of the visible light. Enough visible light was transmitted for video recording of the adult's face with a standard vidicon tube.
9. This camera was a Shibaden HV-14 fitted with a Tivicon 1602 infrared silicon diode tube and an  $f/2.0$ , 100-mm lens.
10. M. M. Haith, *Am. Psychol.* 24, 279 (1969).
11. P. Salapatek and W. Kessen, *J. Exp. Child Psychol.* 3, 155 (1966); W. Kessen, P. Salapatek, M. M. Haith, *ibid.* 13, 9 (1972); M. M. Haith, in *Handbook of Sensory Physiology*, vol. 8, *Perception*, R. Held, H. Leibowitz, H. L. Teuber, Eds. (Springer-Verlag, New York, in press).
12. R. Arnheim, *Art and Visual Perception* (Univ. of California Press, Berkeley, 1971).
13. L. Donnee, thesis, Harvard University (1972); M. J. Mendelson and M. M. Haith, *Monogr. Soc. Res. Child Dev.* 41 (No. 4) (1976).
14. Supported by grant MH23412 to M.M.H. We thank Scott Bradner, Bill Brown, Robert Lentz, and Leah Mann for their assistance in the research and Betty Richardson for typing the manuscript. A preliminary report of these findings was presented at the meeting of the Society for Research in Child Development, Minneapolis, April 1971.

9 August 1977

## Magnification in Striate Cortex and Retinal Ganglion Cell Layer of Owl Monkey: A Quantitative Comparison

**Abstract.** Magnification, the relative size of the neural representation of a portion of the visual field, decreases more rapidly with increasing visual field eccentricity in striate cortex than in the retinal ganglion cell layer of the owl monkey (*Aotus trivirgatus*); the proportion of the cells in striate cortex devoted to central vision is much larger than the comparable proportion of retinal ganglion cells. Magnification in striate cortex is a power function of magnification in the retinal ganglion cell layer. A formula for convergence (ganglion cells to cortical neurons) follows from this relationship.

Sensory surfaces project to mammalian neocortex in orderly topographic fashion. Sensory surfaces associated with behavioral specializations receive expanded representation, for example, the human and monkey hand, and the snout of the pig and coatimundi (1). Differential cortical representation may merely reflect differential innervation of the sensory surface or may be the consequence of an additional cortical adaptation.

In the mouse somatosensory system, cortical representation of the different whiskers may be described in terms of "peripheral scaling," that is, the number of cortical neurons per whisker is directly proportional to the peripheral innervation density (2). In the visual system, peripheral scaling describes the representation of the visual field (or retinal surface) in striate cortex of the cat (3) but not the rabbit (4). In primates, the central portion of the visual field receives a greatly expanded representation in striate cortex (5-7). Investigators disagree, however, as to whether this is solely because of increased ganglion cell density near the center of the retina (8)

or whether the cortex provides additional "magnification" (9). The present study demonstrates that in the owl monkey, *Aotus trivirgatus*, the representation of the center of the visual field is expanded much more than might be expected from the distribution of retinal ganglion cells. This strongly suggests that, in primates, striate cortex is even more specialized than the retina for central vision.

We defined magnification in a given portion of a neural structure as the proportion of the structure devoted to the representation of a particular visual field zone divided by the proportion of the visual field represented (7, 10):

$$M(\phi_1, \phi_2) = \frac{N(\phi_1, \phi_2) \div N_{\text{tot}}}{A(\phi_1, \phi_2) \div A_{\text{tot}}} \quad (1)$$

where  $M(\phi_1, \phi_2)$  is the magnification for the representation of the zone between two isoeccentricity contours of radii  $\phi_1$  and  $\phi_2$  with the center of gaze at the origin;  $N(\phi_1, \phi_2)$  is the number of cells within the representation of that zone in a given structure;  $N_{\text{tot}}$  is the number of cells in the structure; and  $A(\phi_1, \phi_2)$  and  $A_{\text{tot}}$  are the area of the zone and the total

area of the visual field, respectively. For structures where cell density is invariant with respect to eccentricity, volume measurements yield equivalent values for magnification, and where the thickness of the structure also does not change as a function of eccentricity, magnification may be calculated on the basis of surface area.

We calculated magnification in striate cortex of the owl monkey using a three-dimensional model of the brain constructed on the basis of serial sections and receptive field data from a previous electrophysiological mapping study (11). These results were compared to calculations of magnification for the ganglion cell layer of the owl monkey retina (12) based upon ganglion cell counts along both horizontal and vertical meridians made from whole mounts by Webb and Kaas (8). The owl monkey is an excellent subject for studying quantitative relations between representations of the visual field in different structures because: (i) the ganglion cell layer is thin enough to permit cell counts from whole mounted retinas; (ii) ganglion cells are not displaced about a fovea as they are in most other higher primates; (iii) the ratio of rods to cones does not change as a function of eccentricity (13), implying equivalent (normalized) magnification functions for scotopic and photopic vision; (iv) the topographic representations of the visual field have been determined for more structures of the owl monkey visual system than for any other primate (14, 15); and (v) its relatively smooth brain makes it possible to map the cortical visual areas more accurately in the owl monkey than in other species with more convoluted neocortices.

The expanded representation of the center of the visual field in owl monkey striate cortex cannot be attributed solely to peripheral scaling (16). While magnification decreases monotonically as a function of eccentricity in both retina and striate cortex, the decrease is considerably more gradual in the retina, and cortical magnification for the central 10 degrees greatly exceeds retinal magnification (Fig. 1); that is, the proportion of the cells in striate cortex devoted to central vision is much larger than the comparable proportion of retinal ganglion cells. Functionally, this suggests that, in primates, striate cortex is even more specialized than the retina for processing information concerning the center of the visual field. Anatomically, this means that the ratio of retinal ganglion cells to neurons in striate cortex increases with eccentricity. More specifically, the relation between magnification for corre-

sponding portions of striate cortex and the retinal ganglion cell layer is describable by a power function (Fig. 2):

$$M_C(\phi_1, \phi_2) = aM_R(\phi_1, \phi_2)^{2.35} \quad (2)$$

where  $M_C$  is the magnification for striate cortex,  $M_R$  is the magnification for the retinal ganglion cell layer, and  $a$  is the proportionality constant. Malpeli and Baker (9) have recently suggested that a similar relation exists between retinal ganglion cell density and magnification (cubic millimeters per steradian) in striate cortex of the rhesus monkey. They were forced, however, to compare retinal cell counts from rhesus monkey to results for striate cortex (7) which represented a composite of four species (baboon, rhesus, cynomolgus, and vervet) (17).

It follows from our definition of magnification and the observed relation between retinal and cortical magnification that the ratio of ganglion cells to striate neurons is a power-function of the area per ganglion cell (18):

$$\frac{N_R(\phi_1, \phi_2)}{N_C(\phi_1, \phi_2)} = k \left[ \frac{A(\phi_1, \phi_2)}{N_R(\phi_1, \phi_2)} \right]^{1.35} \quad (3)$$

where  $N_R$  and  $N_C$  are the numbers of retinal ganglion cells and striate cortical neurons, respectively, and  $k$  is the proportionality constant. A recent finding in the rhesus monkey (19) implies that although some primate retinal ganglion cells send collaterals to the superior colliculus, all ganglion cells send axons to the lateral geniculate nucleus (LGN). Nearly all the cells in the primate LGN project to striate cortex (20). Therefore, Eq. 3 describes anatomical convergence in the retino-geniculo-striate system, that is, the mapping of the retinal ganglion cell layer onto striate cortex. Convergence depends on eccentricity and thus on retinal area per cell. This may have implications for the study of the development of these connections. Experiments on the development of somatosensory and visual cortex suggest that portions of sensory surfaces compete for cortical representation (21). Our findings further suggest that in the developing primate retino-geniculo-striate system, competitive advantage decreases with eccentricity resulting in increased convergence. It would be of interest to know how such differential convergence is accomplished morphologically in terms of the organization of the neuropil at geniculate and striate levels and how this is reflected physiologically in changes in receptive field size.

The present results and those of recent physiological mapping studies (15, 22) suggest that in primates, each topograph-

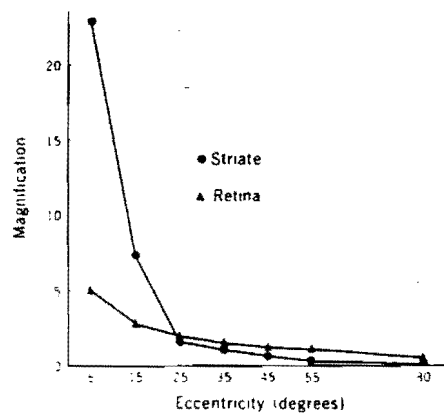


Fig. 1. Magnification in striate cortex and the retinal ganglion cell layer as a function of eccentricity. Values shown are for portions of retina and striate cortex corresponding to representations of concentric zones, each 10 degrees wide, in the visual field from 0 to 60 degrees and of the remainder of the visual field, the zone from 60 to 100 degrees.

ically organized visual structure may be unique in its differential magnification of the visual field. This is indicative of specialization of function in these structures. There exist particularly large differences with respect to magnification in the third tier of cortical visual areas. In the dorsolateral crescent of the owl monkey, approximately 75 percent of the area is devoted to the visual field zone from the center of gaze to 10 degrees, while only 4 percent of the medial area is

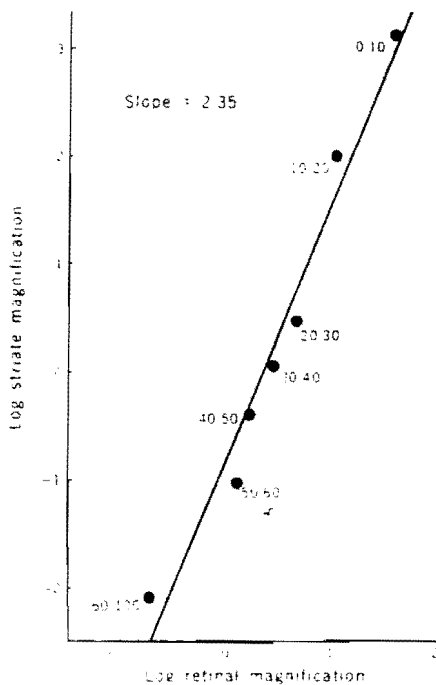


Fig. 2. Magnification in striate cortex as a function of magnification in the retinal ganglion cell layer. Values shown are for portions of retina and striate cortex corresponding to representations of concentric zones, each 10 degrees wide, in the visual field from 0 to 60 degrees and of the remainder of the visual field, the zone from 60 to 100 degrees.

devoted to the representation of this zone compared with 31 percent of striate cortex. Behavioral techniques are now available for the control of fixation during presentation of eccentric stimuli in monkeys (23). This makes possible psychophysical experiments in which stimulus eccentricity is an independent variable. More accurate specification of the relation between magnification and eccentricity at different levels of neural processing may permit determination of the structure involved in a perceptual task on the basis of psychophysical data.

*Note added in proof.* Our findings explain the results of recent autoradiographic studies (24, 25). After intraocular injection of tritiated proline, transneuronal labeling in foveal (dorsal) striate cortex is much less dense than in peripheral (calcarine) striate cortex in both squirrel monkeys and owl monkeys. This is because the ratio of ganglion cells to cortical neurons increases with eccentricity. In addition, degeneration caused by lesions of the LGN is less dense in dorsal striate cortex than in the calcarine fissure (25). These results and those of Malpeli and Baker (9) indicate that differential convergence in the primate retino-geniculo-striate system is accomplished in two steps: between retina and LGN and again between LGN and striate cortex.

JOEL MYERSON

PAUL B. MANIS

FRANCIS M. MIEZIN

JOHN M. ALLMAN

Division of Biology, California Institute of Technology, Pasadena 91125

#### References and Notes

1. E. D. Adrian, *Brain* 66, 89 (1943); W. Penfield and H. Jasper, *Epilepsy and the Functional Anatomy of the Human Brain* (Little, Brown, Boston, 1954); W. I. Welker and G. B. Campos, *J. Comp. Neurol.* 120, 19 (1963); C. N. Woolsey, W. H. Marshall, P. Bard, *Johns Hopkins Hosp. Bull.* 70, 309 (1942).
2. K. J. Lee and T. A. Woolsey, *Brain Res.* 99, 349 (1975).
3. K. J. Sanderson, *Exp. Brain Res.* 13, 159 (1971); J. R. Wilson and S. M. Sherman, *J. Neurophysiol.* 39, 512 (1976).
4. A. Hughes, *Doc. Ophthalmol.* 30, 33 (1971).
5. J. M. Allman and J. H. Kaas, *Brain Res.* 35, 89 (1971).
6. A. Cowey, *J. Neurophysiol.* 27, 366 (1964).
7. P. M. Daniel and D. Whitteridge, *J. Physiol. (London)* 159, 203 (1961); S. A. Talbot and W. H. Marshall, *Am. J. Ophthalmol.* 24, 1255 (1941).
8. S. V. Webb and J. H. Kaas, *Vision Res.* 16, 1247 (1976).
9. J. G. Malpeli and F. H. Baker, *J. Comp. Neurol.* 161, 569 (1975).
10. The present usage might be termed normalized cellular magnification to distinguish it from linear magnification (millimeters of cortex per degree of visual field) as defined by Daniel and Whitteridge (6). Our definition is similar to that employed by Malpeli and Baker (9) except that it is defined in terms of number of cells and normalized for greater ease of application to diverse structures. In choosing a cellular definition, we make no assumptions about the size or nature of functional elements above the cellular level.
11. See Allman and Kaas (5). Cell density and thick-