shows the effects on the redox state of 'b' and respiration. The sequence of events may be described as follows: 1. addition of DCCD increasingly inhibited respiration and shifted 'b' towards the reduced state; 2. addition of 14 μM PCP stimulated respiration by 50% (with respect to the inhibited rate) and slightly reverted the reduction of 'b'; 3. further addition of 72 μM PCP stimulated respiration by 100% (with respect to the inhibited rate) and caused an extensive oxidation of 'b'; 4. final addition of cyanide plus antimycin A completely inhibited respiration and restablished 'b' in the reduced state, demonstrating the reversibility of PCP effect.

T. cruzi epimastigotes contained ATP in a concentration not very different from those reported for animal tissues such as skeletal and heart muscle[16]. Treatment of epimastigotes with cyanide, CCP and DCCD determined significant diminutions of the intracellular concentrationof ATP (Table I). In accordance with those results, antimycin A inhibited phosphorylation (Table II), as shown by the decrease of ^{32}P incorporation into P_7 and P_0, and by the increase of the intracellular concentration of P_i. The effect on the labelling of P_0 (that represents glucose-6-P and derivates[17]) is explained by considering that part of the ATP required to phosphorylate glucose originated in oxidative phosphorylation.

Table II. Effect of antimycin A on a) $^{32}P_i$ uptake in high-energy phosphate and b) the intracellular concentration of P_i

Experiment	Antimycin A (μg/mg of cells)	Uptake of ^{32}P-phosphate in fractions (μg atom ^{32}P) (g of cells [c]) P_7	P_0	Inhibition of respiration (%)
a)	None	0.8	0.4	—
	0.19	0.3	0.2	80
		Diminution of intracellular P_i (μg atom P) (g of cells [c])		
b)	None	6.0		—
	0.11	−1.0		78

Experiment a). Epimastigotes (16 mg phosphate deficient PD-1 (ref. [7]); 3.9 mM ^{31}P-phosphate (6.0×10^8 cpm/mg atom ^{32}P); 5.0 mM glucose; standard saline medium to 3.0 ml. Incubation in Warburg manometers for 2 h at 30 °C. After incubation the cells were analyzed for the incorporation of ^{32}P in P-fractions. Control Q_{O_2}, 5.0 Experiment b). Epimastigotes, 8.9 mg; glucose, 5.0 mM; incubation for 3 h. Initial P_i concentration, 27 μg atom P per g of cells. Other conditions as in experiment a). Control Q_{O_2}, 9.0. After incubation the cells were analyzed for P_i concentration. [c] Fresh weight.

In conclusion, occurence of oxidative phosphorylation in *T. cruzi* is supported by: 1. the oxidation of reduced cytochrome 'b' after addition of uncouplers (Figures 2 and 3); 2. the consistent effects of antimycin A on the redox state of 'b', respiration and phosphate metabolism (Figure 2 and Table II), and 3. the effect of uncouplers and DCCD on respiration and phosphate metabolism (Figures 2, 3 and Table I). These effects are in good agreement with the presence of a Mg^{2+}-activated ATPase[18] and mitochondrial structures in *T. cruzi*[19–21].

Zusammenfassung. In Epimastigoten von *T. cruzi* wird die oxydative Phosphorylierung bewiesen durch: 1. Beseitigung der Atmungskontrolle durch Zugabe von entkoppelnden Substanzen (CCP und PCP); 2. Hemmung der Phosphorylierung durch Antimycin mit gleichzeitiger Reduktion von Cytochrom 'b'; 3. Atmungshemmung durch Dicyclohexylcarbodiimid (DCCD); 4. Verminderung des intrazellulären ATP durch CCP, DCCD und Cyanid.

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24 May 1973.

[16] C. LONG, *Biochemists' Handbook* (E. and F. N. Spon Ltd., London 1961), p. 783.
[17] L. F. LELOIR and C. E. CARDINI, in *Methods in Enzymology* (Eds. S. P. COLOWICK and N. O. KAPLAN; Academic Press, New York 1957), vol. 3, p. 840.
[18] M. B. R. DE SASTRE and A. O. M. STOPPANI, Fedn. Europ. biochem. Soc. Lett. *31*, 137 (1973).
[19] A. SANABRIA, Expl Parasit. *19*, 276 (1966).
[20] K. SHIBATA, A. A. BENSON and M. CALVIN, Biochem. biophys. Acta *15*, 461 (1954).
[21] This investigation was supported by grants from Consejo Nacional de Investigaciones Cientificas y Técnicas (Argentina), Instituto Nacional de Farmacologia and the Wellcome Trust. We are grateful to Professor E.D.P. DE ROBERTIS for allowing the use of the splitbeam spectrophotometer and to M.B.R. DE SASTRE for his assistance with the measurements of ATP.
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Visual Cortical Cells: How Critical is Focus?

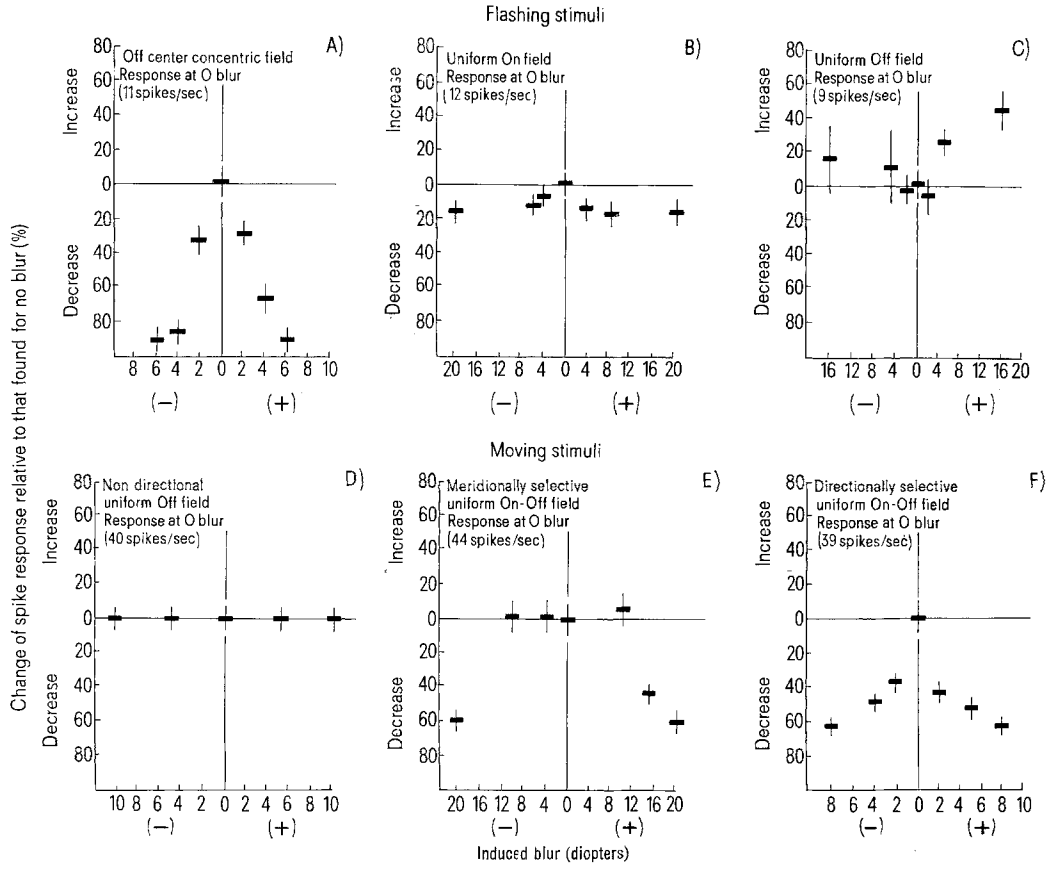
Although performance limits of the retina-central pathways can now be defined using techniques which essentially by-pass the optics of the eye[1–3], little, yet, is known of the specific effects of image blur on single cell performance at successive levels of these pathways.

Initial observations of cat retinal ganglion cell responses do indicate, however, that refractive errors of as little as 0.25 diopters can often be detected through use of a set of neurophysiological criteria[4,5]. A similar susceptibility to blur, but generally less acute, has been demonstrated for particular cells of the rabbit superior colliculus, as well[6].

But how critical is image focus to cortical cell performance? Is the efficiency of every cell equally degraded by blur? And, how is responsiveness to specific trigger-features in the environment, e.g. motion and direction, affected?

Material and method. 123 photically responding cortical cells of the rabbit were investigated here, using the same experimental methods and conditions as in the superior colliculus study reported earlier[6]. As previously, the receptive field of each cell was repeatedly plotted through a series of induced spherical refractive errors.

Results and discussion. The Figure illustrates 6 classes of cortical cell performance in the presence of induced retinal blur. The upper 3 profiles are in response to flashing $^1/_2{}^\circ$ spot where: (A) shows a cell with a very high susceptibility to induced blur (representative of about



Percent change of spike response per sec with dioptrically induced blur, relative to the spike response produced under conditions of no blur, i.e. 0 dioptric error. Convergent lenses (+) were used to induce myopic blur; divergent lenses (−) were used to induce hyperopic blur. Each bar is the mean of 10 or more observations at that blur condition. One standard deviation is indicated above and below each mean.

A) Off center concentric field. Response at 0 blur (11 spikes/sec).
B) Uniform On field. Response at 0 blur (12 spikes/sec).
C) Uniform Off field. Response at 0 blur (9 spikes/sec).
D) Non directional. Uniform Off field. Response at 0 blur (40 spikes/sec).
E) Meridionally selective. Uniform On-Off field. Response at 0 blur (44 spikes/sec).
F) Directionally selective. Uniform On-Off field. Response at 0 blur (39 spikes/sec).

70% of those cells responding to this stimulus); (B) shows a cell whose response efficiency is much less affected by blur (representative of about 20% of the flash responding cells); and (C) a rare cell type which becomes somewhat more responsive to the blurred (scattered) rather than to the sharply focused version of this stimulus.

The lower 3 profiles are in response to moving stimuli. Here too a spectrum of susceptibilities to blur were found ranging from virtually no effect, as illustrated by a non-directional unit in (D), to cells with a limited range of immunity, as seen for the meridionally selective unit in (E), to cells of high susceptibility to blur, as represented by the directionally selective unit in (F).

Can the response profiles above be predicted and/or explained? While many factors must certainly be in play, 2 observations might be emphasized at this time: First, that where stationary, flashing stimuli are used, the relationship of the stimulus area to the agonistic field response area is most critical. A 'spilling' of flux into a clearly antagonist zone due to stimulus blur results in the commonly described lateral inhibition effects. This relationship is most critical for small fields, such as the one illustrated in (A), of the Figure and least critical for large fields as illustrated in (B) and (C).

Second, those movement responsive cells most finely tuned for directionality, such as shown in (F), appear most commonly and critically susceptible to blur, while those less finely tuned, as in (E), or not tuned at all, as in (D), seem generally less susceptible. The specific explanations of such effects, however, must await a fuller deciphering of the underlying neural mechanisms of such cells.

Zusammenfassung. Nachweis, dass rezeptive Kortexfelder der Kaninchen (70% der visuellen Zellen) auf Lichtstimuli bzw. Schärfe-Unschärfe-Änderung mit Amplitudenabfall reagieren.

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[1] F. Campbell and D. Green, J. Physiol., Lond. *181*, 576 (1965).
[2] F. Campbell, J. Kullkowski and J. Levinson, J. Physiol., Lond. *187*, 427 (1966).
[3] D. Mitchell, R. Freeman and G. Westheimer, J. opt. Soc. Am. *57*, 246 (1967).
[4] H. Ikeda and R. M. Hill, Nature, Lond. *229*, 557 (1971).
[5] H. Ikeda, M. J. Wright, S. Young and J. Nuza, Vision Res. *13*, 867 (1973).
[6] J. M. Lee and R. M. Hill, Experientia *28*, 1053 (1972).
[7] This work was supported by U.S.P.H.S. Grant No. EY00576.