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# CORRELATION OF ACTIVITY IN NEIGHBOURING GOLDFISH GANGLION CELLS: RELATIONSHIP BETWEEN LATENCY AND LAG

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SUMMARY

1. Pairs of retinal ganglion cells in the isolated goldfish retina were recorded simultaneously with a single electrode. Repeated flashes of light were delivered to evaluate the response latency of each of the units.

2. The cross-correlation histogram for the maintained discharge of each pair of cells was examined, and its temporal relationships (lags) were compared with the differences in response latencies of the two units. There was a strong correlation between these measures; however, the differences between latencies were often at least twice as great as the lags.

3. The differences between the times to the peaks of the responses of the two units were less reliably related to the lags of the pairs, although the correlation was positive and the differences in time-to-peak generally greater than the lags. The weaker relationship between the difference in time-to-peak and lag than between latency difference and lag is apparently a manifestation of a negative correlation between latency and rise time (from first response to peak). This indicates that cells with a longer latency compensate with a faster rise time.

4. There was a negative correlation between the mean maintained rate of a neurone and its response latency. That is, cells with faster maintained discharge rates respond sooner than those with slower maintained rates.

5. There was virtually no relationship between the lags or the differences in latency and the differences between the magnitudes of the responses to light. Thus, it is unlikely that differences in latency (or lags) could be attributed to unequal effectiveness of the stimuli for the two units.

6. The relationship between differences in latency and lags did not depend on the response categorizations of the two units. Specifically, it did not matter whether the members of the pair were on centre, off centre or on–off centre; neither did it matter whether they were X-like or not-X-like neurones.

7. Consideration of these data leads to the conclusion that there must be 'marked' pathways of differential conduction velocity through the retina.

## INTRODUCTION

The source of the variability in the spike discharge of ganglion cells of the vertebrate retina is unknown. However, the existence of statistical dependencies between the maintained discharges of neighbouring ganglion cells, as witnessed by

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cross-correlation statistics (Rodieck, 1967; Arnett, 1978; Arnett & Spraker, 1981; Levine & Johnsen, 1982) clearly demonstrates that some component of the noise is common to more than one ganglion cell. This 'common noise' must be generated before the ganglion cell level (assuming no cross-talk between ganglion cells).

There is also a significant relationship between the nature of the cross-correlation of a pair of ganglion cells and their response types: cells which have similar response types (e.g. two off centre cells) have a positive cross-correlation, while cells with complementary response types have negative cross-correlations (Arnett, 1978; Arnett & Spraker, 1981; Levine & Johnsen, 1982; K. S. Ginsburg, J. A. Johnsen & M. W. Levine, unpublished observations). This suggests that the source of the common noise lies before the point of sign inversion, most probably as far distally as the outer plexiform layer.

Another noteworthy feature of cross-correlations is that the peaks (or valleys) do not necessarily occur at the zero point (which is the point representing simultaneous events in the two cells). In our experience, positive or negative shifts (lags) of more than 20 ms are not uncommon.

If the common noise source of ganglion cell discharge is located as far distally as the outer plexiform layer, then this noise would have to travel virtually the same path through the retina as do the signals which convey visual information. This leads to a prediction concerning the relation between the lag of the cross-correlation of a cell pair and the response latencies of the individual cells of the pair: if the noise source is at or near the level at which the visual signal originates, then the difference between the response latencies of the two cells should equal the cross-correlation lag (which is the difference in latency of the common noise). To test this hypothesis, we compared the differences in response latencies of pairs of ganglion cells with the lags of their cross-correlations.

## METHODS

Action potentials (spikes) were recorded from the ganglion cells of isolated goldfish retinae (MacNichol & Svaetichin, 1958). Retinae were isolated in dim red light (Shefner & Levine, 1979), and maintained with a flow of pure, moist oxygen (Abramov & Levine, 1972). Following isolation, retinae were placed in a chamber that excluded stray light, and, except for stimulus flashes, remained in complete darkness throughout the experiment. The procedures are described more fully by Shefner & Levine (1979), who also provide evidence of the health, stability and sensitivity of this preparation.

A single platinum–iridium electrode (Wolbarsht & Wagner, 1963) recorded signals from two cells simultaneously; a time and amplitude window discriminator separated the spikes from the two units (K. S. Ginsburg, J. A. Johnsen & M. W. Levine, unpublished). Occasional inevitable errors led to missed or spurious simultaneous spikes; these only affected cross-correlations within 1 ms of zero lag. The time of occurrence of each spike produced by each unit was recorded to the nearest millisecond by a Hewlett-Packard 2100A minicomputer, and transmitted to a PDP 11/03 microcomputer for storage on floppy disk.

The data collection time was divided into discrete periods of 9–30 s, separated by at least 10 s; 8–108 identical periods comprised a single experiment. Photic stimuli were presented at a fixed time, and for a fixed duration (1–10 s), during each data period. The stimuli were 710 nm lights, ranging (across experiments) from 0·32 to 1·0 mm in diameter at the retina. Stimulus sizes were selected arbitrarily before the recording was begun; there was rarely time to measure the receptive fields of the units. We have always found that the receptive fields are concentric with the electrode; since the centres are about 1·25 mm in diameter with little variance (Daw, 1968; Spekreijse, Wagner & Wolbarsht, 1972), centres of adjacent cells must in effect be completely overlapping, and our stimuli confined to the centre region of the cells' receptive fields.

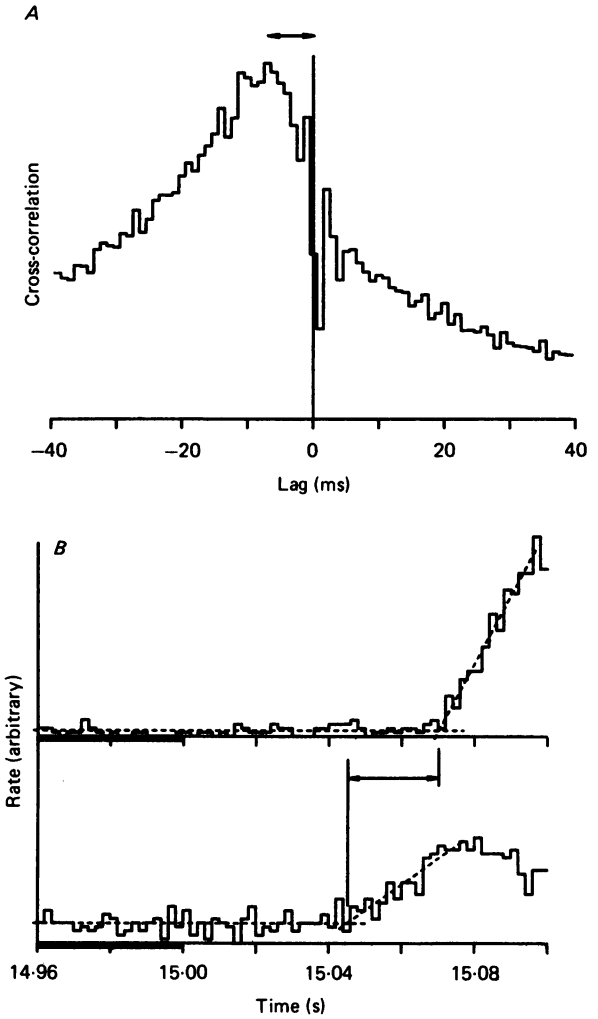


Fig. 1. *A*, measurement of cross-correlation lag for a pair of ganglion cells. Cross-correlation histogram in 1 ms bins; arrow indicates lag (−7 ms). *B*, measurement of response latency difference from p.s.t.h.s for the same pair of cells as in *A*. Responses in 2 ms bins for a period beginning 40 ms before offset of a steady $4{\cdot}6 \times 10^{11}$ quanta $cm^{-2}$ $s^{-1}$ (710 nm) light, 0·56 mm in diameter (light indicated by bars under the abscissae). Horizontal dashed lines indicate the mean firing for the preceding 3 s; sloping dashed lines are the final iterative fits to the rising portions of the responses. Latency difference (−24 ms) indicated by arrow.

Retinal irradiances were adjusted with neutral density filters to 0·5–1·5 log units above the absolute thresholds for the selected stimuli. The levels chosen were sufficient to elicit a small but detectable response from each of the units. These stimuli were effective only for cones sensitive to long wave-lengths, although shorter wave-length stimuli would have elicited rod-driven responses (Shefner & Levine, 1979). Stimuli were within 2 log units of the maximum available, which was $1{\cdot}46 \times 10^{12}$ quanta $cm^{-2}$ $s^{-1}$ (calibrated with a United Detector Technology optometer, model 40A).

Cross-correlation histograms (Perkel, Gerstein & Moore, 1967) with a 1 ms bin-width were derived from the simultaneous spike trains. In each data period, only the periods before the onset of a stimulus were used for cross-correlations. The lag was taken as the amount of time by which the peak (or valley) of the cross-correlation was displaced from the origin (see Fig. 1 *A*).

The response latency of each of the units was computed from peristimulus time histograms (p.s.t.h.s) averaged from all the data periods. We used a modification of the technique presented by Cleland & Enroth-Cugell (1970) that would allow for automatic computation of latency. P.s.t.h.s representing the 200 ms following stimulus onset or offset were plotted with a 2 ms bin-width. A period extending from the stimulus event to a time before the rising portion of the response began to decline was selected, and a straight line was fitted to the data in that period by linear regression. The intersection of the regression line with a horizontal line representing mean maintained discharge (calculated from a period of at least 1 s preceding the stimulus event) delineated a new start-time. Regression was then repeated on the data from the new start-time to the chosen end-time, yielding a new intersection with the mean maintained firing. This procedure was iterated for ten cycles, at the end of which the regression line represented an excellent fit to the rising portion of the response, and the intersection represented our estimate of the time at which the response began; the latency was then taken as the time from the stimulus event to the intersection. Fig. 1*B* gives an example of this procedure, with the final regression lines and the mean levels shown as dashed lines.

We also evaluated latencies by fitting lines to the cumulated (integrated) histograms, as suggested by Enroth-Cugell, Hertz & Lennie (1977; see also Lennie, 1981). We found that the cumulative method could sometimes result in a patently absurd result because of an aberrant burst of activity, while the lines fitted to ordinary histograms always gave results in general agreement with what one would estimate by eye. We therefore used only the regression fits to histograms to measure latencies.

When both cells in a pair were off centre, or at least one member was on–off, a rising portion of the response of each cell occurred at the same phase of the stimulus. In five pairs, however, an on centre cell was paired with an off centre cell; in these cases, the latency of the one unit was measured at stimulus onset, and that of the other unit at offset. In such instances we also computed the latencies for the initiation of reduced firing at stimulus onset for the off centre cells by fitting a line of declining slope to each p.s.t.h. The declining slopes were quite shallow, and therefore the measured latencies less reliable than those measured from rising slopes, but the general result was essentially unaffected.

Measurements of the time of the peak of the responses were made using a dome-shaped (Gamma distribution) pattern, arbitrarily designed to include a weighted average of thirteen successive bins. This pattern was convolved with the 2 ms bin-width p.s.t.h.; the location of the peak of the dome at the maximum value of the convolution was taken as the time of the response peak. This is essentially the same as one of the latency measurements used by Bolz, Rosner & Wässle (1982), except that our distribution was broader than the one they used and had a slight skew to allow for the expected response shape.

## RESULTS

Analyses were performed on twenty-six pairs of units recorded from twenty-four retinae. For each pair we compared the cross-correlation lag (larger spike unit leading the smaller spike unit is positive lag) with the difference in the response latency measurements (smaller spike latency minus larger spike latency). A scatter plot of these results is shown in Fig. 2.

It is clear from Fig. 2 that the lag and the latency difference are correlated ($r = 0{\cdot}77$, $P < 0{\cdot}0001$). Moreover, it is striking that, with a single exception, all the points fall within the triangles bounded by the ordinate and the line of unity slope passing through the origin. That is, the latency difference, while correlated with the lag, is larger. In fact, the best-fit line through the origin (dashed in Fig. 2) has a slope of 2·27. As hypothesized, there is a relationship between the lag and the latency difference; however, the latency difference is generally more than twice as great as the lag.

We also asked whether lag might be related to other characteristics of the cells' responses. One parameter considered was a different kind of latency: the time to the peak of the response. The difference between time-to-peak responses are plotted

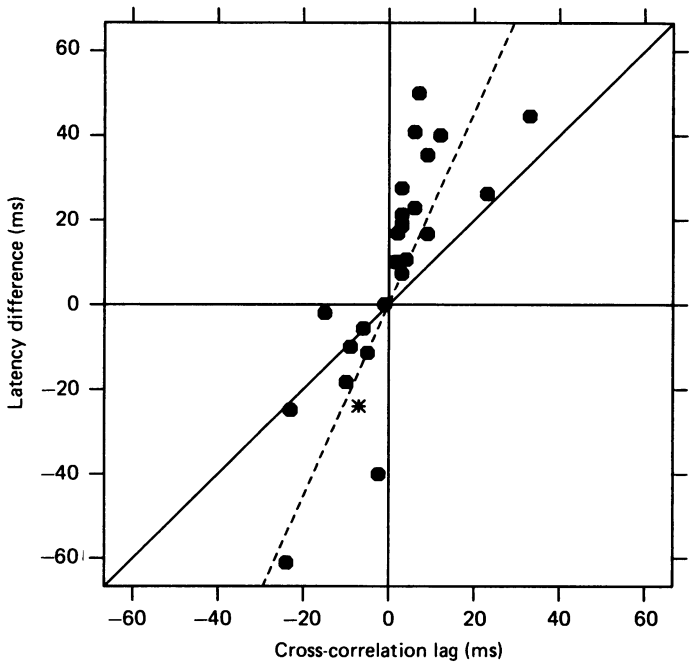


Fig. 2. Scatter plot of latency difference *versus* lag for twenty-six pairs of ganglion cells. The continuous diagonal indicates perfect agreement (latency difference equals lag). The dashed line is the best fit to the points, constrained to pass through the origin. The pair of cells shown in Fig. 1 is indicated by an asterisk.

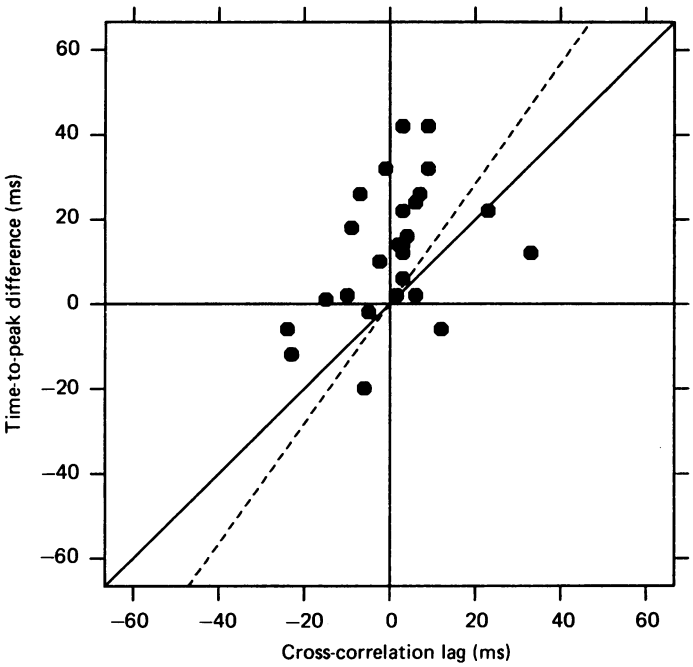


Fig. 3. Scatter plot of differences in time-to-peak response *versus* lag for the same pairs of cells shown in Fig. 2. Conventions are as in Fig. 2, except that the pair shown in Fig. 1 is not specially marked.

against the lags in Fig. 3; the correlation is clearly weaker. While many points lie in the first and third quadrants, seven fall in the second or fourth, for which the signs of the time-to-peak difference and lag are opposites. The correlation between time-to-peak difference and lag is 0·40, barely significant at the level of $P < 0·05$. The slope of the best-fit line through the origin is 1·42, less than the relationship between latency difference and lag, but apparently still implying a greater latency difference than lag.

In order for the time-to-peak difference to be so weakly related to lag, compared with the striking relationship between latency difference and lag, the rise time (from the beginning of the response to the time-to-peak) must partially compensate for the latency. In fact, we found the differences between the rise times to be negatively correlated with latency differences ($r = -0·79$, $P < 0·0001$). That is, the cell in a pair which had the longer latency tended to have the shorter rise time. While this effect was very strong for pairs of cells, when the fifty-two cells were treated individually the correlation between rise time and latency proved to be barely significant ($r = -0·29$, $P < 0·05$). Thus, cells can be faster or slower in both latency and rise time depending on the particular retina or stimulus, but within a simultaneously recorded pair the rise time and latency tend to cancel.

We also sought the correlation between latency difference and parameters of the cells' firing. We found a negative correlation between the difference in mean firing rates and latency difference ($r = -0·54$, $P < 0·005$), indicating that the unit with the higher mean firing rate tended to have a shorter latency than the one with a lower mean rate. Correlation is also evident when cells are considered individually: the correlation between mean maintained firing and latency is $-0·47$ ($P < 0·005$). This can be explained by assuming that the cell with a higher rate is nearer threshold for spike generation, and therefore can produce a response (to either light or a fluctuation in the common noise source) sooner than the cell with a lower rate. While this could explain a latency difference and a lag (and the negative correlation between rise time and latency), it cannot explain why the latency difference is so much larger than the lag; this mechanism should add equally to both latency and lag.

We were concerned that the latency differences and lags might each depend on some other factor, and their correlation be secondary to those relationships. The most obvious source of such an artifact would be if the stimuli were differentially effective for the two neurones in each pair. We therefore considered the difference in response magnitudes (numbers of spikes in the first second after stimulation minus the mean maintained rate), but found no significant correlation between the difference in response magnitudes and the latency difference ($r = 0·31$) or the lag ($r = 0·33$). There is also no significant correlation between the difference between the peak of the responses (the maximum values of the convolutions used to locate the peaks) and the latency difference ($r = 0·10$) or the lag ($r = 0·28$). It thus does not appear that either the latency differences or the lags are a result of stimuli being differentially effective for the two units.

Even if the response magnitudes do not correlate with the latency differences or lags, it remains possible that the stimuli were somehow differentially effective in adapting the two cells. The stimulus is the same for both cells (see Methods), but the amount of adaptation could nevertheless depend on whether large or small stimuli

had been used. Enroth-Cugell & Shapley (1973) have shown that the effectiveness of a diffuse light for a cat retinal ganglion cell depends upon the area of the receptive field centre. If our spots were larger than the receptive field centre of the smaller cell in the pair, we might expect greater adaptation of the unit with the larger field. Since there was no significant correlation between stimulus size and the absolute magnitude of the lag ($r = 0{\cdot}12$) or latency difference ($r = -0{\cdot}04$), we cannot explain our results by such a mechanism.

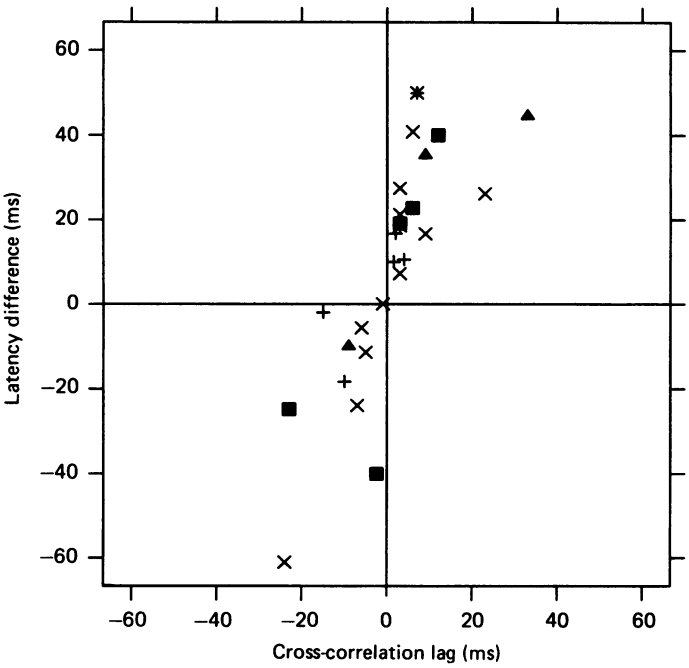


Fig. 4. Scatter plot of latency *versus* lag as in Fig. 2, with cell response types indicated by symbols. Triangles, both cells off centre; squares, one cell on centre, one cell off centre; crosses, on–off cell paired with off centre; plus signs, on–off cell paired with on centre cell; asterisks, both cells on–off. Same data as in Fig. 2.

One final possible artifact must be considered: the cells in each pair might have differed in lag and latency if they were of different response types. To test for this possibility, we flagged the response types of each member of each pair (e.g. two off centre cells, one on–off cell and one off centre cell, and so on) on a scatter plot of latency difference *versus* lag. This plot is shown in Fig. 4. There is no tendency for different pairings to occupy different parts of the plot; each possible pairing can be found anywhere within the cloud of points.

The other possible difference between cells in a pair that might account for the temporal differences could be that one cell in each pair was X-like and the other not-X-like (Levine & Shefner, 1979). If this categorization of goldfish cells is analogous to the dichotomy in mammals (Enroth-Cugell & Robson, 1966; DeMonasterio, 1978; Caldwell & Daw, 1978), one might well expect the X-like units to differ in latency from the not-X-like units (Bolz *et al.* 1982). (It should be noted, however, that each class might contain even slower W-like units (Cleland & Levick, 1974; Stone

& Fukuda, 1974; Sur & Sherman, 1982), which have not been explicitly described for fish.) Using the method of Levine & Shefner (1979), we were able to classify both cells in thirteen of the twenty-six pairs reported here. Of these thirteen pairs, both cells were X-like in five pairs, both were not-X-like in seven pairs, and there was one cell of each type in only one pair. The preponderance of like cells in a pair is not surprising in view of the earlier observation (Levine & Shefner, 1979) that cells come in 'runs', possibly reflecting some selection by particular batches of electrodes. It thus seems likely that there is a similar preponderance of like cells in the pairs for which the information is not available.

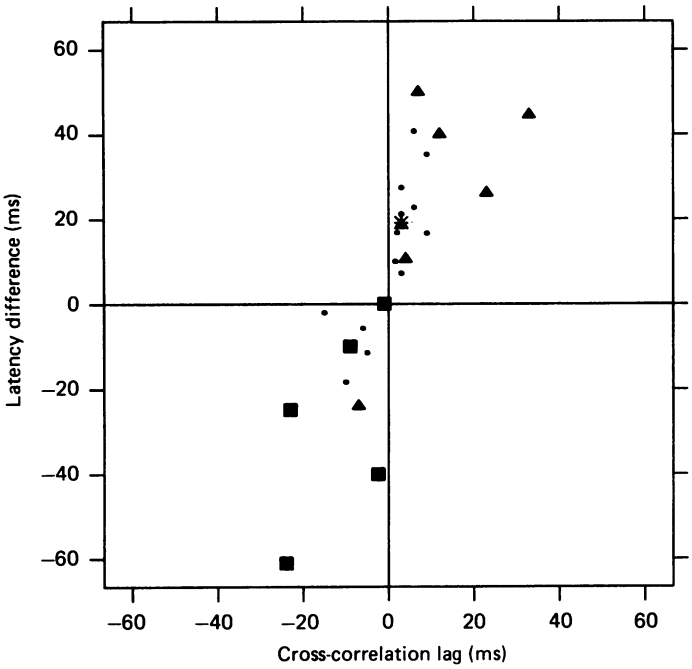


Fig. 5. Scatter plot of latency *versus* lag, with cell types (classified by linearity of spatial summation) indicated. Squares, both cells X-like; triangles, both cells not-X-like; asterisk, one cell X-like, one not-X-like; small circles, cells not classified. Same data as in Fig. 2.

The latency difference is again plotted as a function of the lag, with the cell types indicated, in Fig. 5. There is a curious (and perhaps coincidental) tendency among the five pairs of X-like cells for the cell with the larger spike to be the faster unit, while the opposite tendency was noted for six of the seven pairs of not-X-like units. Nevertheless, the points corresponding to pairs known to have like cells are scattered evenly among the rest of the data points in Fig. 5. It therefore does not seem possible that the differences in latency and the lags were each secondary to the cells in a pair being of a different type.

## DISCUSSION

How may we explain the observation that the latency differences are well correlated with lags, but significantly longer than them? If the common noise source

were introduced into the retinal network at the level of the receptors, and traversed the same pathways as the signals induced by photic stimuli, the latency difference would equal the lag. If the common noise were inserted late in the processing network one would expect minimal lags, and what lag there was would probably not be correlated with the latency difference. One might suggest that there are two common noise sources, one located distally (near or in the receptors: Lamb & Simon, 1977;

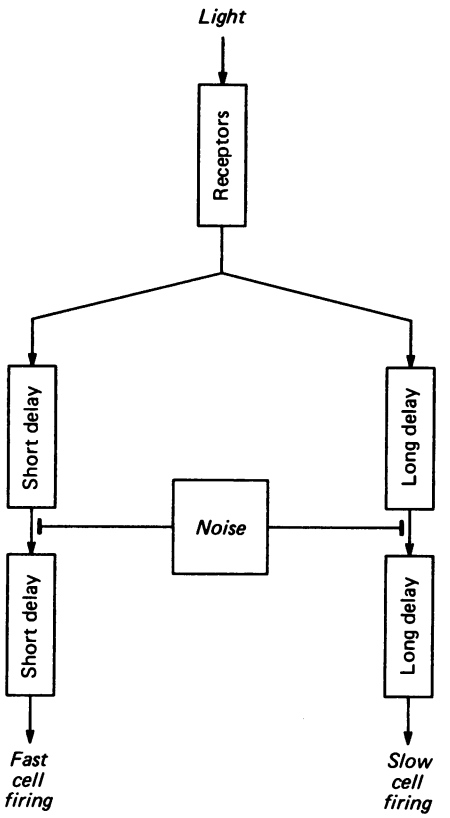


Fig. 6. Model of 'marked' conduction pathways. Photic signals originate in receptors at the top of the Figure, but diverge into 'fast' and 'slow' pathways. Each pathway has serial delays (at least two), with common noise injected part-way through the cascade.

Schwartz, 1977; Ashmore & Copenhagen, 1980; Baylor, Matthews & Yau, 1980) that shares the delays of the photic signals, and one quite proximal (Schellart & Spekreijse, 1973) for which there is no differential delay. The cross-correlation would then be an amalgam of the correlation expected from each source. However, for many of our cross-correlations to have resulted from one peak at the latency difference and one at zero, they would either have to be far broader than they are or show two peaks.

A parsimonious explanation is that there are 'marked' pathways of differential conduction speed in the retina. That is, the signal to one ganglion cell in a pair travels in a slower pathway and thus lags further and further behind that to the other cell at each step through the retina. A representation of this scheme is shown in Fig. 6. The noise can then be inserted part-way between the receptors and the ganglion cells,

and its relative delay on the way to each ganglion cell will be a fraction of the relative delays in the signal pathway from receptors. The longer-latency pathway feeds to a ganglion cell with a lower mean firing rate, further exaggerating both the lag and latency difference. Note that this model works only because the delays in each pathway before the insertion of common noise are correlated with the delays after that point.

An alternative scheme is that the common noise enters after all the delays have operated on the photic signals, but with corresponding differential delays from the common source to each cell. This model seems unnecessarily complicated, since parallel 'marked' delay pathways (one for noise, one for signal) are required. (Note that this model applies to the possibility that dark noise arises in rods while the responses to 710 nm lights arise in cones. However, this configuration seems unlikely since one would expect that the latency difference, which is attributed to the generally faster cone system, would be less than the rod-derived lag; in fact, it is greater.)

Another alternative is that the lag and much of the difference in latency are due to the differences in mean firing rate (discussed above). The 'marking' would then be associated with mean rate, and all of the lag would be at the ganglion cell itself. However, if this were the case, it is hard to see why lag is better correlated with latency difference than either is with the difference in mean firing rates. Moreover, if mean firing rate were the principal determinant of lag, one would expect lag to be altered in the presence of photic stimulation, but it is not (Ginsburg *et al.* 1983). This therefore does not appear to be the main factor in the correlation of lag with latency difference; however, the correlations with rate indicate that it is a factor, and the 'marked' pathways must also recognize or help determine mean maintained firing rate.

However the relative delays are accomplished, it would appear that 'marked' conduction pathways are a feature of retinal processing. The reason for the existence of 'marked' pathways is not patent at this time.

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