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Temporal visual filtering in diabetes mellitus

Nigel Davies [1], Antony Morland *

Biophysics, Imperial College of Science, Technology and Medicine, South Kensington, London SW7 2BZ, UK

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Abstract

The background modulation method was used to investigate the temporal response of the magnocellular pathway in diabetic patients and controls. The luminance threshold for detecting a moving, 2°, achromatic target was measured as a function of background flicker frequency from 5 to 45 Hz. A model of photoreceptor kinetics integrated with difference of Gaussian receptive fields [Vis. Neurosci. 13 (1996) 173] was used to analyse the data. Diabetic patients with significant maculopathy showed raised thresholds at 8.75, 12.5, 15 and 17.5 Hz. Estimates of photoreceptor summation time were the same in both groups, but receptive field centre-to-surround delay showed an increasing trend in the diabetic patients.
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Keywords: Diabetes mellitus; Receptive field; Temporal response; Photoreceptor; Magnocellular

1. Introduction

Diabetic mellitus is a common disease, affecting between 1% and 2% of the population. Diabetic eye disease and in particular diabetic retinopathy is a leading cause of blindness in the working age population in the developed world (Klein, Klein, & Moss, 1984b). The middle and inner retinal layers are affected by diabetes and study of functional changes related to these layers of the visual system is of interest. Presently, the treatment available for sight-threatening retinopathy is based on laser surgery and reduces the incidence of severe visual loss by 50% (Early Treatment Diabetic Retinopathy Study Group, 1985; The Diabetic Retinopathy Study Research Group, 1979). There is increasing evidence that medical treatment will become paramount in reducing the onset and progression of diabetic retinopathy and consequently visual loss (Adler et al., 2000; Stratton et al., 2000; United Kingdom Prospective Diabetes Study Group, 1998a, 1998b). Visual function parameters may be useful as monitors of disease progression or of efficacy of treatment.

Visual function in the spatial domain in patients with diabetes has been investigated extensively using contrast sensitivity (Arden, 1978; Banford, North, Dolben, Butler, & Owens, 1994; Bangstad, Brinchmann Hansen, Hultgren, Dahl Jorgensen, & Hanssen, 1994; Brinchmann Hansen, Dahl Jorgensen, Hanssen, & Sandvik, 1992; Chylack et al., 1993; Collier, Mitchell, & Clarke, 1985; Della Sala, Bertoni, Somazzi, Stubbe, & Wilkins, 1985; Di Leo et al., 1992; Dosso et al., 1996; Ghafour, Foulds, Allan, & McClure, 1982; Harris et al., 1996; Howes, Caelli, & Mitchell, 1982; Hyvarinen, Laurinen, & Rovamo, 1983; Khosla, Talwar, & Tewari, 1991; Moloney & Drury, 1982; Sokol et al., 1985; Trick, Burde, Gordon, Santiago, & Kilo, 1988), with variable results that may be due to the non-selective nature of a grating or letter contrast sensitivity function (Sokol et al., 1985).

Temporal processes within the diabetic eye have not been studied to the same degree (Di Leo et al., 1992; Kurtenbach, Neu, & Zrenner, 1999; Lobefalo et al., 1997; Scase et al., 1990). Flicker perimetry shows a generalised reduction in critical flicker fusion frequency in a group of diabetic patients (Lobefalo et al., 1997). Hue discrimination worsened in diabetics with decreasing stimulus presentation time (Scase et al., 1990), a finding consistent with worsening of wavelength discrimination as stimulus presentation time is shortened (Kurtenbach et al., 1999). In another study, contrast sensitivity of an antiphase grating at 8 Hz was abnormal

* Corresponding author. Address: Department of Psychology, Royal Holloway College, Egham, Surrey, UK. Tel.: +44-1784-443520.
E-mail address: a.morland@rhbnc.ac.uk (A. Morland).
[1] Department of Ophthalmology, Chelsea and Westminster Hospital, 369 Fulham Road, London, UK.

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over a wide range of spatial frequencies (Di Leo et al., 1992).

In this study we have used the second of two achromatic spatiotemporal responses (Barbur & Ruddock, 1980; Holliday & Ruddock, 1983) that are relatively independent of changes in the ocular media. The response is designated 'ST2 temporal' and is obtained measuring the detection threshold of an achromatic moving target against a suprathreshold achromatic background of pseudo-sinusoidal flicker as a function of flicker frequency. This gives a response that is characteristic of the temporal properties of the magnocellular pathway (Holliday & Ruddock, 1983).

2. Methods

Although the original work on the spatiotemporal responses was performed using a Maxwellian view optical system, it was later shown that the functions could be reliably obtained using a system of free viewing (Morland, Bronstein, Ruddock, & Wooding, 1998). A two-beam projector system using 200 W tungsten–halogen bulbs was used to elicit the ST2 temporal response. The projectors were arranged just to the side of the patient, who sat 1.5 m from a viewing screen. The background was projected through an aperture giving a circle of 17° visual angle in diameter. The target subtended 2° of visual angle and its excursion was fixed over the central 10° of the background field. With central fixation on the background, the target excursion on the retina was approximately 3 mm, passing over the macular region defined by the Early Treatment Diabetic Retinopathy Study (Early Treatment Diabetic Retinopathy Study Group, 1985). The detection threshold of the 2° target, moving at a velocity of 20°/s across the central 10° was measured using a single staircase controlled by the operator (the first author for all subjects). An estimate of the error was made from the smallest step size that resulted in a change in response around the threshold point.

The observer wore a pair of trial frames. The left eye was occluded with a blank, whilst the right eye viewed the screen through a 1 mm diameter pinhole (0.785 mm^2), to remove inter-individual variation of pupil size. The observer was instructed to fixate the central region of the background and to respond 'yes' whenever the target was seen traversing the centre of the image and 'no' if the background appeared unchanged during the stimulus presentation. Any sensation of movement across the screen during presentation was reported as a 'yes'.

The light output of both projectors was calibrated for each subject, to ensure accurate control of stimulus conditions. The mean background luminance was 63 cd/m^2 for the ST2 temporal response giving retinal illuminance of 1.70 logT when viewed through a 1 mm diameter pinhole.

The study had approval from the Research and Ethics Committee of St. Mary's Hospital, Imperial College of Science, Technology and Medicine, London, UK. All patients gave written consent to be involved and the tenets of the Declaration of Helsinki were observed. Twenty-two diabetic and 12 controls with normal visual acuity performed the experiment. The mean age of the diabetic group was 45 years (SD 10.7 years) and of the control group 50.4 years (SD 11.5 years), $p = 0.18$. The diabetic patients were examined using slit lamp biomicroscopy with 90 and 60 Dioptre biomicroscope lenses and retinopathy was graded using the modified Airlie House classification system (Klein et al., 1984a). The diabetic group contained 12 patients with no maculopathy, three with grade 1 maculopathy and seven with grade 2 maculopathy. Twelve patients had had no laser treatment, four patients had had macular photocoagulation, five had had pan retinal photocoagulation and one had had both forms of treatment. Ten patients had retinopathy levels 1–2, six patients level 3 and six patients level 6 retinopathy. Blood glucose and glycosylated haemoglobin levels were measured for each patient at the end of the test. The mean blood glucose level was 12.1 mmol/l (SD 5.3) and the mean HbA1c level was 8.3% (SD 1.8).

3. Modelling of the ST2 temporal response

The temporal component of a computational model (Donner & Hemila, 1996) was used to analyse the data. In brief, the model is split into a consideration of the photoreceptor response, ganglion cell response, the stimulus function, and the spatial and temporal responses. The model predicts the output of a single ganglion cell to spatiotemporal stimulation from a drifting, sinusoidal grating. For greater detail the reader is referred to the original work.

A linear response from the photoreceptor and the ganglion cell is assumed. Although the assumption of linearity is restrictive it is applicable to a response obtained at threshold, as in the ST2 temporal experiment.

The photoreceptor response is based on a Poisson kinetic, the order of which depends on the animal species studied and the type of photoreceptor under investigation. Hood and Birch (Hood & Birch, 1993) analysed the *a-wave* of the ERG in humans in terms of Poisson kinetics and found that an order of six gave the best-fit for human cone responses. This value has been used successfully to predict flicker sensitivity as a function of temporal noise (Rovamo, Raninen, Lukkarinen, & Donner, 1996) and the Poisson order has been taken as 6 for this series of calculations.

The model shows that the spatial and temporal components of ganglion cell response are separable in a linear system. With a stimulus of pure flicker and no spatial modulation, the temporal response of a ganglion cell with a balanced circularly symmetric difference of Gaussian receptive field can be described by reducing the Donner and Hemila model to:

$$U = H\sqrt{2 - 2\cos(2\pi fd)} \tag{1}$$

where

$$H = (1 + N^2 f^2 t_i^2)^{-n/2} \tag{2}$$

$$N = \frac{2\pi(n-1)^{n-1}}{e^{n-1}(n-1)!} \tag{3}$$

with n as the order of the Poisson response, f the flicker frequency of the background, t_i the photoreceptor summation time and d the centre-to-surround delay in the ganglion cell receptive field.

Table 1
Mean threshold data for the ST2 temporal response

Flicker frequency (Hz)	Mean controls	SD	Mean diabetics	SD	T test p
5.00	−0.51	0.19	−0.35	0.33	0.09
6.25	−0.46	0.19	−0.25	0.29	0.01
7.50	−0.38	0.20	−0.17	0.20	0.01
8.75	−0.28	0.20	−0.08	0.19	0.01
10.00	−0.18	0.20	−0.05	0.21	0.08
12.50	−0.20	0.18	−0.06	0.17	0.04
15.00	−0.27	0.15	−0.09	0.22	0.01
17.50	−0.36	0.14	−0.19	0.19	0.01
20.00	−0.44	0.21	−0.35	0.23	0.24
22.50	−0.55	0.17	−0.48	0.20	0.32
25.00	−0.56	0.21	−0.56	0.19	0.97
27.50	−0.70	0.20	−0.68	0.19	0.80
30.00	−0.77	0.21	−0.79	0.20	0.87
32.50	−0.84	0.17	−0.86	0.17	0.68
35.00	−0.96	0.12	−0.90	0.16	0.28
37.50	−1.01	0.17	−0.96	0.16	0.36
40.00	−0.99	0.19	−0.96	0.16	0.66
45.00	−1.03	0.19	−0.95	0.14	0.26

4. Results

The mean logMAR visual acuity for the diabetic group was 0.04 log units (SD 0.08). The mean threshold and standard deviation for the ST2 temporal responses were calculated in each subject group and are given in Table 1 along with two sample comparisons (unpaired t test). The Bonferroni correction for multiple t tests was used to set the level required for significance at $p = 0.003$. At this level there were no statistically significant differences in threshold between the two groups. The diabetic patients were separated into groups with respect to grade of maculopathy and the mean values calculated for each group and compared with each other and with the control group, the results shown in Table 2. Statistical comparison of these results is shown in Table 3. The mean threshold values were significantly greater for the grade 2 maculopathy group at frequencies 8.75, 12.5, 15 and 17.5 Hz in comparison with the control group.

The mean thresholds for diabetics and controls are plotted in Fig. 1 and the means for the patients with respect to grade of maculopathy are shown in Fig. 2.

For the diabetic patients there is a rise in the thresholds of the low frequency slope and an increase in

Table 2
Mean thresholds for the control group, the diabetics with grade 0 and 1 maculopathy and the diabetics with grade 2 maculopathy for the ST2 temporal response

Flicker frequency (Hz)	Controls mean	SD	Grade 0 and 1 mean	SD	Grade 2 mean	SD
5.00	−0.51	0.19	−0.38	0.36	−0.28	0.28
6.25	−0.46	0.19	−0.28	0.32	−0.17	0.22
7.50	−0.38	0.20	−0.20	0.20	−0.11	0.19
8.75	−0.28	0.20	−0.11	0.20	0.00	0.15
10.00	−0.18	0.20	−0.09	0.21	0.03	0.21
12.50	−0.20	0.18	−0.12	0.16	0.08	0.13
15.00	−0.27	0.15	−0.15	0.23	0.04	0.12
17.50	−0.36	0.14	−0.26	0.18	−0.05	0.12
20.00	−0.44	0.21	−0.37	0.25	−0.28	0.17
22.50	−0.55	0.17	−0.49	0.23	−0.45	0.14
25.00	−0.58	0.21	−0.56	0.21	−0.54	0.17
27.50	−0.71	0.20	−0.68	0.22	−0.69	0.12
30.00	−0.79	0.21	−0.80	0.21	−0.76	0.18
32.50	−0.88	0.17	−0.86	0.20	−0.87	0.11
35.00	−0.98	0.12	−0.92	0.17	−0.87	0.13
37.50	−1.01	0.17	−0.99	0.18	−0.90	0.10
40.00	−0.99	0.19	−0.99	0.18	−0.91	0.10
45.00	−1.03	0.19	−0.98	0.16	−0.76	0.34

Table 3
Comparison of means for the grouped data given in Table 2 for the ST2 temporal response

Flicker frequency (Hz)	Control vs. grade 0 and 1	Control vs. grade 2	Grade (0 and 1) vs. grade 2
5.00	0.27	0.09	0.47
6.25	0.08	0.01	0.34
7.50	0.03	0.01	0.33
8.75	0.05	0.001	0.15
10.00	0.22	0.05	0.25
12.50	0.25	0.001	0.01
15.00	0.14	0.001	0.02
17.50	0.13	0.001	0.01
20.00	0.48	0.10	0.34
22.50	0.49	0.21	0.62
25.00	0.95	0.82	0.77
27.50	0.79	0.90	0.86
30.00	0.77	0.87	0.66
32.50	0.75	0.64	0.91
35.00	0.47	0.20	0.55
37.50	0.68	0.08	0.17
40.00	0.95	0.24	0.22
45.00	0.51	0.09	0.14

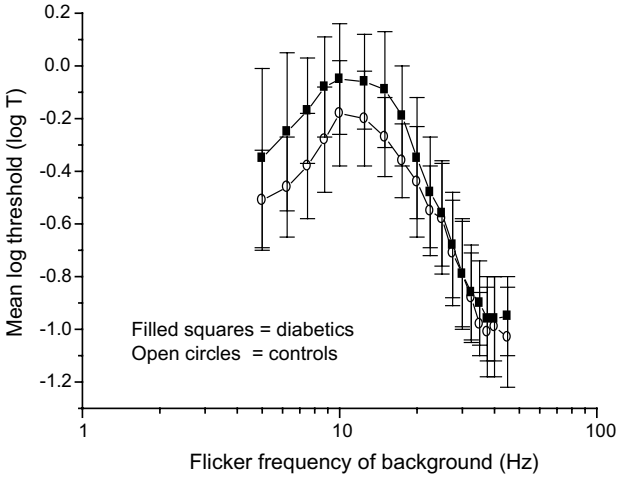

Fig. 1. The ST2 temporal response for the diabetic and control groups. The error bars are ± one standard deviation. The peak of the function occurs at 10 Hz for both groups.

the overall amplitude of the response. The response for the patients with grade 2 maculopathy also shows a slight shift in the peak frequency from 10 to 12.5 Hz.

5. Model fitting

The two freely variable parameters in the temporal model are the receptor summation time (t_i) and the centre–surround delay (d). The effect of variation of these parameters on the model curve is shown in Fig. 3. In Fig. 3(a) the receptor summation time is varied from 15 to 40 ms in 5 ms steps, keeping the centre–surround delay fixed

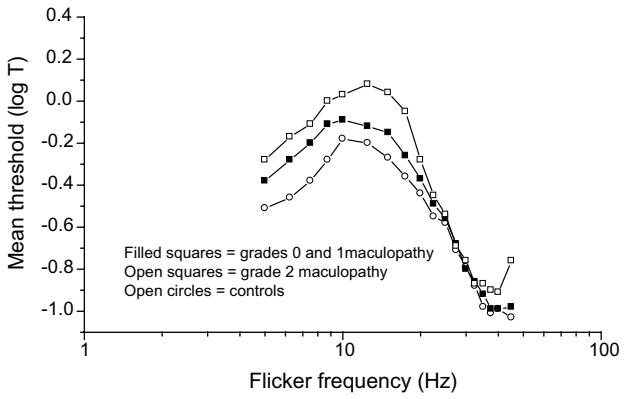

Fig. 2. ST2 temporal response for controls, diabetics with grade 0 or 1 maculopathy and for diabetics with grade 2 maculopathy. Error bars have been omitted for clarity. The means, standard deviations and the inter-group comparisons are shown in the tables below.

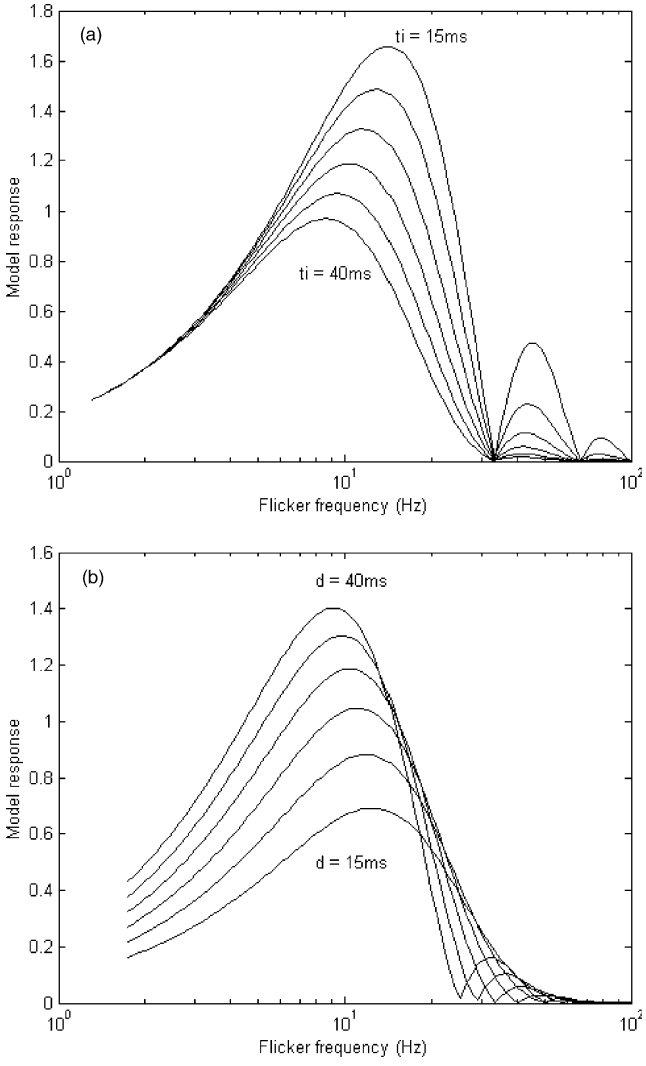

Fig. 3. Model function (a) for increasing t_i in the range 15–40 ms with d held constant at 30 ms (b) for increasing d in the range 15–40 ms with t_i held constant at 30 ms.

at 30 ms. It is seen that increasing t_i has little effect on the low frequency slope, but causes a reduction in amplitude of the response and a shift of the peak frequency to the left. In Fig. 3(b), the receptor summation time was fixed at 30 ms and the centre–surround delay was increased from 15 to 40 ms in 5 ms steps. Increasing d results in an increase in the low frequency slope and an increase in the amplitude of the response. There is also a slight shift in the peak response to a lower frequency.

The results of parametric variation and maximisation of the fit using the least squares method for pooled data for both groups are shown in Fig. 4. Figs. 5 and 6 show the best and worst model fits to the data obtained from individual diabetic and control subjects.

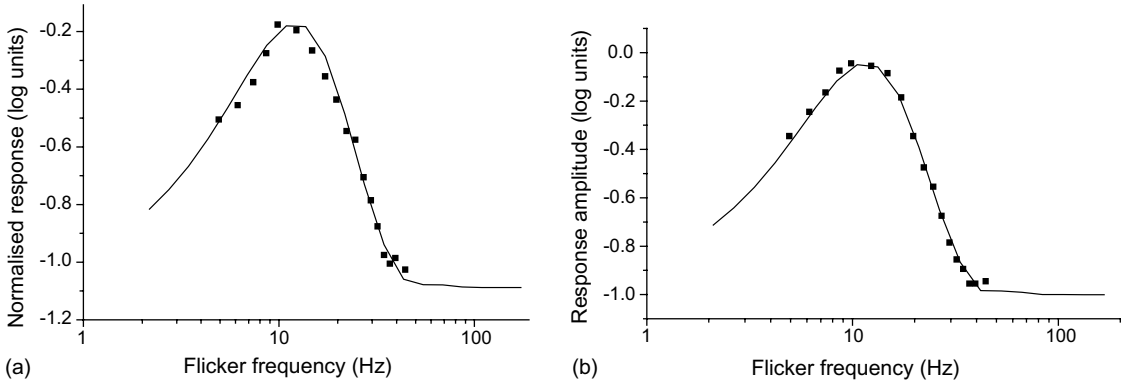

Fig. 4. (a) Best-fit of the temporal model to the pooled experimental data of the control group. The summation time t_i was 29 ms, centre–surround delay 23 ms and $R^2 = 0.99$. (b) Best-fit of model to diabetic group (pooled data). The summation time $t_i = 30$ ms, centre–surround delay $d = 22$ ms and $R^2 = 0.99$.

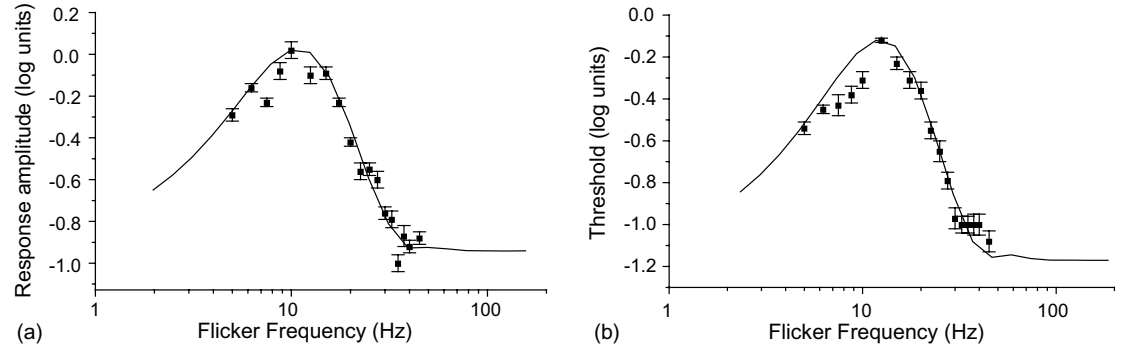

Fig. 5. Best and worst fits to individual data in the control group. (a) Fitting parameters $t_i = 32$ ms, centre–surround delay 24 ms, $R^2 = 0.97$. (b) Fitting parameters $t_i = 21$ ms, centre–surround delay 17 ms, $R^2 = 0.79$.

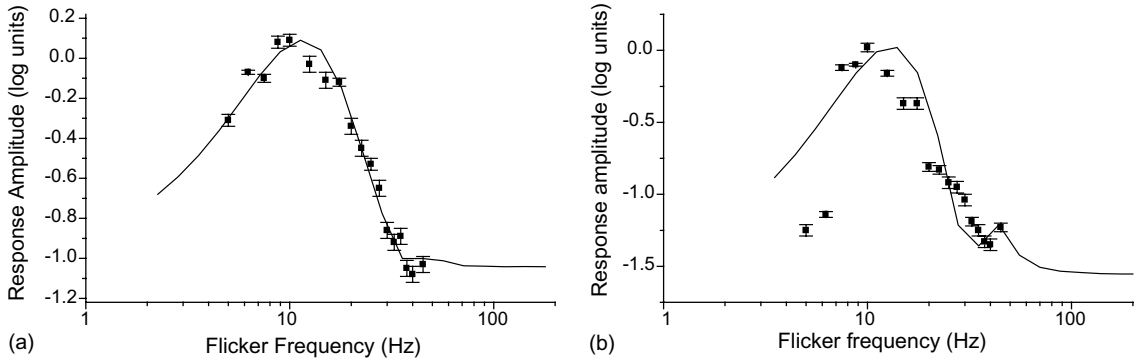

Fig. 6. Best and worst fits for the ST2 model in the diabetic group. (a) Fitting parameters $t_i = 30$ ms, centre-to-surround delay 28.8 ms, $R^2 = 0.98$. (b) Fitting parameters $t_i = 18$ ms, centre-to-surround delay 31.5 ms, $R^2 = 0.73$.

Table 4
Best-fit parameters for ST2 response in controls and diabetic patients

t_i (s)	d (s)	R^2
Control group		
0.023	0.020	0.93
0.026	0.026	0.63
0.037	0.021	0.94
0.027	0.023	0.97
0.029	0.020	0.99
0.032	0.024	0.97
0.037	0.020	0.82
0.026	0.026	0.89
0.026	0.026	0.83
0.029	0.021	0.90
0.021	0.017	0.79
0.027	0.022	0.93
Diabetic patients		
Maculopathy grade 0		
0.035	0.025	0.95
0.030	0.026	0.95
0.029	0.026	0.96
0.029	0.028	0.90
0.025	0.017	0.93
0.042	0.025	0.95
0.031	0.019	0.83
0.025	0.020	0.98
0.028	0.026	0.98
0.035	0.022	0.97
0.024	0.019	0.87
0.023	0.022	0.97
Maculopathy grade 1		
0.035	0.032	0.97
0.018	0.032	0.73
0.026	0.017	0.95
Maculopathy grade 2		
0.024	0.023	0.95
0.030	0.024	0.95
0.032	0.030	0.97
0.024	0.025	0.95
0.025	0.029	0.94
0.028	0.020	0.96
0.031	0.029	0.98

The best-fit parameters obtained for each individuals' results are given in Table 4 with the means and standard deviations in Table 5. Table 6 gives a statistical comparison of the fitting parameters. The results of the curve fitting show a trend in the diabetic patients for an increasing centre–surround delay as the grade of maculopathy increases, although this did not reach statistical significance. The R^2 values for the goodness of fit are very high and the fitting in the diabetic patients is as good as or better than in the controls (see Table 4). The statistically significant difference between the R^2 values in the patients with grade 2 maculopathy in comparison with the controls probably represents a type I error. There was no significant correlation of the best-fit model parameters with either blood glucose or glycoslyated haemoglobin levels.

Table 5
Mean and standard deviations of best-fit parameters for controls and diabetic patients

Group	t_i (s)	d (s)	R^2
Control subjects			
Mean	0.028	0.022	0.882
SD	0.005	0.003	0.102
Diabetic patients			
Mean for whole group	0.029	0.024	0.937
SD	0.005	0.004	0.059
Mean for grade 0	0.030	0.023	0.938
SD	0.006	0.003	0.047
Mean for grade 1	0.026	0.027	0.883
SD	0.009	0.009	0.131
Mean for grade 2	0.028	0.026	0.960
SD	0.003	0.004	0.014

Table 6
Statistical comparison of model fitting parameters (p values, Student's t test)

Group	t_i	d	R^2
Controls vs. all diabetics	0.888	0.128	0.103
Controls vs. grade 0 maculopathy	0.539	0.654	0.104
Controls vs. grade 1 maculopathy	0.728	0.480	0.993
Controls vs. grade 2 maculopathy	0.751	0.066	0.023

6. Discussion

Parametric study of the ST2 temporal function showed the characteristics of a bandpass filter, peaking at around 10 Hz with the same general form as a de-Lange filter (Holliday & Ruddock, 1983). The function saturated at low background contrast modulation depth (30%), was invariant with eccentricity, collapsed for small targets (<0.5°), for slow moving (<1°/s) and for flashed targets. Dichoptic presentation of the stimulus failed to produce the function. The ST2 temporal function thus gives a response that is characteristic of a transient channel in the visual system and is monocular and probably retinal in origin. The use of a model based on a small number of retinal parameters would thus seem appropriate and indeed the model used in this study fits the experimental data extremely well, with correlation coefficients of 0.95 or greater in the majority of subjects. This may be due to the averaging of the output of many ganglion cells in producing the psychophysical response (Donner & Hemila, 1996).

The experimental data show differences in the form of the ST2 function for the diabetic patients, with an increase in the threshold of the low frequency slope and significantly raised thresholds for 8.75–17.5 Hz in the

diabetic patients with grade 2 maculopathy. The results of the model fitting suggest that the response in the diabetics is consistent with an increased centre–surround delay, although for the numbers involved in this study this did not quite reach statistical significance. Interestingly the anatomical disruption in diabetic maculopathy occurs in the middle and inner retinal layers and this is reflected by a change in the inner retinal parameter in the model fit. Using the means and standard deviations of t_i and d for the controls and the patients with different grades of maculopathy we estimate that to detect a significant difference between the groups (at the appropriate level for the number of comparisons involved) would require the test to be performed on 30 patients with each grade of maculopathy.

The time-to-peak response t_p for a photoreceptor is easier to measure in electrophysiological experiments than the summation time, t_i. For Poisson kinetics, Donner and Hemila show that for a sixth order kinetic,

$$t_i = 1.14 t_p \tag{4}$$

Previous values have been given as $t_p = 40$–50 ms for primate cones (Schnapf, Nunn, Meister, & Baylor, 1990), and 35 ms for the dark-adapted cone photoreceptor response in humans (Hood & Birch, 1993). In conditions of light adapation, cone photoreceptor responses accelerate in the ratio (Donner, Koskkelainen, Djupsund, & Hemila, 1995):

$$\frac{t_p^{\text{light}}}{t_p^{\text{dark}}} = \left(\frac{I_{\text{light}}}{I_{\text{dark}}}\right)^{-0.15} \tag{5}$$

The mean value of t_p in the control group is calculated as 24.4 ms (SD 4.7 ms) and for the diabetic group 24.0 ms (SD 3.6 ms). For a background illumination of 63 cd/m^2 and a 'dark background' of between 0.3 and 1 cd/m^2, the results obtained for the receptor summation time in our experiment are in excellent agreement with those obtained from fully dark-adapted cones.

Previous studies of the effect on diabetes on photoreceptor function have produced variable results. In studies of patients with no retinopathy, cone receptor responses appeared to be unaffected whilst inner retinal layers were affected (Di Leo et al., 1994; Ghirlanda et al., 1991). Patients without clinically significant macular oedema had foveal cone implicit times in agreement with those of controls, whilst patients with clinically significant macular oedema showed prolonged implicit time and reduced amplitude (Weiner et al., 1997). Another study using both pyschophysics and electroretinography in patients with varying levels of retinopathy found that both rod and cone receptoral and post-receptoral deficits were present (Holopigian, Greenstein, Seiple, Hood, & Carr, 1997). Other psychophysical studies show specific deficits of the S cone pathway (Hardy, Scarpello, Foster, & Moreland, 1994) with relative preservation of the L and M cone responses, and later studies give evidence that the S cone pathway deficit is probably post-receptoral in origin (Terasaki, Hirose, & Miyake, 1996). In summary it would appear that in diabetes without retinopathy the cones are relatively unaffected, whilst the middle and inner retinal layers are involved and as the retinopathy progresses the functional disturbance expands to include both the receptors and the middle and inner retinal layers. Our study of patients with low grades of maculopathy was unable to detect abnormality in the receptor summation time, which appears consistent with the findings of the above studies.

With regard to the temporal function of the inner retinal layers we find that there is a trend towards an increased centre–surround delay in the diabetic patients as the grade of maculopathy increases, although for the numbers of patients tested here, this did not reach statistical significance. Donner and Hemila note that there is very little information about the total centre-to-surround delay, as published studies fail to distinguish between a direct centre-to-surround delay and time shifts that are caused by unequal excitation of the receptive field centre and surround (Enroth-Cugell & Lennie, 1975). They suggest that the ratio of centre-to-surround delay to summation time is likely to be less than one and give their preferred range as 0–10 ms.

The data given in Table 4 agree with the suggestion of a delay:summation time ratio of less than one, with means of 0.81 (SD 0.16) in the control group, and 0.90 (SD 0.23) in the diabetic group. The results for the model fit to the pooled data also indicate a ratio of less than one. However, the absolute time scale indicated by this experiment is nearer 20–25 ms for the centre–surround delay, rather than that of less that 10 ms suggested. The previous values obtained for the centre–surround delay are in the range 15–40 ms (Enroth-Cugell & Lennie, 1975; Winters & Hamasaki, 1976).

There are relatively few studies to date investigating the temporal aspects of function in diabetes. Flicker perimetry has been performed in a group of type I diabetics without retinopathy (Lobefalo et al., 1997). The mean CFF was reduced in the diabetics and the CFF decrease correlated with the level of glycosylated haemoglobin.

Wavelength discrimination has been assessed in the range 440–540 nm in a group of diabetics without retinopathy for stimulus presentation times of 1 and 0.04 s (Kurtenbach et al., 1999). This study found that the discrimination was unaffected in the diabetics for a 1 s presentation, but significantly worsened for the shorter wavelengths at the shorter presentation time. They also found that the spectral sensitivity of the diabetic group (assessed using heterochromatic brightness matching) was greater in the short wavelength end of the spectrum for the diabetics in comparison with the normals. This latter finding was surprising and was interpreted as an absolute sensitivity loss to the achromatic matching stimulus, producing an apparent increase in sensitivity at

the short wavelengths. They concluded that the decreased wavelength discrimination at the short wavelengths found for the short stimulus time was due to a reduction in stimulus energy at the S – (L + M) opponent site, a functional disturbance consistent with the middle and inner retinal layers. Thresholds for the discriminability of desaturated colour stimuli from a reference white were measured for the cardinal directions of colour space in diabetics with and without retinopathy in comparison to normals under conditions of brief (2 ms) stimulus presentation (Scase et al., 1990). The diabetics both without and with retinopathy showed a major discrimination loss in the blue and yellow directions in comparison with the control subjects and the retinopathic subjects also showed a mild loss in the green.

Using antiphase gratings flickered at 8 Hz, Di Leo found that contrast thresholds were reduced at spatial frequencies below 12.2 cycles per degree as well as noting loss of sensitivity to static gratings from 1.1 to 9 cpd (Di Leo et al., 1992). The authors state that the findings indicate a generalised neuronal dysfunction early in diabetes, although the threshold criterion used to measure the dynamic contrast sensitivity was not reported. This could have been either the detection of flicker or motion (i.e. a transient channel) or detection of spatial structure (a sustained channel).

The ST2 temporal function and the parameters derived using the Donner and Hemila model have allowed investigation into the temporal response of the retina in diabetes. The model can be used to predict the ST2 temporal function with an excellent degree of accuracy and allows estimation of the photoreceptor summation time and centre-to-surround delay in human subjects. The values derived are in agreement with the findings of previous electrophysiological studies. Our preliminary study shows that the form of the function is altered in patients with diabetes and the results of the model fitting suggest that this is due to an alteration in centre–surround delay in the receptive field, with preserved receptor summation time. Further investigation in a larger number of patients would help to characterise these functional changes.

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