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Contrast Response Properties of Magnocellular and Parvocellular Pathways in Retinitis Pigmentosa Assessed by the Visual Evoked Potential

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Abstract

Purpose—To evaluate the contrast response of the visual system in retinitis pigmentosa (RP) under conditions designed to emphasize the parvocellular (PC) and magnocellular (MC) pathways.

Method—Visual evoked potentials (VEPs) were measured in 10 patients with RP and in 10 age-equivalent control subjects with normal visual acuity and color vision, using an array of isolated checks that were presented against a steady yellow background. The checks were modulated sinusoidally either in isoluminant chromatic contrast (5.6 Hz) to favor the chromatic PC pathway, or in luminance contrast (5.6 and 11.2 Hz) to favor the MC pathway. Response amplitude and phase at the stimulus (fundamental) frequency were derived from Fourier analysis, and contrast response functions were fit with a Michaelis-Menten equation to derive R_{max} , the maximum response amplitude, and σ , the contrast required to produce $R_{max}/2$.

Results—For the control subjects, the mean amplitude function for chromatic modulation increased approximately linearly with increasing contrast, whereas the function for luminance modulation increased sharply at low contrasts and saturated at contrasts above approximately 30% for both temporal frequencies, as expected. The patients with RP showed primarily a reduction in R_{max} with little change in σ for all testing conditions. The reduction in R_{max} was equivalent for chromatic modulation and luminance modulation at 5.6 Hz, but was substantially lower for luminance modulation at 11.2 Hz.

Conclusion—Contrast processing was impaired within both the MC and PC pathways in these patients with RP, but the degree of impairment within the MC pathway depended on temporal frequency. These VEP results are in general agreement with recent psychophysical studies of contrast sensitivity losses in patients with RP, and further, they characterize contrast processing deficits of these patients at suprathreshold levels.

Keywords

Contrast sensitivity; visual evoked potential; retinitis pigmentosa; retinal degeneration; color vision

Introduction

Individuals with retinitis pigmentosa (RP) typically exhibit night blindness, peripheral visual field depressions or scotomata, abnormalities in the electroretinogram (ERG) of both the rod and cone systems, intraretinal bone-spicule-like pigmentation, and narrowing of the retinal vessels.¹ Although the rod system appears to be the primary target of the disease, the foveal cone system can also be affected. Histologic studies of the maculae of RP donor eyes have shown evidence of a reduced spatial density of the foveal cones.^{2–4} Clinically, foveal dysfunction in RP is manifested as a reduced visual acuity.⁵ However, the exact relationship between the cone photoreceptor degeneration and the foveal dysfunction that tends to occur in RP is presently unclear. In particular, the neural remodeling that can take place in retinal degenerations⁶ makes it difficult to predict *a priori* the exact nature of the visual deficits that might be experienced by patients with RP.

One of the characteristics of foveal vision loss observed frequently in patients with RP is a loss of contrast sensitivity across a range of spatial frequencies.^{7–13} The explanation for the reduction in contrast sensitivity is presently uncertain. However, the ability to discern spatiotemporal variations in luminance forms the basis for perception of the visual environment, and reduced contrast sensitivity can lead to difficulty in the performance of tasks of everyday life.^{14–16} Therefore, it is of interest to better define the nature of the contrast processing deficits in RP.

Contrast processing within the normal visual system is thought to be mediated by two processing streams with different response properties: the magnocellular (MC) and parvocellular (PC) streams. At the level of the retina and lateral geniculate nucleus, the MC stream of the primate visual system has a high contrast gain and approaches saturation at relatively low contrasts, whereas the PC stream has a low contrast gain and a more linear contrast response function.¹⁷ It is thought that the MC pathway is involved in the detection and discrimination of briefly presented, achromatic patterns of low contrast, whereas the PC pathway is presumed to be responsible for visual resolution and chromatic processing.¹⁸

Whether the foveal cone photoreceptor degeneration that can occur in RP has a preferential effect on MC- or PC-pathway function is presently unclear. The initial studies of contrast sensitivity deficits in patients with RP^{7–13} were not intended to determine whether contrast sensitivity was mediated by the MC or PC pathway. Recently, the question of the relative impact of RP on contrast processing within the MC and PC pathways was examined psychophysically using “steady-pedestal” and “pulsed-pedestal” paradigms^{19,20} designed to target the MC and PC pathways, respectively. In a test of spatial contrast sensitivity,²¹ the deficits of patients with RP were equivalent for the two paradigms, indicating a similar degree of sensitivity loss for the MC and PC pathways. However, in a test of contrast discrimination,²² patients with RP showed evidence for a greater threshold elevation for the steady-pedestal paradigm, favoring the MC pathway. Further, temporal integration for contrast discrimination was prolonged in patients with RP under conditions that emphasized the MC pathway, but not under conditions that favored the PC pathway.²³ Consequently, there is some inconsistency as to the effect of RP on contrast processing within the MC and PC pathways.

Further, the suprathreshold contrast response of patients with RP has not been characterized. As has been noted previously in patients with amblyopia,²⁴ deficits in contrast perception that are apparent at threshold may not extend to suprathreshold levels. The reduced contrast sensitivity that has been observed previously in patients with RP^{7–13} could result either from a change in response scaling (ie, reduced response amplitude at all contrasts) or from a change in effective contrast (ie, shift of the response function to higher contrasts). Information about the nature of the contrast response function in RP would be helpful in understanding the visual

constraints that are imposed by this disorder. For example, a reduction in effective contrast would indicate that contrast enhancement techniques might be beneficial in improving the visual performance of patients with RP, whereas a change in response scaling would indicate that these techniques would likely be of minimal value.

The present study employed the visual evoked potential (VEP) to evaluate the contrast response properties of the MC and PC pathways in RP, and to examine further whether there is evidence for a preferential response deficit within these contrast processing pathways. The testing paradigms were based on those of Zemon and colleagues (Zemon V, et al. *IOVS* 1991;32:ARVO Abstract 1795) and have been applied previously to patients with glaucoma²⁵ and schizophrenia.²⁶ In the present study, the contrast response functions of a group of patients with RP were obtained to luminance modulation, in order to bias performance toward the MC pathway, and to isoluminant chromatic modulation, with stimuli equated for isoluminance individually for each subject, in order to favor the chromatic PC pathway. Given the psychophysical evidence for prolonged temporal integration in patients with RP under conditions favoring the MC pathway,²³ VEP responses to luminance modulation were obtained at two temporal frequencies in order to determine whether there is a parallel deficit in their VEP response.

Method

Subjects

Ten patients with typical RP (age range, 27 to 62 years) participated in the study. Their characteristics are given in Table 1. In this table, patients are listed in order of decreasing visual acuity (increasing log MAR). All patients had better than 20/50 best-corrected visual acuity in the tested eye (which was chosen at random), and had minimal or no posterior subcapsular cataracts. Two patients (Nos. 7 and 10) had mild epiretinal macular membranes in the tested eye, and one patient (No. 5) had a perifoveal, atrophic-appearing, annular macular lesion, but no patient had clinically observable macular cysts.

The VEP results from the patients with RP were compared with those of 10 visually normal, age-equivalent control subjects (age range, 24 to 59 years). The control subjects had best-corrected visual acuities of 20/20 or better in the tested eye, normal color vision, clear ocular media, and normal-appearing fundi on ophthalmologic examination. The study adhered to the tenets of the Declaration of Helsinki, and it was approved by an institutional review board of the University of Illinois at Chicago. Informed consent was obtained from all subjects after the nature and possible consequences of the study had been explained to them. All participants were remunerated for their participation.

Stimuli and Procedure

The stimuli were generated using a VENUS system (Neuroscientific Corp., Farmingdale, NY) and were presented on an RGB video display (Diamond Pro 900U; NEC-Mitsubishi Electric Visual Systems Corp., Tokyo, Japan) at a frame rate of 90 Hz (noninterlaced). The VENUS system incorporates a linearization procedure that compensates for nonlinearities in the gamma functions of each gun separately. In the present experiments, only the red and green guns were used; the blue gun was disconnected for all conditions.

VEP responses were recorded using an electrode positioned 3 cm above theinion (O_z) that was referenced to a site on the vertex of the head (C_z), with the ground electrode placed on the midline equidistant from O_z and C_z . The EEG was amplified (20K, 0.5 to 100 Hz), digitized, and stored in a computer. Subjects viewed the display monocularly through the best optical correction in a trial frame at a test distance of 114 cm, with the untested eye occluded. Testing

was begun after 2 min of adaptation to the display mean luminance. Each condition was run 5 times, and subjects were given a brief rest between conditions.

The stimuli used to obtain the contrast response functions were based on those used previously.^{25,26} Swept contrast VEPs were elicited in response to the modulation of an array of 32 X 32 isolated checks (each check subtended 11.4 min of visual angle) presented against a steady yellow surround that had a luminance of 55 cd/m², with each check separated from its neighbors by one check width. The square stimulus array subtended 12.2 deg (6.1 deg horizontally and vertically from a central fixation mark). The checks were modulated sinusoidally in appearance-disappearance mode either in chromatic contrast at 5.6 Hz (isoluminant red checks) to favor the chromatic PC pathway, or in luminance contrast (yellow checks that were either all of positive contrast or all of negative contrast), at both 5.6 Hz and 11.2 Hz, to favor the MC pathway. The total sweep duration was 7.5 s for all conditions. For both luminance modulation and chromatic modulation, there were 7 contrast steps (including an initial step of 0% contrast). At 5.6 Hz, responses to 6 stimulus cycles were obtained for each step; at 11.2 Hz, responses to 12 stimulus cycles were obtained.

Luminance contrast (C_W) was defined as Weber contrast:

$$C_W = (L_t - L_s) / L_s \quad (1)$$

where L_t is the maximum luminance of the checks and L_s is the surround luminance. The definition of chromatic contrast was based on Eq. 1 applied to the modulation of the red gun. The green gun was always modulated in counterphase with the red gun, at a depth of modulation used to yield isoluminance for a given subject. Isoluminance was derived by varying the depth of modulation of the green gun (G) in counterphase to the red gun (R) while keeping the red gun (R) contrast fixed at 100%.

Isoluminance was evaluated first. To obtain the isoluminance measure, the temporal frequency was 11.2 Hz, and the total sweep duration was 11.8 s. There were 11 discrete steps of G:R ratios, with responses to 12 stimulus cycles obtained at each step. Isoluminance was defined as the G:R ratio that produced a minimum in the VEP amplitude and/or an inflection in the phase function. This ratio was used to obtain the chromatic contrast response functions, which were measured next. Luminance contrast response functions were then measured, first at 11.2 and then at 5.6 Hz. Responses at each temporal frequency were obtained first with increasing negative contrast and then with increasing positive contrast.

Finally, VEP responses were recorded to luminance modulation of isolated checks that ranged in total number from 4 to 128 (check width, 90.5 to 2.8 min). The purpose was to verify that abnormalities in the swept-contrast VEPs of the patients with RP were not due to the use of an inappropriate check size. The luminance of each check was modulated sinusoidally in appearance/disappearance mode relative to the surround luminance (85 cd/m² for this test condition) at a temporal frequency of 11.2 Hz. The total sweep duration was 8.5 s, and there were 8 discrete steps of stimulus sizes, including an initial step that consisted of a uniform field. The check contrast (C_W) was fixed at 60%, and responses to checks of positive contrast were measured first, followed by responses to checks of negative contrast.

Prior to the VEP testing, the visual acuity of each subject was assessed with a transilluminated Distance Visual Acuity Test (Lighthouse International, New York, NY), and letter contrast sensitivity was measured with a Pelli-Robson Contrast Sensitivity Chart (Haag-Streit, Koeniz, Switzerland), using procedures described previously.²⁹ The visual fields of the patients with RP were obtained with a Goldmann perimeter in a separate session. All patients with RP had central visual field diameters for the V/4e target that were greater than the 12.2-deg extent of

the stimulus array, with the exception of Patient No. 6, whose central visual field diameter for this Goldmann target was approximately 8 deg.

Data analysis

Response amplitude and phase at the stimulus fundamental frequency were derived from a discrete Fourier transform of the data for each of the 5 repetitions for each condition for each subject. These five individual amplitudes and phases were then vector averaged to obtain a mean amplitude and phase for each condition for each subject. Next, the mean amplitudes and phases of the patient and control groups were obtained by vector averaging. The mean contrast response functions of the patient and control groups were then fit with a Michaelis-Menten function of the form:

$$R = \frac{R_{\max} C W^n}{C W^n + \sigma^n}, \quad (2)$$

where R is response amplitude, $R_{\max}$ represents the maximum response amplitude, σ represents the contrast that produces $R_{\max}/2$, and n is a dimensionless slope parameter. Data were fit using the Marquardt-Levenberg algorithm of the nonlinear curve-fitting routine provided by SigmaPlot (Systat Software, Inc., Point Richmond, CA). For the control subjects, all three parameters were derived from the fit. The derived values of n for the control subjects were then used in fitting the mean data of the patients with RP.

Results

The relationship between check width and VEP amplitude is illustrated in Fig. 1. This figure presents mean VEP amplitude as a function of check width for the patients with RP (diamonds) and the control subjects (circles), with the results for increments (open symbols) and decrements (filled symbols) plotted separately. The mean spatial response functions for the control subjects showed a peak at an intermediate check width, as expected.³⁰ The mean spatial response functions for the patients with RP showed a peak at the same check width as for the control subjects, but the responses of the RP patients were reduced compared to normal. The check width that was used to obtain the contrast response functions was 11.4 min, which is just to the left (smaller checks) of the peak of the spatial response function. There were no consistent differences between the results for increment and decrement checks for either the patients with RP or the control subjects in this or any of the other testing paradigms. Consequently, the results for increment and decrement checks were averaged in the following analyses.

Fig. 2 illustrates the mean contrast response functions for isoluminant chromatic modulation. The mean contrast response function of the control subjects (open circles) did not begin to rise until an intermediate level of chromatic contrast, and then it continued to rise at high contrasts, consistent with previous findings.²⁵ The mean contrast response function for the patients with RP (filled circles) showed the same general pattern as that of the control subjects, but the patients' responses were reduced below normal at the higher contrast levels. As indicated in Table 2, the primary difference between the contrast response functions of the two groups was a decrease in $R_{\max}$ for the patients with RP, with little change in σ . The value of $R_{\max}$ for the patients was 0.39 times that of the control subjects.

The mean contrast response functions for luminance modulation at the two temporal frequencies are shown in Fig. 3. In comparison to the results for chromatic modulation, the mean contrast response functions of the control subjects for luminance modulation (open symbols) rose steeply at low contrasts and saturated at a contrast of approximately 30%, as

expected.²⁵ Further, the results for the control subjects were equivalent at both temporal frequencies. The contrast response functions of the patients with RP were similar in shape to those of the control subjects, but amplitudes were reduced at both temporal frequencies, more so at the higher temporal frequency. At 5.6 Hz, the R_{max} value for the patients with RP was 0.46 times that of the control subjects (Table 2), which is approximately equal to the R_{max} ratio observed for chromatic contrast. However, the R_{max} ratio was 0.11 at the temporal frequency of 11.2 Hz. Thus, the patients with RP showed a disproportionate reduction in mean VEP amplitude for luminance modulation at the higher temporal frequency.

The fundamental response phases for luminance modulation at 5.6 and 11.2 Hz are presented in Fig. 4. The control subjects (open symbols) showed a phase advance with increasing contrast at both temporal frequencies, consistent with a previous report.²⁵ The patients with RP (filled symbols) showed a similar phase advance with increasing contrast for both temporal frequencies. By a repeated measures analysis of variance, the patients with RP showed a significant phase lag at 11.2 Hz compared to the control subjects ($F = 13.63$, $P < 0.01$), with no significant group by contrast interaction ($F = 0.85$, $P = 0.45$). There was also a slight phase lag at 5.6 Hz for the patients with RP, but the difference from the control group was not statistically significant ($F = 0.65$, $P = 0.43$).

Discussion

This study investigated the contrast response properties of the VEP in a group of patients with RP within the framework of the MC and PC pathways. The spatial characteristics of the VEP response to luminance modulation were similar for the patients with RP and the control subjects (Fig. 1). The mean spatial response functions of the two groups peaked at the same angular check size, but the response amplitude of the patients was reduced overall. A similar finding of a reduced amplitude but equivalent spatial scaling was reported for the pattern-reversal VEP in patients with RP.³¹

There were no systematic differences between the VEP responses of the subjects to luminance increments and decrements (Fig. 1), although asymmetries for increments and decrements have been reported previously for the VEP of visually normal subjects.³⁰ However, the stimulus conditions used here were likely not optimal for observing such asymmetries. Amplitude differences in the response to increments and decrements were reported to be most evident at intermediate temporal frequencies (approximately 3 to 10 Hz), and with large (> 100 min) and small (< 10 min) check sizes.³⁰ In comparison, the temporal frequency used for the spatial response functions in the present study was 11.2 Hz, and the check size used for the contrast response functions was 11.4 min.

The patients' contrast response functions showed primarily a reduction in R_{max} for both chromatic and luminance modulation (Figs. 2 and 3). Further, the ratios of the R_{max} values of the patients vs. controls were approximately equivalent for chromatic modulation and luminance modulation at a temporal frequency of 5.6 Hz (Table 2). This result is consistent with a previous psychophysical study of spatial contrast sensitivity in patients with RP as measured with steady-pedestal and pulsed-pedestal paradigms that targeted the MC and PC pathways, respectively.²¹ In that study, patients' deficits in contrast sensitivity were equivalent for both paradigms. The VEP results indicate further that the decreased psychophysical contrast sensitivity of the patients with RP is likely the result of a change in response scaling rather than a change in contrast scaling. Still to be determined is why patients with RP showed equivalent losses of spatial contrast sensitivity under conditions that targeted the two pathways,²¹ but showed a greater deficit under conditions that favored the MC pathway when contrast discrimination was evaluated,²² using equivalent stimulus durations.

The fact that the patients with RP showed a decreased R_{max} for chromatic modulation, which emphasizes the PC pathway, is in agreement with a previous psychophysical study of contrast discrimination in patients with RP.²² In that study, increment threshold functions for contrast discrimination within the pulsed-pedestal paradigm, which favors the PC pathway, were fit with a quantitative model (Eq. 3 of Ref. 19) to derive the characteristics of the contrast response function. The discrimination thresholds of the patients with RP were better fit by a decreased R_{max} than by an increased σ . The decreased R_{max} for chromatic modulation is also in agreement with a previous psychophysical study that estimated the contrast response of the cone system in patients with RP using a probe-on-flash technique,³² which favors the PC pathway. A psychophysical analysis of the contrast response function for the MC pathway has not been made in patients with RP, because a pedestal-delta-pedestal paradigm is required,¹⁹ and this is difficult even for visually normal subjects to perform.

The patients with RP in the present study showed a greater VEP response deficit at the higher temporal frequency of luminance modulation (Fig. 3). This finding is consistent with the increased time constant of temporal integration that was observed psychophysically in patients with RP under conditions that emphasized the MC pathway.²³ That is, an increased time constant of temporal integration corresponds to a lower corner frequency of the temporal response function (i.e., frequency at which the sensitivity has decreased by 3 dB).³³

It seems unlikely that this VEP deficit at such a relatively low temporal frequency is due to a response abnormality at the level of the cone photoreceptors, given that cone photoreceptors normally can respond at temporal frequencies up to 100 Hz.³⁴ It is also unlikely that the substantially greater amplitude reduction in the VEP at 11.2 Hz represents pathology occurring within the early retina, because the focal ERGs of patients with RP show only a small amplitude loss compared to normal at this temporal frequency.^{35,36} Instead, it is more likely that the temporal frequency deficits observed psychophysically and in the VEP response represent the impact of cone photoreceptor degeneration on contrast processing within the MC pathway. For example, there is evidence that the spatiotemporal integration properties of cortical cells are related to the level of synaptic background activity.³⁷ A reduced synaptic input, owing to a loss of cone photoreceptor signals, would decrease input conductance, which in turn would increase the effective time constant of the cells and act as a low-pass temporal filter.³⁷

The control subjects showed a relative phase advance with increasing contrast at both temporal frequencies of luminance modulation (Fig. 4), as reported previously.²⁵ This was also the case for the patients with RP. This phase advance is consistent with a nonlinear gain control mechanism, as has been described for neurons within the MC pathway.³⁸ Further, there was a phase advance between the VEP responses to temporal frequencies of 5.6 and 11.2 Hz for both groups. A similar phase advance between these two temporal frequencies is observed in the full-field flicker ERG,³⁹ which suggests that it originates at an early retinal level. It is unlikely, however, that the phase lag observed at 11.2 Hz in the VEP of the patients with RP compared to normal is due to an abnormality at the same retinal level, because the phase of the focal ERG is normal in patients with RP whose visual acuities are within the range of those included in the present study.^{35,36} The relative phase lag of the patients' luminance-modulation VEP at 11.2 Hz more likely represents a difference in VEP response latency, which would produce the type of frequency-dependent phase shift observed in Fig. 4.

Given the overlap of response properties that may exist between the MC and PC processing streams,⁴⁰ the VEP procedures used in this study may not generate responses that are entirely selective for the MC and PC pathways. However, the contrast response functions of the control subjects for chromatic and luminance modulation had the characteristics expected from electrophysiological studies of the MC and PC pathways,¹⁷ and the functions corresponded to VEP responses reported previously under equivalent testing conditions.²⁵ In addition, there

was consistency between the results obtained for the VEP and those obtained psychophysically in patients with RP, as discussed above.

In summary, the patients with RP in this study showed VEP contrast response functions for chromatic and luminance modulation that were characterized predominantly by a decrease in R_{max} , which corresponds to an overall reduction in response amplitude. The decrease in R_{max} was essentially equivalent for chromatic and luminance modulation at a temporal frequency of 5.6 Hz, indicating equivalent response deficits for the PC and MC pathway. However, there was a substantial reduction in VEP amplitude at a temporal frequency of 11.2 Hz under the same adapting conditions, indicating a marked impairment of function within the MC pathway. The results are in agreement with a previous conclusion that the relative functional impairment within MC and PC processing streams in patients with RP depends on the nature of the stimuli and testing protocols that are used to evaluate these functional deficits.

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Fig 1.

Mean VEP fundamental amplitude as a function of the angular width of the isolated checks, on semi-log coordinates, for the patients with RP (diamonds) and control subjects (circles), for increment (open symbols) and decrement (filled symbols) checks. Error bars represent ± 1 standard error of the mean (sem).

Chromatic modulation

Fig 2. Mean VEP contrast response functions for chromatic modulation, on semi-log coordinates, for the patients with RP (filled circles) and control subjects (open circles). Error bars represent ± 1 sem. Curves represent the least-squares best-fits of Eq. 2.

Luminance modulation

Fig 3. Mean VEP contrast response functions for luminance modulation at 5.6 Hz (squares) and 11.2 Hz (triangles), on semi-log coordinates, for the patients with RP (filled symbols) and control subjects (open symbols). Error bars represent ± 1 sem. The curves represent the least-squares best-fits of Eq. 2.

Fig 4. Mean VEP fundamental phase as a function of luminance modulation at 5.6 Hz (squares) and 11.2 Hz (triangles), on semi-log coordinates, for the patients with RP (filled symbols) and control subjects (open symbols). Error bars represent ± 1 sem. Solid lines represent least-squares regression lines fit to the mean phases for each group.

Table 1

Characteristics of the patients with RP.

Patient No.	Sex	Age (Years)	Log MAR	Log CS	Log VFA [*]	PSC Grade [†]	Fundus (Macula)	Genetic Type [‡]
1	M	43	0.01	1.35	3.97	0.0	Normal	Iso
2	M	29	0.01	1.68	3.58	0.0	Normal	Iso
3	M	61	0.05	1.65	3.77	0.0	Normal	Iso
4	F	47	0.09	1.65	3.71	0.0	Normal	AD
5	F	62	0.15	1.55	1.55	0.25	Annular Lesion	Iso
6	F	39	0.18	1.50	1.64	0.0	Normal	Iso
7	F	35	0.20	1.48	2.61	0.0	Epiretinal Membrane	Iso
8	F	50	0.25	1.65	2.41	0.0	Normal	Iso
9	F	46	0.28	1.23	1.90	0.0	Normal	AR
10	M	27	0.32	1.23	2.27	0.0	Epiretinal Membrane	Unc
Control Range		24 to 59	−25 to −0.01	1.65 to 1.95	4.00 to 4.14			

^{*} Visual field area (log sq. deg) using a Goldmann II/4e target; the control range is from Ref.27.

[†] Based on the grading scale of Ref.28.

[‡] AD - autosomal dominant; AR - autosomal recessive; Iso – isolated, no other known affected family member; Unc - uncertain (patient was adopted).

Table 2

Parameters for the least-squares best fits of Eq. 2 to the mean contrast response functions of the control subjects and patients with RP.

	Chromatic Modulation (5.6 Hz)		Luminance Modulation (5.6 Hz)		Luminance Modulation (11.2 Hz)	
	Control	RP	Control	RP	Control	RP
R_{max} (μV)	10.25	3.96	4.46	2.07	4.25	0.49
σ (% contrast)	44.12	50.42	11.86	16.26	13.44	7.39
n^*	2.98	2.98	2.62	2.62	3.49	3.49

* The same value of n was used for the patients with RP and control subjects.