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PERFORMANCE OF GENETICALLY-COLORBLIND INDIVIDUALS ON A RAPID DARK ADAPTATION TEST BASED ON THE PURKINJE SHIFT¹

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Summary.—An experiment was conducted to determine whether or not genetic colorblindness would limit performance on a rapid dark adaptation test (RDAT) which is based on the Purkinje shift in retinal sensitivity to lower wavelengths of light energy under mesopic/scotopic conditions of illumination. No differences in RDAT performance between age-equivalent colorblind and non-colorblind subjects was observed.

Nightblindness has been established as a symptom of vitamin A deficiency. Recently, evidence for a vitamin A-resistant, zinc-responsive impairment of dark adaptation has been presented (Morrison, et al., 1979; McClain, et al., 1979; Warth, et al., 1981). Moreover, there is reason to speculate that deficiencies of vitamin E and taurine might produce retinal injury that would be manifest as reduced capacity for night vision. Thus, determination of dark adaptation defects represents a versatile tool for nutritional assessment.

The conventional procedure for assessing dark adaptation uses a Goldmann-Weekers darkadaptometer and is tedious, time-consuming and complex. In 1977, Thornton proposed a novel rapid dark-adaptation test (RDAT) based on the Purkinje shift, that phenomenon which makes the retina more sensitive to light of shorter-wavelengths during adaptation to twilight and night vision. Objects that are blue or purple in daylight become visible earlier and appear more brilliant than red objects, albeit in tones of gray, to the dark-adapting eye. The sequential separation of white and then blue discs from red discs on a black background under a standard very dim illumination is the basis for the Thornton procedure. Vinton and Russell (1981) validated a modification of the Thornton procedure against the formal darkadaptometry standard of the Goldmann-Weekers test, and demonstrated a high sensitivity and specificity for RDAT performance in diagnosing vitamin A deficiency.

The purpose of the present study was to determine if genetic colorblindness in which the retinal cones discriminate poorly among certain hues in daylight vision would affect performance on a night vision test based on sensitivity to different wavelengths of light.

METHOD

Subjects

The subjects were unpaid, male volunteers from the university community

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of M.I.T. They were recruited on the basis of their self-perception of their color discrimination ability, as either colorblind or not colorblind. Twenty subjects were enrolled into each cohort, based on the objective measurement of their color discrimination.

Color Discrimination Test

Apparatus.—The color discrimination test was the Standard Farnsworth-Munsell 100-Hue test (Farnsworth, 1957), involving the correct spectral arrangement of 85 special colored discs mounted in plastic caps with hues ranging from red through violet, under standard illumination of a 100-watt bulb on an easil lamp (Macbeth) filtered through a Roundel filter (Corning).

Test procedure.—After a full explanation of the test, subjects donned plastic gloves to protect the surfaces of the discs and were presented the four trays of 21 discs, each with their contents distributed color-side up on the surface of the table at the base of the lamp. Subjects were given unlimited time to arrange the discs within each tray.

Scoring and data analysis.—The number of errors was determined for each tray and represented graphically on the Munsell scoring sheet. A numerical score was assigned as per instruction manual (Farnsworth, 1957), and for all subjects with an error score of greater than 100, a pattern of analysis was made to classify the color-discrimination defect into a spectral-genetic sub-type: protan ("red blindness"); deutan ("green blindness"); or tritan ("blue blindness").2

Rapid Dark Adaptation Test

Apparatus.—The RDAT was a minor modification of the procedure described by Thornton (1977).3 Standard illumination is provided by a 7.5-w bulb shielded by a 1% transmittance neutral density filter mounted in a standard darkroom lamp fixture (Yankee Photo Co., Culver City, CA). Eighteen, non-glossy plastic poker chips-5 white, 6 blue and 7 red-are distributed on a black, cloth surface 122 cm below the filter. Timing is performed with a laboratory timer and an illuminated digital stopwatch. The test is administered in a light-tight darkroom. In our modification,8 retinal bleaching was performed with a 15- × 6-in. fluorescent X-ray view-box (HealthCo., Boston, MA).

Test procedure.—The tester and subject sat opposite one another. The room was illuminated with red light for the initial 5 min. of the testing session while the procedure was explained. Immediately thereafter, the red light was extinguished and the view-box illuminated. The subject stared at the center

²As a diagnostic instrument, the Farnsworth-Munsell 100 Hue test cannot discriminate the true dichromats (protanopes, deuteranopes, and tritanopes) from anomalous trichromats (protanomalous, deuteranomalous, and tritanomalous).

²N. W. Solomons, A. M. de Guerrero, N. Schlossman, L. Mejia, & O. Pineda, Standardization of a rapid dark adaptation test for nutritional diagnosis. (Submitted for publication)

of the view-box from a distance of 45 cm for 2 min. During the trial, the only light source was the overhead darkroom lamp. Subjects were instructed to separate sequentially the five white chips, and then the six blue chips; any errors in selection were corrected by the tester as they occurred. The time to identify each color was recorded in seconds. The testing session included four consecutive trials, each preceded by a 2-min. retinal-bleaching period with the X-ray view-box.

Scoring and data analysis.—The first time-trial of the session was considered to be a warm-up and the data excluded. The definitive scoring was calculated individually for white and blue chips using the mean time in seconds of the two most rapid post-warm-up trials.³

RESULTS

Each of the 40 subjects' initial self-estimation of his own color discrimination capacity was verified on the Farnsworth-Munsell 100 Hue test. The data for the colorblind subjects are shown in Table 1 and those for the non-colorblind

TABLE 1

RESULTS OF FARNSWORTH-MUNSELL 100 HUE TEST AND THORNTON'S RAPID

DARK ADAPTATION TEST IN COLORBLIND INDIVIDUALS

	Didd IDii Infox 1231 in Colonbino Indiabatic						
Subject No.	Age	FM 100 Hue Error Score	Pattern of Color Defect	RDAT* for White Chips (sec.)	RDAT* for Blue Chips (sec.)		
01	19	231	Protan	37	126		
02	22	153	Deutan	110	463		
03	30	145	Deutan	54	154		
04	27	144	Deutan	70	172		
05	30	221	Deutan	90	186		
06	23	179	Deutan	33	148		
07	24	121	Protan	68	198		
08	20	169	Deutan	29	90		
09	21	223	Deutan	54	138		
10	30	260	Deutan	47	130		
11	19	111	Deutan	47	93		
12	24	203	Deutan	33	123		
13	22	167	Deutan	62	188		
14	22	133	Deutan	48	118		
15	22	197	Deutan	32	95		
16	22	103	Deutan	85	156		
17	21	125	Protan	37	93		
18	20	153	Protan	83	161		
19	19	113	Deutan	39	135		
20	28	255	Protan	31	121		
Arithmetic M		170.3		54.4	154.4		
SD		48.8		23.1	79.6		
Geometric M				50.3	142.8		

^{*}Two of three most rapid times for the post-warm-up trials (arithmetic mean).

subjects in Table 2. The mean age for the colorblind and non-colorblind subjects (\pm SD) was 23 yr. \pm 4 mo. and 20 yr. \pm 2 mo., respectively. The arithmetic mean for the Farnsworth-Munsell 100 Hue test for the colorblind subjects, 170.3 \pm 48.8 (range: 103—260), was distinctly different from the mean of the non-colorblind cohort, 30.4 \pm 21.6 (range: 0—74) as would be expected by the respective definitions of color discrimination acuity (Farnsworth, 1957) (Fig. 1). Five of the colorblind subjects were classified as protans, and 15 were deutans.

TABLE 2

RESULTS OF FARNSWORTH-MUNSELL 100 HUE TEST AND THORNTON'S RAPID DARK ADAPTATION TEST IN NON-COLORBLIND INDIVIDUALS

Subject No.	Age	FM 100 Hue Error Score	RDAT* for White Chips (sec.)	RDAT* for Blue Chips (sec.)
001	20	19	31	131
002	20	00	36	143
003	18	31	37	128
004	18	20	44	138
005	22	12	73	346
006	18	19	3 3	120
007	19	72	45	113
008	19	16	39	108
009	18	74	31	82
010	21	62	73	207
011	19	19	56	127
012	19	34	30	94
013	25	00	46	123
014	22	34	42	133
015	19	31	71	174
016	20	31	41	140
017	19	33	67	173
018	20	34	35	110
019	19	59	30	103
020	24	08	54	170
Arithmetic M		30.4	45.7	143.1
SD		21.6	14.9	56.4
Geometric M			43.6	135.7

^{*}Two of three most rapid times for the post-warm-up trials.

The arithmetic mean times (\pm SD) to separate the white chips were 54.4 \pm 23.1 and 45.7 \pm 14.9 sec., respectively, in the colorblind and non-colorblind subjects. The geometric means were 50.3 and 43.6 sec. The arithmetic mean scores for separation of the blue chips were 154.4 \pm 79.6 for the colorblind subjects and 143.1 \pm 56.4 for the non-colorblind individuals; the respective

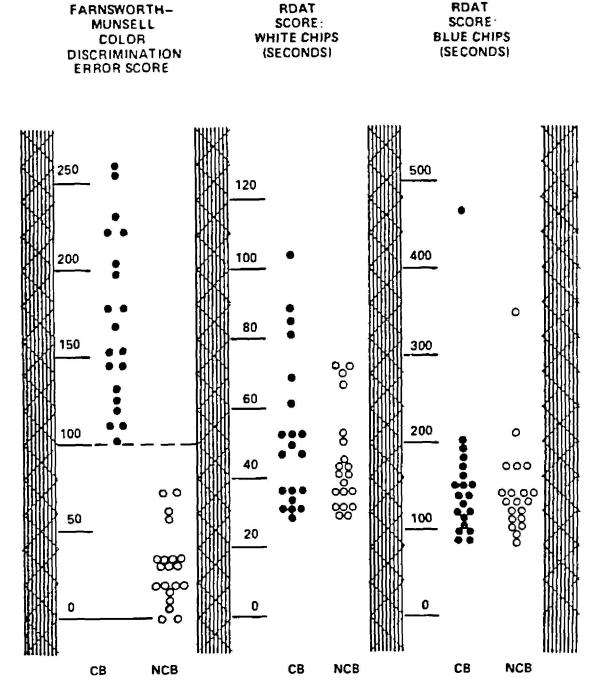


FIG. 1. The individual error scores on the Farnsworth-Munsell 100 Hue test and the individual separation times (mean of the best two out of three valid trials) for the removal of white and blue chips for the Thornton RDAT test for the 20 colorblind (•) and the 20 non-colorblind (•) subjects

geometric means were 142.8 and 135.7 sec. Within the colorblind group, the geometric means for the time to separate the blue chips were 135.2 sec. for the 5 protans and 145.0 sec. for the 15 deutans.

The geometric mean for these two cohorts studied in Cambridge, MA were closely similar to the 148 sec. for the blue-chip score from 100 healthy adults studied with an identical procedure in Guatemala.³ The range of performance for the blue-chip-separation phase of the RDAT in Guatemala was 70 to 426 sec. Two of the present subjects, one from each cohort, had relatively prolonged scores of 346 and 463 sec. The remaining subjects had a

blue-chip score of less than 240 sec. (Fig. 1). Over-all in the present study, no statistically significant difference in RDAT performance was detected between colorblind and non-colorblind subjects.

DISCUSSION

While colorblindness involves the retinal cones, night vision is the domain of the retinal rods. The degree of differential participation by the two types of retinal photoreceptor cells under the condition of illumination specified by Thornton (1977) for the RDAT has not been resolved. Thornton (1977) believed it to be a cone-adaptation phenomenon; Vinton and Russell (1981) feel it represents a rod function, as it responded to vitamin A deficiency, and the RDAT scores correlated with the final dark-adapted thresholds. Such a distinction is important for colorblind individuals who show hue-dependent changes in luminosity which differ from normals (Hecht & Shlaer, 1936; Collins, 1959). Moreover, when foveal adaptation (adaptation exclusively of the cones) has been examined in colorblind individuals with different hues, differences in maximal luminosity perception have been observed (Chapanis, 1946a, 1946b, 1947; Hecht & Hsia, 1947). On the other hand, these same studies showed final dark-adapted thresholds of the rods with white light and various monochromatic hues to be equivalent in color defectives and subjects with normal color discrimination. Finally, the peripheral retina (parafoveal region), composed predominantly of rods, also shows a luminosity shift with increased perception of objects with shorter wavelengths (Pirenne, 1944) despite the fact that all hues are perceived as "colorless" (gray) in scotopic conditions. Given the hue-dependent performance of both cone- and rod-adaptation, it was of practical interest to us to determine whether or not healthy, genetically colorblind individuals would have an equivalent performance to that of color-discriminating controls on the Thornton RDAT, a physiological test expressedly based on the Purkinje shift in retinal sensitivity during dark adaptation.

In our sample of 20 colorblind individuals and 20 normal controls, not only the group means of performance-times on the RDAT but also their distribution was equivalent for the separation of white and blue chips as shown graphically in the figure. There are two potential caveats, however. (1) There was a non-statistically significant tendency toward the ranking of the time-to-blue score means in the order protans > normals > deutans. It is conceivable that such an effect—suggestive of cone participation—might achieve significance if large numbers of subjects were studied. (2) Insofar as the target objects to be detected and separated in the second phase of the Thornton RDAT procedure are blue, it is unfortunate that no subjects with tritanopia or tritanomaly—with the defect in the yellow-blue axis—were

included. This is not unexpected, however, since this pattern of color-discrimination abnormalities is exceedingly rare as a genetic variant, but we are left with a margin of doubt as to whether or not tritans would have RDAT performances equivalent to those of protans, deutans, and trichromats.

The comparability of the mean performance in our modification of the RDAT procedure in 100 healthy subjects in one laboratory (Guatemala City) and 40 healthy subjects in another (Cambridge, MA) increases our confidence in the generalizability of our modification of the Thornton RDAT. Two individuals in the present series—one in each cohort—were extreme outliers with respect to separation times for the blue chips. Since no nutritional indices nor ophthalmological examination were performed, however, no precise explanation for the cause of these extremely prolonged response-times can be offered.

Chapanis (1947) used the differential pattern of loveal and peripheral retinal responses to dark adaptation with lights of different hues in studies of colorblind subjects to argue for the strict dissociation of rods and cones. It is tempting to invert that argument here to suggest that the conditions of illumination specified by Thornton (1977) for his RDAT examines dominantly scotopic phenomena, albeit within the first 2 to 3 min. of dark adaptation. As noted, the inclusion of tritans in our series would have provided a more convincing test of this hypothesis. In practical terms, however, we can conclude that the Purkinje shift-based rapid dark adaptation test procedure proposed by Thornton (1977) and validated by Vinton and Russell (1981) is as applicable to most of the genetically-colorblind population as it is to individuals with normal color discrimination as a screening test for nutritional deficiency.

REFERENCES

- CHAPANIS, A. The dark adaptation of the color anomalous. Federation Proceedings, 1946, 16, 5. (a)
- CHAPANIS, A. The dark adaptation of the color anomalous. American Journal of Physiology, 1946, 146, 689-701. (b)
- CHAPANIS, A. The dark adaptation of the color anomalous measured with lights of different hues. Journal of General Physiology, 1947, 30, 423-437.
- COLLINS, W. E. The effect of deuteranomaly and deuteranopia upon the foveal luminosity curve. Journal of Psychology, 1959, 48, 285-297.
- FARNSWORTH, D. Manual for the Farnsworth-Munsell 100-Hue Test for the examination of color discrimination. Baltimore: Munsell Color Co., 1957.
- FREY, R. G., GORESCH, J., HEILIG, P., & THALER, A. Dark adaptation in achromats (mathematical analysis). Albrecht von Graefe's Archiv für Klinische und Experimentelle Ophthalmologie, 1975, 196, 299-302.
- HECHT, S., & HSIA, Y. Luminosity losses in the spectrum for dichromats. Journal of General Physiology, 1947, 31, 141-152.
- HECHT, S., & SHLAER, S. Color vision of dichromats: wavelength discrimination, brightness distribution and color mixture. Journal of General Physiology, 1936, 20, 57-82.

- LAKOWSKI, R., & CREIGHTON, D. Foveal chromatic dark adaptation functions of red-green deficient subjects. *Modern Problems in Ophthalmology*, 1976, 17, 41-45.
- McClain, C., Van Thiel, D., Parker, S., Badzin, L., & Gilbert, H. Alterations in zinc, vitamin A and retinol-binding protein in chronic alcoholics: a possible mechanism for night blindness and hypogonadism. Alcoholism: Clinical and Experimental Research, 1979, 3, 135-141.
- MORRISON, S. A., RUSSELL, R. M., CARNEY, E. A., & OAKES, E. V. Zinc deficiency: a cause of abnormal dark adaptation in cirrhotics. *American Journal of Clinical Nutrition*, 1979, 31, 276-281.
- PIRENNE, M. D. Rods and cones, and Thomas Young's theory of colour vision. Nature, 1944, 154, 741-742.
- THORNTON, S. P. A rapid test for dark adaptation. Annals of Ophthalmology, 1977, 9, 731-734.
- VINTON, N. E., & RUSSELL, R. M. Evaluation of a rapid test for dark adaptation.

 American Journal of Clinical Nutrition, 1981, 34, 1961-1966.
- WARTH, J., PRASAD, A., ZWAS, F., & FRANK, R. Abnormal dark adaptation in sickle cell anemia. Journal of Laboratory and Clinical Medicine, 1981, 98, 189-194.

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