Contributions of the Genes Albinism *(c)* and Retinal Degeneration *(rd)* to a Strain-by-Training Procedure Interaction in Avoidance Learning

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Wahlsten, D. Contributions of the genes albinism (*c*) and retinal degeneration (*rd*) to a strain-by-training procedures interaction in avoidance learning. Behavior Genetics, 1973, 3, 303-316.

Abstract:

The effects of *c* and *rd* on jump-out and one-way avoidance learning were examined using both inbred strains and test crosses. Neither gene was found to retard either escape or avoidance performance in the jump-out task, although *rd* led to less accurate jumping. One-way performance, which required running through a small hole in a wall, was greatly disrupted in mice homozygous for *rd*; both escape latencies and learning rate were slower than those of mice with normal retinas. Two inbred strains with albinism did poorly on the one-way task, but no deleterious effects of *c* on one-way performance were detected in several test crosses. The absence of an albinism effect was not surprising, since all training was given under dim red light.

Article:

INTRODUCTION

In seeking to identify strains of mice which are typically fast or slow to acquire an anticipatory avoidance response, one is frequently struck by the inconsistent rank-orderings of the same strains studied by different investigators. Since these inbred mouse strains are generally obtained directly from a single source (The Jackson Laboratory), it is likely that procedural and task differences are the causes of reversals in relative rates of learning.

Strain differences in underlying, central processes such as motivation, memory, activity, or emotionality could readily lead to reversals of relative learning rates when tasks are employed that differ in intensity of motivation, length of trial spacing, or mode of the required response (see review by Wahlsten, 1972b). Likewise, differences in the amount of information which must be processed to learn two particular tasks may lead to strain differences only on the more complex task.

Peripheral processes may lead to interactions as well. Strain differences in, say, visual acuity should lead to divergent learning rates only on tasks demanding the utilization of visual cues.

Such differences in the peripheral aspects of the mouse visual system are the subjects of the present study. The experiments presented herein are of particular relevance to questions raised by a previous report of strain differences in avoidance learning (Wahlsten, 1972a). That report concluded with rather strong statements about the lack of relation between initial reaction to electric shock and rate of avoidance learning. Hence attribution of strain differences to genes known to affect the eyes would modify previous conclusions. One defect of interest is albinism (*c*), for the worst strain under most training conditions was the albino A/J, and albinism has been reported by several investigators (see reviews by Wilcock, 1969; Wahlsten, 1972b) to retard active avoidance learning. Another strain, CBA/J, was beset by retinal degeneration (*rd*), and it performed well on a jump-out task but poorly on a one-way task. It seemed possible that blind mice could jump out of an open box, yet not be able to find their way readily through a small hole in the wall. The following experiments were intended to evaluate the contributions of these two genes to retarded avoidance learning.

EXPERIMENT 1

The first experiment simply subjected highly inbred mouse strains which had either albinism, retinal degeneration, both, or neither to avoidance training in which either jumping out of a box (jump-out) or running through a small hole into a safe compartment (one-way) was the correct response. Several strains in addition to those previously tested on the same apparatus (Wahlsten, 1972a) were employed.

Method

Subjects

Ten male and ten female mice of each strain were obtained from The Jackson Laboratory at 7 weeks of age and were tested when 55-60 days old. The strains employed and their genotypes at the albinism and retinal degeneration loci were as follows: A/J (*c/c*, +/+), BALB/cJ (*c/c*, +/+), SWR/J (*c/c*, *rd/rd*), C3H/HeJ (+/+, *rd/rd*), CBA/J (+/+, *rd/rd*), CBA/CaJ (+/+, +/+), C57BL/CJ (+/+, +/+), and B6AF$_1$ /J (*c/*+, +/+). The F$_1$ hybrid was included because it was to be important for the second experiment. All Ss were housed five per standard plastic mouse cage with free access to water and dry food.

Apparatus

The training apparatus was a 6-inch-square black plexiglas box with a shock grid which could be escaped either by jumping the 4-inch walls onto a large masonite platform or by running through a 2-inch-square doorway into an adjacent box with a smooth floor (see Wahlsten, 1972a, for a detailed description). Jump-out training was given by closing the small door but leaving the top off, while one-way training was given with a closed top and open door. All training was given under the dim light of a single 25-w red bulb 48 inches above the grid, which provided 2.2 ft-c of incident light at the grid.

Procedure

Equal numbers (five) of males and females of each strain were trained with either the jump-out or the one-way procedure. Each trial was initiated when *S* was placed onto the grid, 5 sec following which a 180-μ amp constant-current shock commenced and remained for 30 sec or until *S* escaped. *S* was then returned to a holding cage between trials. One minute elapsed between the starts of successive trials. Each *S* was given 30 training trials in a single session regardless of its performance. This deviation from the previous methodology was adopted because several groups were expected to perform very poorly, making training to a fixed criterion impractical. The study was run one replication at a time, with the order of testing Ss from each group randomized within replications.

Data collected on each trial included time until *S* made the correct response and notes on S's behavior. For Ss given jump-out training, the point where their forepaws landed on the masonite platform was recorded in polar coordinates using the center of the shock box as the origin. This measure was included because CBAJJ mice had been observed to make long, inaccurate leaps in previous experiments.

Results and Discussion

The latency of escape from shock on the first training trial and the total errors during the 30 training trials are shown in Fig. 1. Each group was composed of nine or ten *Ss*, one *S* having been rejected because of experimenter error in five of 16 groups.

The latency of first escape, which is indicative of the unconditioned response to electric shock, revealed the very poor jumping reaction to shock exhibited by A/J in the previous study (Wahlsten 1972a). This single comparison of jump-out latency for A/J and all other groups accounted for 62.5% of the entire between-groups sum of squares. The difference in first escape latency between jump-out and one-way training was significant only for A/J ($p < 0.001$) and CBA/J ($p < 0.005$), CBA/J being slower to run than jump. Since the other strains with defective eyes escaped as quickly as normal strains on either task, visual system effects were not apparent for initial escape from shock. Observation of Ss suggested that the frantic jumping and careening off the walls on the first shock trial injected an element of randomness into the first escape latency.

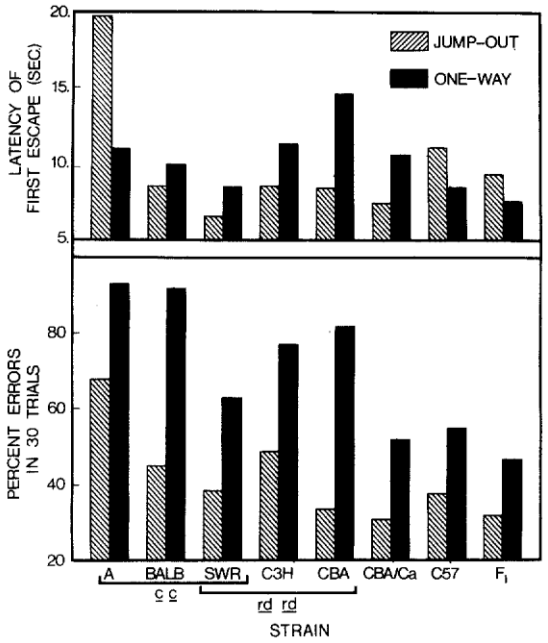

Fig. 1. Mean latency of the first escape and mean percent errors in 30 trials on jump-out and one-way avoidance training for several mouse strains.

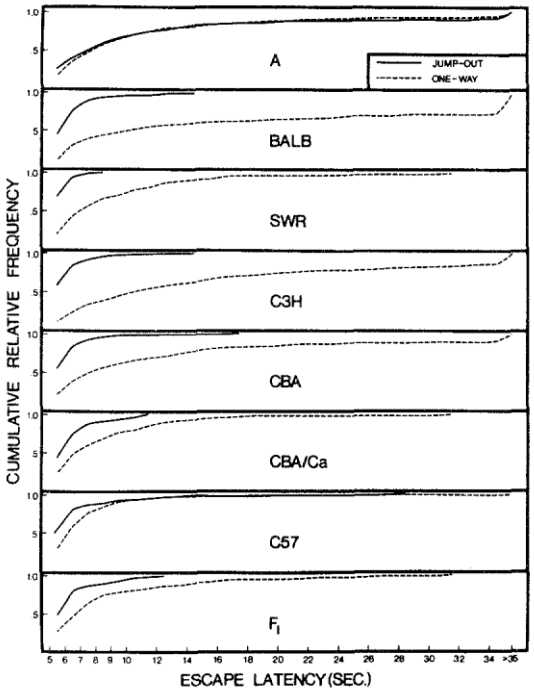

Fig. 2. Cumulative relative frequency distributions of total escape latencies on jump-out and one-way training for seven mouse strains. Group distributions represent the average of individual distributions of proportions of responses in each interval, which weights each *S* equally regardless of its number of escape trials.

Latencies to escape shock over all error trials were compared by pooling the frequency distributions of response times for all Ss in a group, weighting each *S* equally regardless of its number of errors. The cumulative frequency distributions for all groups are shown in Fig. 2. It was clear that only A/J mice encountered prolonged difficulty in jumping out of the box on error trials. Running (one-way) was generally slower than jumping for all other groups, with certain strains showing a large deficit compared to jump-out escapes. Three strains had an appreciable frequency of trials in which the shock box was never escaped at all. BALB/cJ mice were particularly talented at learning to stand spread-eagled in order to contact only the grid bars which were wired in common, a distance of 1.5 inches; this unusual adaptation occurred only in one-way training. As a result, albino strains were generally slower when required to run. Retinal degenerate strains were slightly faster than phenotypically normal strains on jump-out escape ($p = 0$ .05) , but they were worse than normal strains on one-way escape ($p < 0.01$, Kolmogorov-Smirnov two-sample test).

Learning rate was indicated by percent errors in 30 trials as depicted in Fig. 1. A Newman-Keuls test ($\alpha = 0.05$) on jump-out scores revealed that A/J did more poorly than all other strains, while no differences among the other strains were significant. For one-way training, one set of strains (A, BALB, CBA, C3H) did rather poorly, and the remaining strains (SWR, C57, CBA/Ca, $B6AF_1$) performed relatively well; differences within each of these two sets were not significant, while all differences between strains in separate sets were significant.

The distance from the edge of the shock box at which S's forepaws landed on successful avoidance trials confirmed earlier observations of the springy nature of CBA/J mice. The mean distance for CBA/J (8.48 cm) was far greater than that for any other strain, the single comparison of CBA/J to all other strains accounting for 94% of the between-groups sum of squares. The next longest leapers, C3H/HeJ (3.28 cm), were not significantly different from the more restrained strains, all of whose means fell in the range of 1.6-2.1 cm from the edge of the box. These results are barely suggestive of an effect of *rd* on jumping accuracy.

Thus neither the *c* nor the *rd* gene appeared to have a consistently deleterious effect on jump-out avoidance performance, as indicated by both escape latencies and total errors. One-way performance, on the other hand,

was generally poor in those strains possessing either *c* or *rd*. The obvious exception to this conclusion was the strain SWR/J. Actually, most Ss of this strain did poorly on one-way training, but one remarkable *S* made only three errors. It should also be mentioned that SWR mice were certainly the jumpiest, wildest, most savage subjects ever tested in avoidance training by this author; it was often necessary to use a leather glove and, in some instances, a large pair of forceps to recapture Ss which had hurtled blindly beyond the test apparatus when being put into or removed from the holding cage. As a result, their performances cannot be confidently compared to those of other strains.

Since the effects of *c* and *rd* on one-way avoidance behavior could not be conclusively demonstrated using inbred strains alone, it was deemed necessary to explore further their effects in additional experiments.

EXPERIMENT 2

The second experiment examined the influence of albinism on escape and avoidance behavior in a segregating generation. Other studies had found albinism to retard active avoidance learning when placed on either a segregating background (Winston *et al.*, 1967) or an isogenic background (Henry and Schlesinger, 1967), but the results could have occurred because of freezing under bright lights (Wilcock, 1969). Since both the previous (Wahlsten, 1972a) and present studies in this laboratory were conducted under dim red light, photophobic reactions to light should have been absent. Although the first experiment showed that albino strains can learn jump-out quite well, corroboration of the lack of an albinism effect was sought in segregating generations as well.

The primary question was whether the poor active avoidance of strain A/J was attributable to albinism or to other unidentified loci. The design used to answer this question entailed crossing males of several strains with females of B6AF $_1$ J (*c/+;* from cross of C57BL/6J female × A/J male) which were heterozygous for albinism. C57BL/6J males (+/+) yielded offspring with black pigmentation and no loci homozygous for other A/J-type alleles. C57BL/6J-c^J (c^J/c^J) males carrying the albino mutation yield half albino and half black offspring which were not homozygous for any other A-type alleles. A/J males (*c*/*c*) yielded half albino and half pigmented offspring which were homozygous for an average of half the other alleles carried by A/J. Other albino males randomly chosen from a four-way cross similarly gave half albino offspring but with unpredictable homozygosity at other loci. Thus an effect of albinism should be observed in all three crosses involving an albino male parent, while effects of other recessive genes should be observed only in the litters sired by an A/J male.

Method

Subjects

Female parents of the hybrid B6AF$_1$/J and male parents of the inbred strains A/J, C57BL/6J, and C57BL/6J-c^J were procured from The Jackson Laboratory. Three albino males from the fourth and fifth generations of a four-way cross between A/J, CBA/J, C57BL/6J, and DBA/2J (see Wahlsten, 1972a) were also used, and they were designated as H4 males (*c*/*c*).

All subjects were conceived in and raised until weaning at 21 days by females of the B6AF$_1$/J hybrid cross (C57BL/6J female × A/J male). All females were experimentally naive at the beginning of the experiment. Male parents were of varied experience. They were housed with the females only until definite signs of pregnancy were evident.

Matings were continued until sufficient offspring were available to comprise groups of ten or more for each training condition. Within each litter, equal numbers of offspring were assigned to either jump-out or one-way training. All offspring in each litter were included in the study. Subjects were from 55 to 60 days of age at the time of testing. The numbers of subjects of each sex under the various training conditions are shown in Table I.

Table I. Summary of Subjects Tested in Experiment 2

	Jump-out training				One-way training			
	c/c		+/–		*c/c*		+/–	
Male parent	Male	Female	Male	Female	Male	Female	Male	Female
C57BL/6J	–	–	7	4	–	–	8	4
C57BL/6J-c^J	7	7	5	5	7	6	5	6
A/J	8	8	9	6	8	8	8	7
H4	8	8	7	3	8	7	7	4

Apparatus and Procedure

The apparatus and procedure for jump-out and one-way training were identical to those described for Experiment 1.

Testing was done blind insofar as the experimenter did not know the strain of the male parent, but no method was found to conceal the identity of albino subjects.

Results

The mean latency of the first escape is shown in Fig. 3. Although albino Ss generally jumped out more slowly, the effect was not significant ($F = 1.83$, $p > 0.10$) owing to large within-group variance. Such large "error" variance would be understandable if certain groups were segregating at other loci affecting the behavior. Jump-out latencies were significantly longer for groups with an A/J parent than for those with a C57BL/6L-c^J parent ($F = 5.43$, $p < 0.025$); this single comparison accounted for 74% of the sum of squares between groups. There were no significant effects for one-way escape latency attributable to albinism or strain of male parent (all $F <$ 1.0).

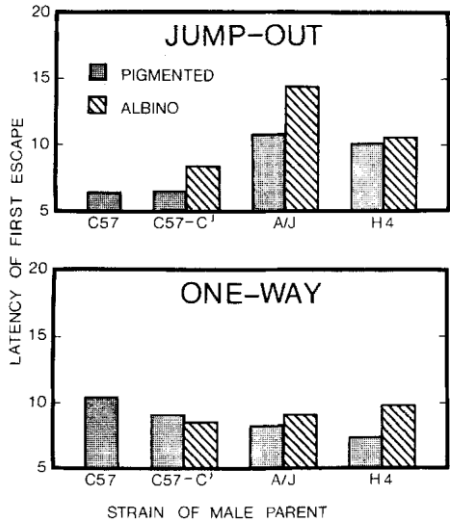

Fig. 3. Mean latency of the first escape for albino and pigmented offspring from several test crosses. Since all offspring had a B6AF$_1$/J mother, only the strain of the male parent is shown.

Comparison of distributions of escape latencies on all trials revealed a similar pattern. No deleterious effects of albinism were evident, but jump-out escape latencies were generally slower for Ss with an A/J parent than for those with a C57BL/6J-c^J parent.

The total errors in 30 trials were not significantly different for any comparison of groups in either jump-out or one-way training (all $F < 1.0$); large within-groups variation was again the rule. The total errors averaged over all groups were not very dissimilar for jump-out (14.28) and one-way training (16.99), compared to training-type differences for the parent strains.

Discussion

Since no significant effects of albinism were detected in any mating combination for any measure of escape or avoidance behavior, the substantial difference between A/J and other strains commonly observed in this laboratory cannot be attributed in large degree to the albino gene. Although the possibility of a small is effect or perhaps a maternal effect cannot be entirely excluded, their potential importance is minimized by the observation that the bizarre A/J-type escape behavior was in fact observed in pigmented offspring of pigmented mothers, provided that they had an A/J father. The results also demonstrate that the poor one-way avoidance learning by the three albino-strains in Experiment 1 is not seen in albino Ss of heterogeneous background.

The genetic bases for the A/J-type behavior appear to be different for initial escape and avoidance learning phenotypes. Poor jump-out escape behavior was observed in the $F_1 \times$ A/J backcross with a frequency which suggested the influence of a major recessive gene, although the additive effects of several loci could also have led to the observed distributions. Avoidance learning, on the other hand, was seldom as poor in Ss of the backcross as in A/J mice. A polygenic system may exist in which A/J is homozygous recessive at several genetic loci influencing the generation of anticipatory avoidance responses.

EXPERIMENT 3

In Experiment 1, Ss with retinal degeneration were found to do well on a jump-out task, but they had great difficulty in escaping or avoiding via a hole in the wall in a one-way task. The CBA/CaJ strain with normal retinas performed normally on the one-way task, but it probably differed from CBA/J at several other loci besides *rd.* Thus the present experiment was conducted to evaluate the effects of *rd* on a heterogeneous background. Offspring of a backcross of CBA/J x C57BL/6J to CBA/J were tested on either jump-out or one-way avoidance training.

Method

Subjects

F_1 mice were derived from a cross of CBA/J female (*rd*/*rd*) and C57BL/6J male (+/+). These F_1 mice (*rd*/+) were then backcrossed to the CBA/J strain in a reciprocal backcross. Offspring from three litters of F_1 female × CBA male and three litters of CBA female × F_1 male were tested from 55 to 60 days of age. A total of 56 mice from these six litters were used.

Apparatus

All training apparatus was identical to that of Experiment 1.

Procedure

Ss were trained using a triple-blind control procedure. Equal numbers of males and females from each litter were randomly assigned to jump-out or one-way training by an *E* who had no knowledge of the state of their retinas. Training was administered by another *E* who did not even know the purpose of the experiment. Finally, a substantial number of Ss were blind themselves.

All training was given as in Experiment 1, with each *S* receiving 30 trials. Response latency and landing point of the front paws were recorded on each trial.

Histology

Within 1 week of training, each *S* was killed by cervical dislocation, and its eyeballs were rapidly removed and immersed for 24 hr in Kolmer's fixative. The retinas were then dissected from the eyeballs with a cataract knife, washed thoroughly, and later encased in 10% gelatin. Sections (25 μ) were cut parallel to the foveal axis in several regions of the retina and were then stained with Harris's hematoxylin. Retinas were evaluated with the aid of published descriptions of *rd* (Tansley, 1954; Sorsby *et al.,* 1954).

Results

Careful examination of the stained retinas confirmed previous findings that *rd* is a completely recessive, autosomal gene. Either a S's retinas were quite normal or both retinas were completely devoid of the rods, outer nuclear layer, and outer fiber layer. Twenty-nine of 56 Ss were retinal degenerate, the proportions of defectives in jump-out and one-way training being 0.56 and 0.48, respectively.

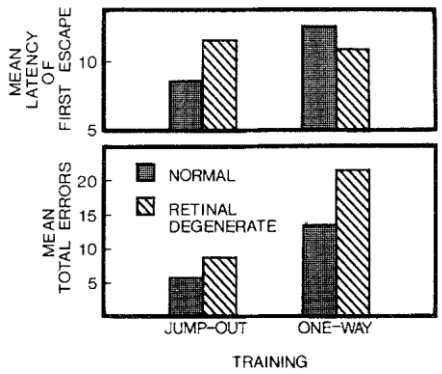

Fig. 4. Mean latency of the first escape and mean total errors in 30 trials on jump-out and one-way training for retinal degenerate and normal offspring of a reciprocal test cross.

Latency of the first escape and total errors in 30 trials are shown in Fig. 4. Differences between normal (*rd/+)* and *rd/rd* Ss in initial escape latency were not significant for either jump-out or one-way training (both $t < 1.0$). Total escapes did not differ in jump-out training, while retinal degenerate Ss generally escaped more slowly in one-way training ($p = 0.05$, Kolmogorov-Smirnov two-sample test).

Total errors in jump-out training were slightly less for normal Ss ($t = 1.80$, $0.10 > p > 0.05$), but the small difference in means was primarily a result of two degenerate Ss which learned very slowly. However, retinal degeneration greatly retarded one-way avoidance learning ($t = 3.68$, $p < 0.001$). In fact, this difference in total errors between normal and *rd/rd* Ss (8.2 errors difference) was as great as the original difference in one-way errors between C57BL/6J and CBA/J strains (8.3 errors) in Experiment 1.

The lengths of leaps on jump-out avoidance trials were somewhat longer for retinal degenerate Ss (3.25 cm) than for normal Ss (1.15 cm), but the difference was only marginally significant ($t = 2.0$, $p < 0.05$). Six of 15 *rd/rd* mice made leaps outside the normal range, with concomitant high variability. The two *rd/rd* Ss which made few avoidances nonetheless jumped accurately.

Discussion

The results of Experiments 1 and 3 demonstrate that *rd* does not retard jump-out escape or avoidance behavior; no differences appeared on jump-out performance between inbred strains CBA/J (*rd/rd*) and CBA/CaJ (+/+), while retinal degenerate and normal offspring of a backcross were likewise similar. Apparently, *rd/rd* led to less accurate jumping, but this deficit hardly attenuated avoidance learning. One-way performance was greatly disrupted by retinal degeneration, however. Both inbred mice and offspring of a backcross required more time to escape shock and more trials to anticipate shock successfully when they had retinal degeneration.

The fact that jump-out avoidance is easily learned by *rd* mice demonstrates that jump-out and one-way tasks differ in sensory as well as response-mode requirements. This makes one-way avoidance learning more difficult on two accounts: first, visual cues must be utilized to direct the response properly and, second, Ss must run instead of jump to avoid shock, running being a response that most mice shun after a few shocks when a jumping response is also available (Wahlsten, 1972a).

These results also strongly suggest that poor learning by C3H and CBA strains previously reported is indeed a manifestation of their blindness. Studies which specifically employed visual cues with these strains revealed slow learning (Wimer and Weller, 1965; Bovet *et al.*, 1968; Bovet-Nitti, 1969), while those using position or

auditory cues showed learning to be quite good compared to that of normal strains (Royce and Covington, 1960; Collins, 1964; Stasik, 1970). Hopefully, future research with strains carrying *rd* will be done only when the reduction of visual information is desired. Of course, care should be exercised in such applications, for *rd* does not seem to yield total blindness[2] (see Fuller and Wimer, 1966), especially at younger ages (Nagy and Misanin, 1970). It is also conceivable that *c* and *rd* may interact to attenuate the deficit produced by each alone, for *rd* increases the visual intensity threshold, while *c* increases the amount of available light at the retina. In fact, this may have been the reason that some SWR/J mice (*rd/rd, c/c*) seemed to learn one-way avoidance reasonably well.

GENERAL DISCUSSION

From the present research in conjunction with published reports from many other laboratories, it is clear that both c and *rd* genes have potent, deleterious effects on visual sensation which, nonetheless, may be largely circumvented by appropriate modifications of discriminanda and ambient light. Albinism appears to have minor effects on open-field behavior under dim red light (DeFries *et al.,* 1966; McReynolds *et al.,* 1967), and active avoidance behavior is similarly unaffected. Retinal degeneration seems to have no effects beyond blindness, since *rd* strains learn well with nonvisual cues.

It is also concluded that previous statements about the lack of relation between initial reaction to shock and subsequent jump-out avoidance learning (Wahlsten, 1972a) need not be modified because of *c* and *rd* effects, since training was given under dim red light on a task not requiring normal vision.

There are, however, aspects of these sensory deficits which indicate that more subtle effects may exist. Degeneration of the rods in the retina leads to abnormally small cells in certain areas of the visual cortex (Gyllensten and Lindberg, 1964), but it is conceivable that other visual areas can acquire new functions which may increase auditory or tactile sensitivity. This is especially plausible because the degeneration occurs during a time when cortical organization and synapse formation are not quite complete.

Albinism may lead to differences in visual acuity which are not evident in a simple avoidance task or open-field exploration. Lashley (1930) found an albino rat strain to have poorer visual acuity than a pigmented strain, while Owen *et al.* (1970) found greater acrophobia for albino mice, even under dim red light. Apparently, the primary visual projections from the retinas of albino animals provide inadequate ipsilateral inputs to the lateral geniculate body (Guillery *et al.,* 1971) and to the superior colliculus (Kalil *et al.,* 1971), differences which may be manifested in the visual evoked potentials recorded from cerebral cortex of the albino rat (Creel *et al.,* 1970). Since these deficiencies should substantially impair binocular vision, and since binocular vision leads to lower visual intensity and acuity thresholds (Pirenne, 1967), it follows that albino animals should have impaired visual acuity and depth perception.

Note:

[2] Recent research on light-induced retinal degeneration in albino rats (Anderson and O'Steen, 1972) has detected substantial visual discrimination ability in animals suffering virtual total loss of rods, outer nuclear layer, and outer fiber layer.

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