ALBINISM, A SEMI-LETHAL AUTOSOMAL MUTATION IN *LEBISTES RETICULATUS*

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THE condition of xanthism or yellowness, accompanied by a generalised defective development of melanin pigment, is well known among fishes of many families. The "golden tench" (*Tinca tinca*) and the golden orfe (*Idus idus*) are familiar examples of such races among the Cyprinidæ, and the common goldfish *Carassius auratus* offers an interesting case of the genetically controlled progressive destruction of melanophores during the life of the individual which has been investigated by Goodrich and others. Similar aberrations of melanin development and distribution are familiar among various small freshwater Teleosts commonly kept as aquarium fishes, such as the Cichlid *Pterophyllum eimekei*, and the Cyprinid *Brachydanio albolineatus*. In these the genetics of the melanin deficiency does not seem to have been well worked out. In the Cyprinodont genus *Oryzias*, however, careful and extensive work has shown that the genes for melanin production, as well as those for other pigments of body colouration, constitute a series of Mendelian recessives allelic to the wild type (Goodrich, 1933).

Among the viviparous Cyprinodontes deficiencies in melanin pigmentation have long been familiar and can be readily analysed genetically. Xanthism has been found in aquarium-bred races of *Heterandria formosa*, *Platypoecilus maculatus* and *variatus*, *Xiphophorus hellerii*, and *Lebistes reticulatus*, and may well be discovered in related genera not yet as extensively examined. It has been studied exhaustively in *Platypoecilus* and *Xiphophorus* by Gordon and others to whom we have given the later references and in *Lebistes* by Goodrich, Haskins and Druzba, and others. The genetic characters involving the loss of melanin pigment in these fishes have been found to behave as Mendelian single-factor recessives allelic to the wild-type and to be located in the autosomes.

Albinism, involving a substantial or total loss of melanin in body pigmentation and a complete or nearly complete absence of melanin in the eye, has been less often observed among Teleost fish, although it is by no means a rare occurrence. It is well known in the trout (*Salvelinus fontinalis*) and strains largely devoid of melanin have been maintained by hatcheries for a number of years. In the Paradise fish (*Macropodus opercularis*) albinism has been shown to behave as a single-factor Mendelian autosomal recessive, as would be expected on

the basis of other experience (Goodrich and Smith, 1937; Kosswig, 1935). Among the viviparous Cyprinodontes it has been reported from aquarium-bred races of *Xiphophorus hellerii* and *Mollienesia latipinna*, and is known to behave similarly genetically in these forms (Gordon, 1937, 1941, 1942; Schreitmuller, 1934). Curiously, it has never been reported in *Platypoecilus*, nor has it been found in *Lebistes*, so far as is known to the authors, previous to the cases here described. It has never been recorded in the related genera *Limia*, *Poecilla*, *Micropoecilla*, *Parapoecilla*, or *Allopoecilla*, nor among the *Heterandriini* nor the *Gambusiini*, despite the fact that members of some of these groups have been bred in large numbers under artificial conditions.

Lebistes reticulatus is unusually favourable material for a variety of genetic work. It seems potentially particularly well adapted for studies in the dynamics of natural populations. For these reasons, and because the character for albinism has apparently not been previously reported within the species, it seems justifiable to describe its genetic behaviour.

DESCRIPTION OF MATERIAL

The detection of albinism in *Lebistes*, like its discovery in *Xiphophorus*, was accidental. The mutation appears to have segregated in highly inbred stocks which had been intensively selected for qualities of colour and form by Dr Abbs, of Ampere, New Jersey. After the death of Dr Abbs these stocks were made available to aquarists and two albino males were detected among the progeny of phenotypically wild-type males by Mr Frederick Kraus in 1944. Subsequently a few additional homozygous albinos evidently appeared among heterozygotes of the Abbs stock distributed to other persons. In consequence, several colonies were built up in isolation. Despite the fact that these were apparently derived originally from the same clone, they exhibit some recognisable phenotypic differences. Two of these stocks were employed in the present work. The first was obtained through Mr Harrison Miller and Mr Frederick Kraus and included only stock from the homozygous albino male originally discovered. The second, obtained through Dr Myron Gordon, had been segregated by Mr B. H. Kaminer from heterozygous stock of the original Abbs material. Additional data of Dr Gordon are also included taken from a third stock independently segregated from the original Abbs material.

The character *Albino* in *Lebistes* leads in the homozygous condition to the production of macroscopically almost white fish, exhibiting a marked degree of translucence. Dendritic melanophores superficial to the scale and melanin-containing pigment cells in the underlying dermis have not been identified in the adult fish. The material examined in this connection includes two hundred scales from many parts of several individuals and a fairly careful examination has been

made of the dermis. In the nearly mature embryo, however, dendritic melanophores of essentially normal form can frequently be detected. They expand and contract as do the melanophores of the wild-type, but are extremely lightly pigmented, appearing in outline as faint white smudges or " ghosts " against the light skin background. The eye is very largely unpigmented, producing the characteristic albino " pink eye." Occasional individuals showed appreciable iris pigmentation. Their genetic behaviour was identical with those in which melanin was virtually absent. This small development of melanin may well be a function of lowered temperature, but this has not been definitively determined.

It seems clear from both developmental and genetic evidence that albinism in *Lebistes* is a semi-lethal. Albino fish exhibit much less tolerance to variations in either the temperature or the acidity of the

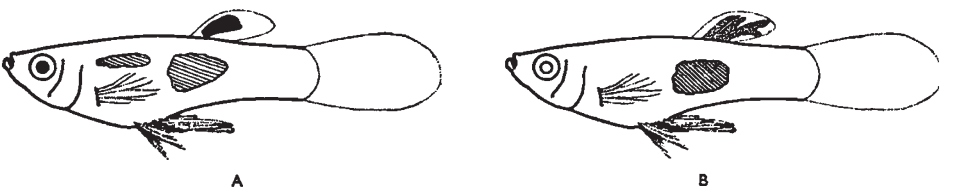


FIG. 1.—A. Male *Maculatue* wild-type, showing pigmented dorsal fin, with dense aggregation of fully coloured melanophores. B. Male *Maculatus* albino, showing sparse accumulation of melanin and less distinct definition of dorsal-fin spot (×3).

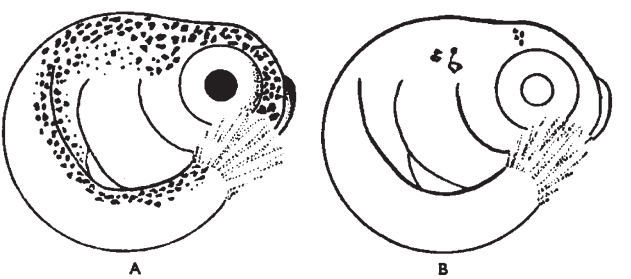


FIG. 2.—A. Ma ıre embryo, wild-type, showing melanophore development and pigmentation. B. Mature embryo, albino, showing lack of pigmentation in eye and melanophores.

water than do normal individuals. Females are very short-lived even under optimal circumstances. Though kept under the best conditions which could be devised, a single pair often being placed for breeding for long periods in forty gallon tubs or tanks heavily and naturally planted and fed with living food, it has proved impossible to maintain any individual albino female for so long as a year. The average fish has sickened and died after the production of the third or fourth brood. Males have been maintained for two years, but fall far short of the normal length of life in the wild-type.

The albino breeds very poorly. In part, this seems to be due to the inability of albino young to survive parturition. The largest brood of living albino young obtained from albino parents has been

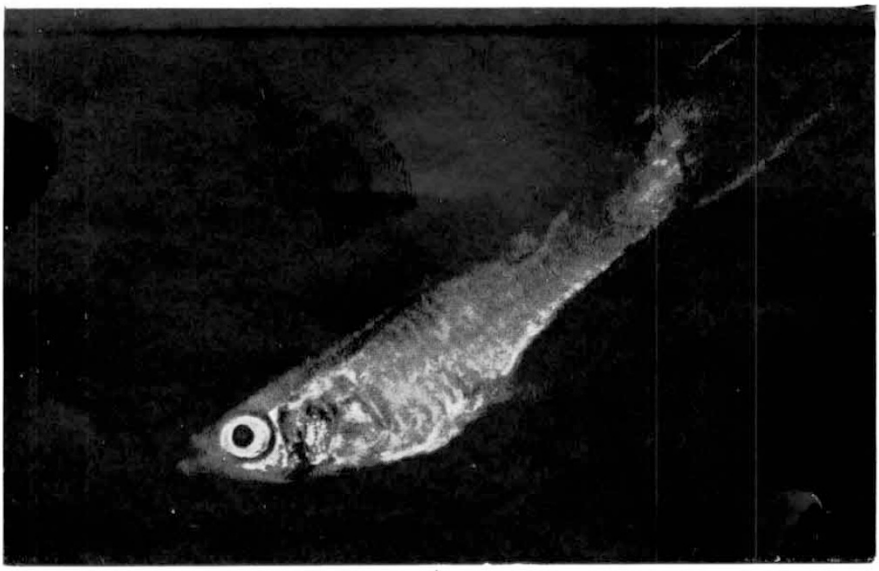
seven, the average being three or four.* As will later be shown, the ratios of albino among F_2 and backcrossed broods are always very low, although dissection of females in the week before the young are ready for parturition indicates nearly normal ratios among the embryos. In part, the low reproduction rate appears to be due to semi-sterility in the male, and probably also in the female. Long exposures of wild-type females to albino males, and of the reverse cross, were found necessary even for the production of phenotypically wild-type young. Further, females which had been fertilised by albino males usually proved on dissection to be sterile after the birth of one brood unless reimpregnated. This is practically never true in wild-type fish, and suggests a very limited viability of the sperm of albino males when lodged in ovarian tissue, regardless of the nature of that tissue.

FORM AND METHODS OF PROCEDURE

In addition to testing the behaviour of the new mutant with wild-type, it seemed desirable to investigate its behaviour with certain other alleles to wild-type melanin pigmentation in *Lebistes*. Two of these, described by Goodrich *et al.* (1944) and named by him *Golden* and *Blond* (*g* and *b*) were selected. These are both known to behave genetically as single-factor Mendelian autosomal recessives to wild-type, and are non-allelic to one another. The first of these mutations was obtained in pure culture in this laboratory in 1934 and has been inbred since that time. The second was obtained in 1938 and has been maintained genetically pure and under carefully controlled conditions since. Stock of the double recessive, called by Goodrich *Cream*, was obtained in 1943 through the kindness of Dr Goodrich from his laboratory and has been maintained here since. Although the *Golden* and *Blond* stocks were not obtained originally from Dr Goodrich's laboratory, cross-tests between his stocks and ours, involving somewhat over one thousand fish, have shown them to be genetically identical.

The mutation *Golden* in the homozygous condition yields a fish of a distinctly yellow, but not translucent, cast showing scattered patches of melanic pigment in the body and fins. Melanin is accumulated especially about the edges of the scales and in the underlying diamond pattern, resulting in a marked accentuation of the normal reticulate markings of *Lebistes*. Goodrich has found that in *Golden* fish both the dendritic and corolla types of melanophores are larger than in wild-type, but are more sparsely distributed, there being only about 50 per cent. as many per unit area as in either wild-type or *Blond*. The eye is completely pigmented. The mutation appears to be fully recessive, the heterozygotes being indistinguishable from wild-type.

* In table 4 are shown some results obtained by Dr Myron Gordon which are of significance in this connection.



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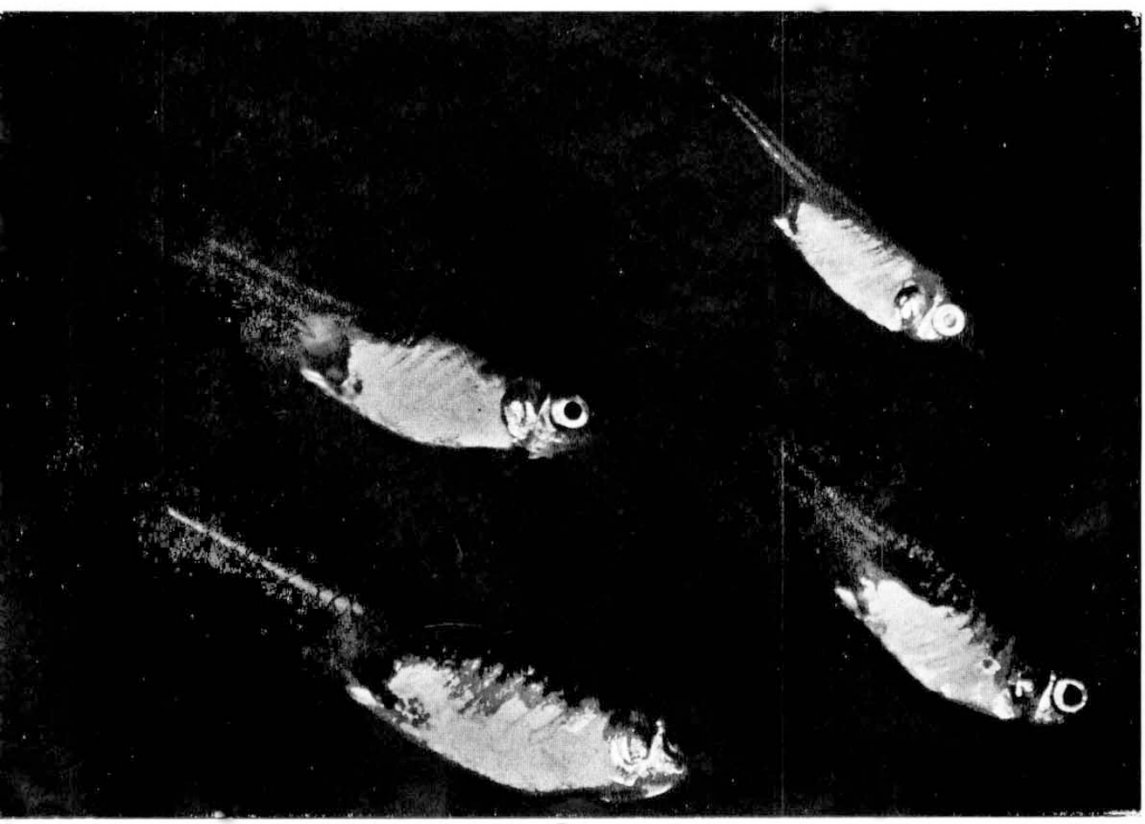

B

A. Male *Armatus* albino, showing light eye pigmentation and characteristic retention of sex-linked colour patterns.

B. Female albino (upper right) compared with *Blond* (upper left), *Golden* (lower right), and wild-type (lower left).

The mutation *Blond* leads in the homozygote to the production of a light yellow, semi-translucent fish with no macroscopic evidence of melanin pigmentation, and hence no reticulate pattern. Actually, the diamond pattern beneath the scales is maintained, but the corolla-type melanophores which in *Wild-type* and *Golden* contribute so conspicuously to the marking are replaced by much smaller, macroscopically invisible punctate cells. Dendritic melanophores are present, but are smaller than those of either *Wild-type* or *Golden*. The density of melanophores is roughly equivalent to that in *Wild-type*. The eye is fully pigmented. The mutation, like *Golden*, is completely recessive.

The double-recessive *Cream* in the homozygous condition phenotypically resembles *Blond*, but, as there is even less melanin and no chromatophores are macroscopically visible, it is of a rather warmer yellow colour and is even more translucent. Small punctate melanophores are present, which tend to follow the diamond pattern, and dendritic cells are also found. The eye is fully pigmented.

In *Golden* and in *Blond*, as well as in *Cream* and in *Albino*, the factors for sex-colouration in the male, characteristically behaving as though located in the Y- or the X-chromosomes, are phenotypically expressed in so far as the deficient melanin available will permit. It seemed desirable, both for the sake of maintaining stock which was genetically uniform and was known so far as possible, and for the purpose of studying the expression of these patterns in the albino and of determining whether any linkage existed, to combine the body-colour factors in the autosome with certain known sex-patterns.

For this purpose, five genes for sex-colouration were used. All of them were obtained through the kindness of Dr Winge from his laboratory in 1934 and have been maintained here in pure culture since that date. The first of these, *Maculatus*, was described by Winge in 1922. It leads in the wild-type to the production of an intense melanic spot in the dorsal of the male. As Goodrich *et al.* (1946) has shown, the development of this spot is markedly influenced by the sex-hormones, being repressed by estradiol and probably requiring the presence of methyl testosterone. In the presence of *Blond* body colouration, the *Ma* gene fails of expression to the extent that the area usually occupied by melanophores is very largely replaced by iridocytes. Goodrich, in the same publication, has indicated that, in the presence of *Golden*, the *Maculatus* gene is normally expressed. We have found this to be true in somewhat less than 50 per cent. of the 800 males tested since 1934. In the abnormal cases the spot is represented by scattered clusters of melanophores only slightly more dense than in the surrounding fin tissue. In the albino, some melanin is present in the dorsal spot, as shown in the figure, but is so "dilute" as to present macroscopically a bluish or reddish tint rather than a densely black one. Many iridocytes are also present.

The characteristic form of the spot is maintained, in contrast to the abnormal males of *Maculatus Golden*.

Armatus and *Pauper*, the remaining Y-chromosome sex factors used in combination with *Golden*, *Blond*, *Cream*, and *Albino*, were obtained by Winge in 1922, the former from Danish domestic stock, the latter from the West Indies, and were described in 1927. They lead respectively to the elongation and colouration of the lower rays of the caudal fin, producing a " sword-like " effect, and to the formation of characteristic patterns of body-colouration about the tail. Since neither depends upon melanin deposition to any extent, both are reproduced almost typically in the males of all the body-colour mutants. The remaining two characters used, *Coccineus* and *Vitellinus*, likewise depend largely upon non-melanin patterns for their macroscopic phenotypic expression, and both are approximately reproduced in the light mutants. Both behave as though located in the X-chromosome. All these characters show crossing-over from one sex chromosome to the other, as Winge has indicated. The percentage of such crossing-over is very low in the cases of *Armatus* and *Pauper*.

The infertility and lack of hardiness of albino *Lebistes* make the accumulation of genetic data unusually lengthy and tedious if reasonable numbers of progeny are to be secured. The work described was carried on over a period of approximately three years. It was conducted in a greenhouse especially designed for the breeding of viviparous *Poeciliids* and containing between four and five hundred tanks. Young fish for breeding were isolated shortly after birth and were reared to maturity in individual containers. Crosses were made in one-gallon, two-gallon, five-gallon, twenty-gallon, and forty-five-gallon tubs or tanks, heavily planted with *Elodea*, *Sagittaria*, *Vallisneria*, *Hydrocleis*, and young specimens of *Nymphaea*. Culture conditions were the standard ones for *Lebistes*. The progeny of each mating were counted shortly after birth and the evidence seemed good that the cover provided for the young fish was thick enough so that nearly all viable individuals escaped. Embryos which never became activated, however, were probably picked up by the parent females almost immediately after birth and hence were not detected.

GENETIC ANALYSIS

Crosses in the F_1 were made in both directions between *Albino* and *Wild-type*, *Albino* and *Golden*, *Albino* and *Blond*, and *Albino* and *Cream*. Crosses in the F_2 were also made in both directions, and in the various possible combinations within each pair of types depending on the lineage of each parent in the cross. Backcrosses were made in the same fashion, in the four possible combinations for each type, depending upon the lineage of the heterozygous parent. All the progeny of the F_1 crosses, except when both parents carried a *Golden*, *Cream*, or *Blond* mutation in addition to the albinism of one of them,

were phenotypically wild-type. It was evident, therefore, that the mutation *Albino* was both recessive to *Wild-type* and was non-allelic to the other body-colour mutations. This situation is analogous to that reported by Gordon (1941) for albino *Xiphophorus hellerii*. In the F_2 generation, Golden, Blond, and Albino all assorted independently, as shown in table 6, although the respective dihybrid and trihybrid ratios vary far from the expected, largely because of the low viability of the albino forms. Fish homozygous for both *Golden* and *Albino* and for both *Blond* and *Albino* were phenotypically indistinguishable from simple *Albino*, but could of course readily be detected genetically.

DISCUSSION

It is evident that the gene for albinism is an autosomal mutation, non-allelic with those for *Golden* and *Blond*. It assorts independently from the genes for *Golden*, *Blond*, *Armatus*, *Pauper*, or *Maculatus*. It is probably not linked to *Coccineus* or *Vitellinus*, but the data are not yet sufficient to establish this with certainty. It is clearly recessive to *Wild-type*. The ratios in the F_2 and the backcross, however, are so extraordinarily aberrant that no more specific conclusions than these can be drawn from them. While it is true that the F_1 gives the expected 100 per cent. of pigmented eyes, the ratio in the F_2, instead of being 1 albino : 3 non-albino proves to be 1 albino : 51·1 pigmented, and the backcross, instead of being 1 : 1, proves to be 1 : 9·1.

This situation, though more extreme, is reminiscent of that described by Gordon in the case of albino *Xiphophorus hellerii*. As a further check, therefore, mature pregnant females were sacrificed a few days before their broods were ready for delivery and the embryos were examined for eye pigmentation. At this stage of maturity the eyes of normal embryos are heavily pigmented, and albino individuals, showing little or no eye pigmentation, contrast sharply with them and can readily be distinguished. The results of these counts are presented in table 5.

These ratios, although still somewhat low on the albino side, are much closer to the theoretical and suggest that albinism in *Lebistes* is indeed a Mendelian single-factor recessive, but is semi-lethal in character, and that the greatest mortality of albino embryos occurs at the time of birth. This is also strongly indicated by an analysis of an albino brood made by Dr Myron Gordon and shown in table 4.

Certain other factors suggest the same conclusion. The work reported included the data from 173 broods in the F_2 and the backcross taken from over 100 females. It was noticed that among these females a few consistently produced almost the theoretical number of living albino young, in marked contrast to the great majority. This again recalls the condition described by Gordon, but was even more

extreme. The successful females were usually themselves phenotypically non-albino, but in one or two cases albino females produced appreciable numbers of albino young in backcrosses. Thus the definite impression was given that certain females are more successful in producing viable albino young than others.

A second impression, which is difficult to demonstrate quantitatively at present because of the meagreness of the data, is that a higher proportion of living albino young is produced in the summer than in the winter months. Feeding was maintained uniform in quantity and quality the year around, but light was more intense in the summer and there can be no doubt that infusoria were more abundant in the tanks in summer than in winter. This may have been a contributing factor.

It is proposed to designate this new recessive mutation by the symbol *a*.

SUMMARY AND CONCLUSION

A new mutation in *Lebistes reticulatus*, producing albinism in the homozygote, has been described and analysed. It has been shown to be autosomal and recessive to wild-type, and to be non-allelic to the recessive pigmentation characters *Golden* or *Blond*. It is not genetically linked to them nor to the sex-factors *Armatus*, *Pauper* nor *Maculatus*. Evidence obtained from the examination of unborn embryos indicates that it is a single-factor Mendelian character. Evidence obtained from the counts of young fish is wholly inconclusive on this score, owing to an exceedingly high mortality of albino embryos at birth. In addition to its semi-lethal character with respect to the survival of the young fish, the gene for albinism appears highly inimical to the adult. The homozygous adult albino is typically semi-sterile in both sexes, is of much shorter average life than normal *Lebistes*, especially in the female, and is more susceptible to disease and to adverse physical conditions. Work is planned to investigate the physiology of the young and the adult albino to determine the cause or causes of this semi-lethality.

TABLE 1

Totals of F_1 progeny (816 fish)

Cross	Pigmented eye	Albino
Wild-type × Albino (AA × aa)	147	0
Albino × Wild-type (AA × aa)	144	0
Golden × Albino (AA × aa)	61	0
Albino × Golden (AA × aa)	100	0
Blond × Albino (AA × aa)	65	0
Albino × Blond (AA × aa)	69	0
Cream × Albino (AA × aa)	153	0
Albino × Cream (AA × aa)	77	0

TABLE 2

Totals of F_2 progeny

Cross	Pigmented eye	Albino
Wild-type × Albino * (Aa × Aa) .	339	7
Albino × Wild-type (Aa × Aa) .	33	0
Golden × Albino (Aa × Aa) a) .	75	6
Albino × Golden (Aa × A) . .	329	0
Blond × Albino (Aa × Aa . .	168	7
Cream × Albino (Aa × Aa) . .	146	3
Albino × Cream (Aa × Aa) † . .	86	0

* Data kindly furnished in part by Dr Myron Gordon.
† Phenotypically wild-type.
General totals : Pigmented, 1176 ; Albino, 23.

TABLE 3

Totals of backcross progeny

Cross	Pigmented eye	Albino
(Wild-type × Albino) † × Albino (Aa × aa) * . .	138	15
Albino × (Wild-type × Albino) † (aa × Aa) . .	188	14
(Golden × Albino) † × Albino	102	16
Albino × (Golden × Albino) † (aa × Aa) . . .	107	14
(Blond † × Albino) × Albino (Aa × aa) . . .	107	16
Albino × (Blond × Albino) † (aa × Aa) . . .	20	0
(Cream † × Albino) × Albino (Aa × aa) . . .	87	8
Albino × (Cream × Albino) † (aa × Aa) . . .	226	24

* Data kindly furnished in part by Dr Myron Gordon.
† Phenotypically wild-type.
General total : Pigmented, 975 ; Albino, 107.

TABLE 4

Totals of pure albino

Cross	Pigmented	Albino
Albino × Albino (aa × aa) . .	0	43

Notes.—(1) Stock derived in part from Kaminer material.
(2) Data for one brood of 26 furnished by Dr Myron Gordon. All other data refer only to viable young. This figure, however, refers to a complete brood, distributed as follows :—
Died at birth, 14 ; Premature, 10 ; Viable, 2.

TABLE 5

Counts of embryos before birth

F_1

Cross	Pigmented eye	Albino
Wild-type × Albino (Aa × Aa)	174	52
Golden × Albino (Aa × Aa)	58	20
Blond × Albino (Aa × Aa)	88	31
Cream × Albino (Aa × Aa)	57	13
General totals : Pigmented, 377 ; Albino, 116 Ratio : 3·3 Pigmented ; 1 Albino		

Backcross

Cross	Pigmented eye	Albino
(Albino × Wild-type) × Albino (Aa × aa)	39	35
(Golden × Albino) × Albino (Aa × aa)	6	5
(Blond × Albino) × Albino (Aa × aa)	7	8
(Cream × Albino) × Albino (Aa × aa)	19	18
General totals : Pigmented, 71 ; Albino, 66 Ratio : 1·1 Pigmented ; 1 Albino		

TABLE 6

Linkage tests between Albino, Golden, and Blond

(i) *Albino and Golden* (F_2)

	Wild-type	Golden (gg)	Albino (aa)
Gold × Albino (AaGg × AaGg) and recip.	339	56	6

(ii) *Albino and Blond* (F_2)

	Wild-type	Blond (bb)	Albino (aa)
Blond × Albino (AaBb × AaBb)	73	22	8

(iii) *Albino and Cream* (= *Golden-Blond*) (F_2)

	Wild-type	Golden (gg)	Blond or Cream (bb or bbgg) †	Albino (aa)
Cream × Albino * (AaBbGg × AaBbGg) and recip.	143	31	42	11

* Phenotypically wild-type.

† Blond (bbGG) and Cream (bbgg) are sometimes distinguishable on gross examination but not reliably so, in the experience of the author. Hence, no attempt was made here to classify them separately. It has not been possible as yet to complete genetic tests to distinguish them.

Acknowledgments.—I should like to thank Dr Myron Gordon for his interest and assistance, for making available a strain of albino *Lebistes* which I did not have, for contributing data of his own on a third strain of albino *Lebistes*, and for making possible a number of the drawings, which are due to Mr Donald R. Rosen, as well as for contributing the colour photograph of albino *Lebistes* taken by Mr S. C. Dunton. I should also like to express my gratitude to Mr Harrison Miller and Mr Frederick Kraus for much help in obtaining the strain of the albino on which most of the work was done.

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